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EDITORIAL

THE CHANGING FACE OF MEDICAL PRACTICE

The practice of medicine has been with man since antiquity. Man has always endeavoured to overcome adversity and the bid to overcome the devastating effects of disease has always been a prime concern of mankind since creation. From humble beginnings, employing herbs and other materials available in the environment and relying on minimal personal skills, medical practice has evolved to the highly scientific pursuit now familiar to us. Thus, it is possible to recognise distinct phases or eras through which medical practice has evolved.

The earliest era has been described as the era of Ancient Medicine which may be said to have started in pre-historic times to about 500AD. More commonly it is dated from about 3000BC when records first began in Mesopotamia concerning such practices, to 500AD, enclosing the times of such illustrious names as Hippocrates (460-379BC), Erasistratus (about 300BC) and Galen (130-201AD). During this period, causation of disease was ascribed to the supernatural and later to changes in the "humours". Some of our local Ghanaian practices of the art of healing may be said to belong to this earliest era in the worldwide evolution of medical practice.

The next era in the evolution of medical practice is the era of Medieval Medicine also described as the era of "Monastic or Library Medicine" which was dominated by religious dogma and intolerance. Practice of medicine during this period was largely in the hands of clerics and became more theoretical than practical. In many ways, large areas of medical practice in Ghana and many developing countries may be said to belong to this era.

Renaissance Medicine (16th to 18th century) started in earnest with the development of 'bedside medicine' and saw the shift from the theory of 'spontaneous generation' of disease to the theory of 'contagion' which spurred the search for organic causes of disease, culminating in the discovery of microorganisms as a cause of disease. Towards the end of this era, large treatment centres or hospitals have become established and the concept of the cell as the unit of the body set the stage for further scientific study of the origin, as well as mechanisms in the causation of disease.

The current era of medical practice (19th century to date) is described as the era of Modern Medicine and has been aptly described by some as the era of scientific medicine, with emphasis on diagnosis of diseases. The scientific backbone of medical practice has been provided by the concurrent development and expansion of 'Laboratory Medical practice or Pathology'. The technical requirements of this aspect of medical practice have spawned rapid and

monumental technological advances in medical practice in general. The quest to understand the 'science behind the cure' has resulted in rapid expansion in the contribution of laboratory medicine, and more recently, imaging studies to diagnosis of disease and further patient management. It has also helped in the development of specialties and sub-specialties of medicine, all in the quest to improve patient management.

With changing practices in medicine and increasing respect for 'patient autonomy', the practice of medicine is undergoing further changes. With current increasing availability and usage of information on the internet, patients are able to access data on various medical conditions, as well as their treatment options. Thus, patients often come to see their doctor already armed with information gleaned from the internet. Some patients may have even tried treatment options recommended by online doctors before coming to see their doctor. This state of affairs is seen increasingly in the developed countries, requiring doctors to be aware of such possibilities. The negative aspect of this increasing self-medication is that some patients may present to the doctor with a disease which they have self-managed wrongly or inadequately. Alternatively, they may even present to a health centre as an emergency, with complications of self-administered treatment. This has necessitated a paradigm shift in modern clinical practice, leading to the emergence of the new specialty of Emergency Medicine.

This specialty has arisen out of the recognition that patient emergency conditions require collaborative activities of virtually all diagnostic and intervention specialties or subspecialties to act in concert under the same umbrella in order to optimally manage patients with these conditions. Doctors trained in emergency medicine are therefore, equipped with the skills to either handle emergency patients themselves, or summon other specialists to assist in the emergency situation. As a pre-requisite, hospitals in developed countries now operate comprehensive 'one-stop' emergency centres which are equipped to deal with virtually all emergencies. This practice is only now becoming available in developing countries. In Ghana, the first comprehensive emergency centre has recently been opened in Komfo Anokye Teaching Hospital in Kumasi and similar centres are being constructed or planned for other teaching and regional hospitals. The Ghana College of Physicians and Surgeons also, in recognition of this need, has commenced training of residents in emergency medicine.

One might argue that literacy rate in Ghana being low, Ghanaians are unlikely to explore the internet for

knowledge of and treatment for their various illnesses to the same extent as patients in developed countries. While this may be true to some extent, there is a fair proportion of well educated Ghanaians who fall into that category. More importantly, emergency centre care is equally necessary in Ghana, perhaps for different reasons. With poor access to health care facilities for the majority of Ghanaians, coupled with difficulties the newly established National Health Insurance Authority is going through, many patients first seek medical assistance from traditional healers, prayer camps or the numerous quacks in the society. Deeply ingrained traditional beliefs tend to drive them to the traditional healer first in many cases, especially in difficult-to-reach rural areas. The proliferation of mushroom churches, coupled with difficult economic circumstances also tend to drive the patients to various prayer camps. In addition to all these, uncontrolled peddling of unlicensed medications in public places (including public transport) and in the various media, promotes self-medication, sometimes with contaminated products. It is not surprising that patients present to hospital or health centres for the first time requiring emergency treatment for complications of these unorthodox methods of treatment. One must also add the daily carnage on Ghanaian roads through road-traffic accidents, with the many victims requiring emergency medical care. Whereas intensified education about the serious consequences of the above practices may help reduce the numbers of Ghanaians requiring emergency treatment for such misadventure,

the need for emergency centres in our hospitals is likely to grow because of the changing worldwide trend described earlier.

Emergency medicine therefore, has come to stay and marks the changing face of medical practice in our times. Hitherto, many hospitals have operated small emergency medical and/or surgical rooms, which are no more than sorting places for patients who might require admission for further management. The television series (ER from the USA or St. James's from the UK), although highly dramatised have helped in our understanding and appreciation of the need for comprehensive emergency facilities in modern medical practice.

That Ghana has come to realisation of the urgent need for equipping hospitals with comprehensive emergency centres is a laudable development. The initiative taken by the Ghana College of Physicians and Surgeons on this issue is commendable. The College has further advanced this cause in the 2014 Annual General and Scientific College Lecture, titled "Improving Emergency Care in Ghana" which was ably delivered by no less a person than Dr. G.D. Oduro, the inaugural Chair of the College's Faculty of Emergency Medicine. The gist of that lecture has been published in Volume 4, number 1 issue of the College's Journal. It is my submission that other developing countries with circumstances similar to Ghana would also require re-orientation of their healthcare delivery along the lines outlined for Ghana above.

JT Anim. FGCP



ORIGINAL ARTICLES

MALIGNANT SKIN TUMOURS IN KUMASI: A FIVE YEAR REVIEW

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Abstract

Introduction: Ultraviolet radiation is the primary aetiological agent in malignant melanoma (MM), squamous cell carcinoma (SCC) and basal cell carcinoma (BCC). Its effect on carcinogenesis can be influenced by endogenous and exogenous factors.

Objective: To document the clinical and epidemiological features of patients presenting with malignant skin tumours at Komfo Anokye Teaching Hospital (KATH), from January 2009 to December 2013.

Patients and Methods: Records of patients treated for malignant skin tumours at KATH were retrieved from the surgical out-patient department and theatre records and analysed.

Results: 38 patients comprising 16 males and 22 females were treated for malignant skin disease from January 2009 to December 2013. Their ages ranged from 12 to 84 years (mean 48.4, SD=20.2). Predominant lesions were SCC (17 cases), MM (12

cases), and dermatofibrosarcoma protuberans (DFSP), (four cases). SCC were located on the scalp (eight cases), lower limbs (six cases), upper limb (two cases) and trunk (one). All MM lesions were located on the foot. DFSP lesions were found on the leg (one case), trunk (one case), and shoulders (two cases). Basosquamous carcinoma (BSC) was found on the trunk of an albino.

All patients were treated surgically (48 procedures); three SCC patients had radiotherapy; one MM patient had chemotherapy.

Conclusion: SCC, MM and DFSP were the main malignant skin tumours managed. Chronic wounds, scars and skin bleaching were the exogenous factors; whilst albinism and xeroderma pigmentosum were the endogenous factors identified. For prevention, early case detection, adequate treatment of wounds and sun avoidance are advocated.

Key Words: Squamous cell carcinoma, basal cell carcinoma, malignant melanoma, xeroderma pigmentosum, Dermatofibrosarcoma

Introduction

Malignant tumours of the skin are either primary or secondary. Primary malignant skin tumours arise from the skin and its appendages. A secondary malignant skin tumour originates from a deeper tissue and spreads to involve the skin¹.

Ultraviolet radiation (UVR) is considered to be the primary aetiological agent involved in cutaneous carcinogenesis². It is known to have a causative effect related to malignant melanoma (MM), squamous cell carcinoma (SCC) and basal cell carcinoma (BCC). Both familial and environmental factors play a role in the aetiology of MM.

The familial/genetic factors include skin type, number of naevi, having atypical naevi, and having a family history of skin cancer. Intermittent exposure to UVR is the major environmental factor for MM, especially in combination with the endogenous factors³. MM is

uncommon in black Africans⁴ and Asians⁵ due to a better protection afforded by a larger amount of melanin pigment in the skin⁶. MM appears more often on the non-pigmented areas of the skin in non-Caucasians, are often of the acral lentiginous type and appear on the palms of the hands, soles of the feet and under the nails⁷.

UVR especially ultraviolet B (UVB) contributes to the formation of SCC⁸ and BCC⁹. These tumours develop through a multistep process involving activation of proto-oncogenes and/or inactivation of tumour suppressor genes in the human skin keratinocytes. High doses of UVR also lead to skin cancers by inducing reactive oxygen species that play an important role in tissue injury¹⁰. Common exogenous carcinogenic agents for SCC and BCC include tobacco use, human papilloma virus¹¹, previous burns¹², immunosuppression¹³, inflammatory lesions and ulcers of long standing¹⁴.

Since melanin pigment protects against the effects of UVR⁶ the malignant skin tumours seen in pigmented races are more likely to be due to these exogenous carcinogenic agents. Moreover genetic conditions such as albinism¹⁵ and xeroderma pigmentosum¹⁶ which predispose to skin cancer formation are also prevalent amongst Ghanaians.

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There have been some publications about primary malignant skin tumours in Ghanaians¹⁷ including those developing from exogenous causes¹⁸. However there has been no publication about the aetiology, and the epidemiology of malignant skin lesions in Kumasi or in the catchment area of Komfo Anokye Teaching Hospital.

The objective of the study was to document the clinical and the epidemiological features of patients presenting with malignant skin tumours at Komfo Anokye Teaching Hospital in Kumasi between the period from January 2009 to December 2013. This knowledge could shed some light on the risk factors for malignant skin disease in Ghana and help to institute preventive measures.

Patients and Methods

This is a retrospective study. The records of patients treated for malignant skin lesions at Komfo Anokye Teaching Hospital in Kumasi between January 2009 and December 2013 were retrieved from the records of the surgical out-patient department and from operating theatre records and analysed. Patients were included in the study only if their lesions had been confirmed histologically as malignant. Data recorded included the name, age and sex of the patients, the occupation, the site of the lesion, any previous illness, any previous surgery, the surgical procedure performed, the outcome of treatment and the histological diagnosis. Data was also collected on any adjuvant therapy given to the patient.

As a departmental policy, patients presenting with skin tumours at the Plastic Surgery Unit of Komfo Anokye Teaching Hospital were examined clinically; the lesion was staged and clinical photographs taken. The lesion was excised with either direct closure of the defect, skin grafting, flap repair, or left to heal by secondary intention as appropriate. Since most cases of MM were advanced at presentation (Breslow thickness >2mm) a minimum excision margin of 2cm was used¹⁹. SCC lesions were excised with 0.5 to 1cm excision margins²⁰. BCC lesions were excised with 2 to 3mm margins because of their location on the face. All other malignant lesions were excised with a minimum margin of 3cm, depending on the location and availability of local tissue. Block dissection of palpably enlarged regional lymph nodes was also performed where indicated. Patients requiring chemotherapy and/or radiotherapy were referred to the oncologist or radiotherapist for the appropriate treatment. The patients were followed up monthly for six months; three monthly for one year, and thereafter six monthly.

Ethical approval for the study was obtained from the Committee on Human Research Publications and Ethics (CHRPE) of the School of Medical Sciences, Kwame Nkrumah University of Science and Technology, Kumasi. Consent to participate in the study was obtained from all the patients, and from the parents or guardians, in the case of children during the

postoperative follow ups. All the patients consented to undergo the surgical procedures required for the treatment of their disease as a requirement of the hospital.

Results

A total of 38 patients were treated for malignant tumours of the skin. They were made up of 16 males and 22 females, giving a male to female ratio of 1:1.4. Their ages ranged from 12 to 84 years with a mean age of 48.4 (SD=20.2) with a median age of 49.2. The age distribution of the patients is depicted in Fig. 1.

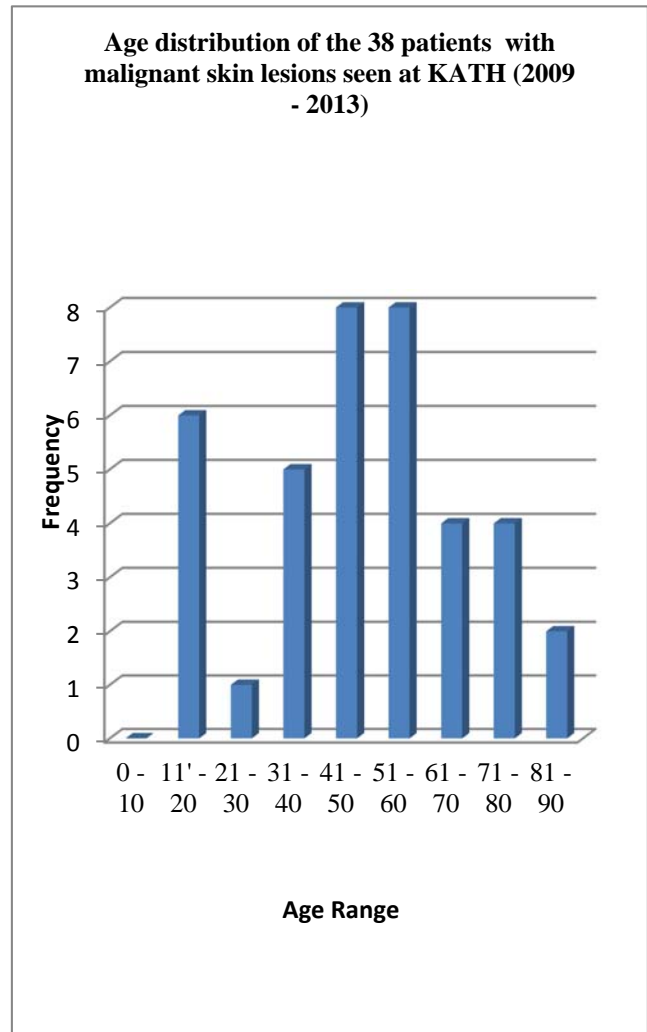


Fig. 1: Age distribution of 38 patients with malignant skin lesions seen at KATH (2009 – 2013)

The types and frequency of skin lesions treated during the study period, and the sex distribution are shown in Figs. 2 and 3. The youngest patient, a boy aged 12 years presented with a myxoid liposarcoma involving the left big toe, whilst the oldest patient was an 84 year old man with MM of the sole of the left foot.

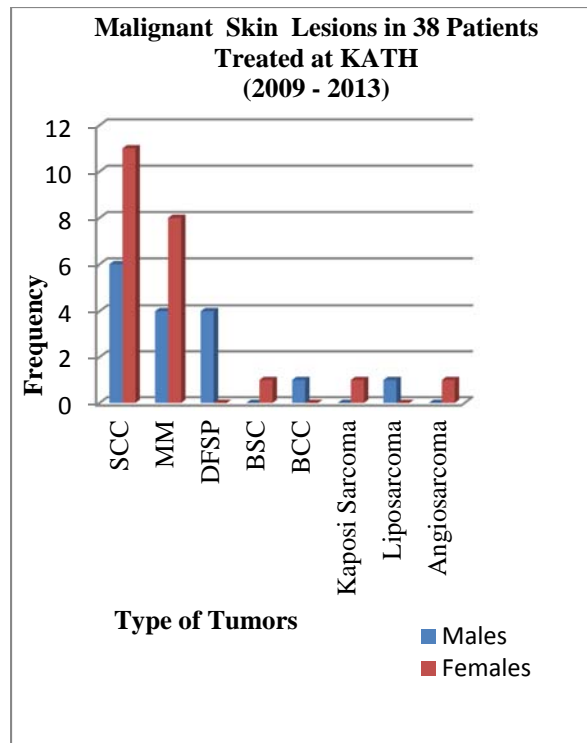


Fig. 2: Malignant skin lesions in 38 patients treated at Komfo Anokye Teaching Hospital from 2009 to 2013.

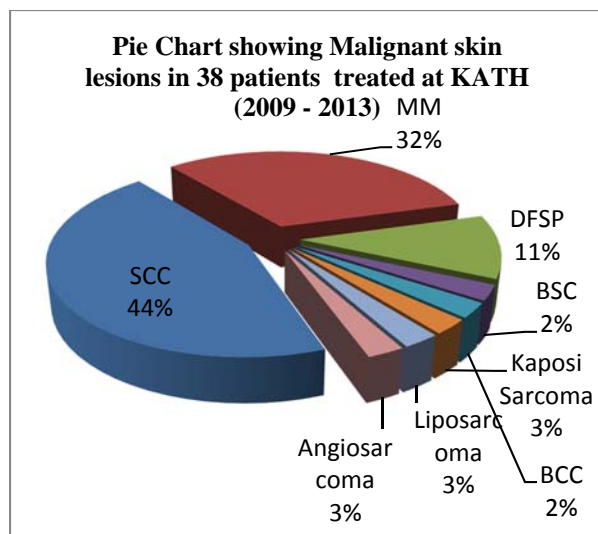


Fig. 3: Types of malignant skin tumours treated at Komfo Anokye Teaching Hospital from 2009 to 2013.

The predominant lesions were SCC (N=17) and MM (N=12). The age and sex distribution of these two lesions are shown in table 1.

The mean age for the patients with SCC was 45.0 (SD=15.2), whilst the mean age for MM was 65.5 (SD=15.4). Four of the cases of SCC arose de novo from normal skin; four of the cases developed from chronic traumatic wounds (Fig. 4), two from chronic burn wounds, two from Buruli ulcers, and two from atrophic skin resulting from skin bleaching.

Table 1: Age distribution for patients with squamous cell carcinoma and malignant melanoma (N=29).

Age range	SCC			MM		
	Male	Female	Total	Male	Female	Total
0 - 10	0	0	0	0	0	0
11 - 20	2	0	2	0	0	0
21 - 30	0	1	1	0	0	0
31 - 40	1	1	2	0	1	1
41 - 50	3	2	5	0	1	1
51 - 60	0	5	5	1	1	2
61 - 70	0	2	2	2	1	3
71 - 80				1	2	3
81 - 90				0	2	2
Total	6	11	17	4	8	12

Three of the patients who had the hereditary condition of xeroderma pigmentosum presented with two SCC lesions each. The distribution of the SCC lesions on various parts of the body was as follows: eight on the scalp, six on the lower limb, two on the upper limb and one on the trunk. All the 12 patients who presented with MM had their primary lesion on the foot, especially on the sole.



Fig 4: SCC (Marjolin's ulcer) developing from a chronic traumatic wound : (a) before and (b) after excision and skin grafting.

Dermatofibrosarcoma protuberans (DFSP) occurred in four male patients on the leg, trunk, and shoulder (two cases) respectively (Fig. 5). All the DFSP lesions were recurrent; three were excised three times; the fourth patient had a fourth excision and undergone radiotherapy.

Pre-operative**After excision and skin grafting**

Figs. 5a & 5b: Dermatofibrosarcoma protuberans of the shoulder in an adult male, before and after excision and skin grafting.

A female albino presented with basosquamous carcinoma (BSC) of the back of the trunk which was treated by excision and skin grafting.

A total of 48 surgical procedures were performed for the patients with malignant skin lesions. Excision was the commonest (N=41) surgical procedure; this was combined with partial thickness skin grafting in 25 cases. One 79 year old Caucasian who had BCC of the nose had the lesion excised and the defect reconstructed with a bilobed flap. Direct closure of a defect after excision was not possible in any of the patients owing to the larger sizes of the tumours presented; post excision defects were either skin grafted, covered with a flap, or left to heal by secondary intention (Table 2).

Table 2: Surgical procedures performed for patients with malignant skin tumours at KATH

Surgical procedure	Number of patients	Total
EXCISION		41
Excision & STSG	25	
Excision	15	
Excision & flap repair	1	
AMPUTATION		3
Below elbow	1	
Above knee	1	
Below knee	1	
BLOCK DISSECTION		3
Groin	2	
axilla	1	
INCISION		1
Incision biopsy	1	

Block dissection of the inguinal lymph nodes was performed for two female patients who had metastatic MM. Block dissection of the axillary lymph nodes was performed for one patient who had Marjolin's ulcer of the right forearm from a previous burn scar, the primary lesion having been excised and the defect skin grafted. The details of the surgical procedures performed are depicted in table 2. Three of the patients who had recurrent SCC also underwent radiotherapy; one patient with disseminated MM also had chemotherapy. Two cases of DFSP and two of SCC did not have clear excision margins from the pathology report. The former underwent re-excision; the latter had radiotherapy.

Discussion

The incidence of malignant skin tumours has been observed to be increasing worldwide; this increase is evenly distributed between developing and developed countries¹. The effects of the various aetiological factors may differ from one geographical area to another⁷, and this may affect the incidence and pattern of the disease.

In the current study malignant skin tumours did not occur in any patient younger than eleven years (Fig.1) even though some of the predisposing factors such as xeroderma pigmentosum and albinism were found in this age group. This emphasizes the significance of a minimum period of exposure to the carcinogenic agents required for the development of malignant skin disease.

SCC the commonest tumour tend to occur in the younger age group (mean age = 45years) than MM (mean age = 65years). This is because most of the cases of SCC were Marjolin's ulcers developing from chronic wounds resulting from trauma (four), burns (two) and Buruli ulcer (two). These conditions tend to occur more commonly in young adults than in the elderly. In addition, two middle-aged women presented with SCC developing from atrophic skin due to skin bleaching, a practice known to be carcinogenic in Ghana since the agents used contain hydroquinone¹⁸. The distribution of the SCC lesions showed a higher predilection for the scalp (47%) than the lower limbs (35%). Whilst several studies in the West African sub-region indicate SCC as the commonest malignant skin tumour, most of them identify the lower limbs as the commonest site, due to the higher incidence of injuries, ulcers and scars at this site^{21, 22, 23}. The higher number of these lesions on the scalp, as compared to the lower limbs in the current study could be due to the number of cases arising from old burn scars on the scalp, the three cases of xeroderma pigmentosum who had scalp lesions (Fig. 6), and cases arising de novo from the scalp.

SCC lesions on scalp Hyper and hypopigmented macules on hands



Figs. 6a & 6b: Adult female patient with two SCC lesions on the scalp with hyper and hypo-pigmented macules on the scalp and arms, typical of xeroderma pigmentosum.

MM comprised 32% of the malignant skin tumours (Fig. 3), second only to SCC (44%). MM occurred in the older age group (mean age=65), with a female preponderance (male to female ratio=1:2). The average duration of the primary lesion at presentation was 3.4 years. Most of the patients presented with advanced disease, most of which had already metastasized (Fig. 7). About 60% of the patients with MM had died of disseminated disease by the end of the study period whereas all of the SCC patients were alive. This apparent high mortality for MM could be explained by the late presentation, and the histological finding that most (75%) of the lesions were of the acral- lentiginous type, which is notorious for a poorer prognosis in black Africans²⁴.



Figs.7a&7b: Malignant melanoma on the sole of the left foot of an adult female patient with left inguinal lymph node metastasis.

Conclusion

Squamous cell carcinoma, malignant melanoma and dermatofibrosarcoma protuberans are the common malignant skin tumours seen in Kumasi. Though most of them arose de novo from normal skin, chronic wounds, scars and skin bleaching, as well as xeroderma pigmentosum and albinism are some of the aetiological factors identified. Prevention can be achieved by early case detection and treatment, prompt and adequate management of all wounds, education on sun

avoidance and protective clothing by the susceptible hereditary groups.

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References

1. Barton RM, Malignant Tumours of Skin in: Plastic Surgery, 2nd Ed vol 5 Tumours of the Head, Neck and Skin Ed Mathes SJ Saunders Elsevier Philadelphia 2006: 273-304
2. Narayanan DL, Saladi RN, Fox JL Ultraviolet radiation and skin cancer. *Int J Dermatol* 2010; 49: 978-986
3. Armstrong BK, Kricger A. How much melanoma is caused by sun exposure? *Melanoma Res* 1993; 56: 395-401
4. Parkin DM, Ferlay J, Hamdi-Cherif M. Sitas F et al Cancer in Africa: Epidemiology and Prevention 2003: IARC Press Lyon 162-165
5. Ohtsuka H, Nagamatsu S. Changing trends in number of deaths from malignant melanoma in Japan. 1955 – 2000 *Dermatol* 2003;207: 162-165
6. Brenner M, Hearing VJ, The protective role of melanin against UV damage in human skin. *Photochem Photobiol* 2008; 84: 539-549
7. Bellows CF, Belafsky P, Fortgang JS, Beech DJ. Melanoma in African- Americans: trends in biological behavior and clinical characteristics over two decades. *J Surg Oncol* 2002; 78: 10-16
8. Kubo Y, Murao K, Matsumoto K, Arase S. Molecular carcinogenesis of squamous cell carcinoma of the skin. *J Med Invest* 2002;49; 111-117
9. de Gruji FR, Longstreth J Norval M, Cullen AP et al. Health effects from stratospheric ozone depletion and interactions with climate change. *Photochem Photobiol Sci* 2003;2: 16-28
10. Joshi PC, Copper (II) as an efficient scavenger of singlet molecular oxygen. *Indian J Biochem s Biophy* 1998; 35: 208-215
11. Nguyeri M, Song S, Liem A, Androphy E et al. A mutant of human papilloma virus type 16 E6 deficient in binding alpha helix partners displays reduced oncogenic potential in vivo. *J Virol* 2002; 76:13039-13048
12. Bartle EJ, Sun JH, Wang XW. Cancers arising from burn scars: a literature review and report of twenty one cases. *J Burn Care Rehabil* 1990; 11:46-49
13. Rosenblatt L, Marks R. Deaths due to squamous cell carcinoma in Australia: is there a case for public health intervention? *Australas J Dermatol* 1996;37: 26-29

14. Hader RM, Brideman Shah S. Skin cancers in African Americans. *Cancer* 1995;75:667-673
 15. Yakubu A, Mabogunje OA. Skin cancer in African American albinos. *Acta Oncol* 1993;32:621-622
 16. Adu EJK Xeroderma Pigmentosum in Ghanaians: a report of three cases and review of literature. *West African J Med* 2014;33:82-85
 17. Adu EJK, Annan C. Primary malignant skin tumours in Ghanaians: a prospective study of 31 cases. *Nigerian J Plast Surg* 2008; 4:7-12
 18. Addo HA. Squamous cell carcinoma associated with prolonged bleaching. *Ghana Med J* 1981;34; 144-146
 19. Balch CM, Urist MM, Karakoussis CP et al Efficacy of 2cm surgical margins for intermediate thickness melanomas 1 to 4 mm. Results of a multi institutional randomized surgical trial. *Ann Surg*; 1993;218: 262-7
 20. Brodland DG, Zitelli JA, Surgical margins for excision of primary cutaneous squamous cell carcinoma. *J Am Acad Dermatol* 1992;27:241-8
 21. Ochicha O, Edino S T, Mohammed A.Z., Umar A.B. et al Dermatological malignancies in Kano, Northern Nigeria: A histopathological review. *Ann African Med* 2004;3 188-191
 22. Ayanlowo O, Daramola A O, Akinkugbe A, Olumide Y M et al Skin tumours at the Lagos University Teaching Hospital, Nigeria. *West African J Med* 2013;32: 286-290
 23. Mork F Cutaneous ulcers, sinuses and fistulae in: Badoe E A, Archampong E Q., da Rocha – Afodu J T,(eds). Principles and Practice of Surgery, including Pathology in the Tropics 3rd ed Ghana Publishing Corporation, Accra, 2000: 65-76
 24. Giblin A.V., Thomas J.M., Incidence, mortality and survival in cutaneous melanoma. *J Plast Reconstr Aesth Surg* 2007: 60:32-40
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PROSTATE CANCER DIAGNOSTIC METHODS IN KORLE BU TEACHING HOSPITAL, ACCRA, GHANA

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Abstract

Objective: The diagnosis of prostate cancer is based on a combination of digital rectal examination (DRE), serum prostate specific antigen (SePSA) estimation and trans-rectal ultrasound guided biopsy (TRUS-B) of the prostate, the latter being the gold standard for prostate cancer diagnosis. This study compared the diagnostic rate of prostate cancer in patients attending the urology clinic at Korle-bu Teaching Hospital, Accra, Ghana, using these methods.

Patients and Methods: One hundred and fifty male patients 45 years and older with abnormal DRE and raised or rising SePSA had TRUS biopsy done. The biopsies were processed routinely and all cancer positive slides were graded using the Gleason scoring

system. DRE findings were comparatively analysed statistically against SePSA and histological findings.

Results: Of the 150 subjects, 71(47.3%) were diagnosed as benign and 79(52.7%) had cancer on TRUS-B. Cancer diagnosis rate using a combination of DRE and SePSA was slightly higher (66.4%) than using DRE (64.5%) or SePSA (53.7%) in isolation.

Conclusion: DRE was found to have a high positive predictive value, probably due to the late presentation of majority of the patients in this study. SePSA alone is not very reliable, and results must be interpreted with caution due to significant false positive rates. Combining DRE and SePSA improves cancer diagnosis rates.

Key Words: DRE, SePSA, TRUS-biopsy, prostate cancer

Introduction

Prostate cancer is currently regarded as the most common cancer in men and the second leading cause of cancer related deaths in men in the United States of America^{1, 2}. The diagnosis of prostate cancer is based on a combination of digital rectal examination (DRE), serum prostate specific antigen (SePSA) estimation and trans-rectal ultrasound guided biopsy (TRUS-B) of the prostate. DRE has been found to have a poor rate of early cancer detection, but is more useful in diagnosing and staging locally advanced cancer^{3, 4}. SePSA estimation detects more tumours and at an earlier stage than DRE^{5, 6}. However, an elevated SePSA is not necessarily specific for cancer, because SePSA has been found to be elevated in some benign diseases of the prostate also^{7, 8}. In some cancers the SePSA is normal or lower than the traditional limit of 4ng/ml^{5, 6, 7}. TRUS-B is said to be the gold standard of diagnosis with a very high sensitivity⁹, although a cancer may be missed by biopsy due to inadequate sampling^{9, 10}. Because of these individual limitations, it is recommended that these three tests be used in combination in order to improve the diagnosis of prostate cancer^{7, 11}. The objective of this study was to evaluate and compare the diagnosis rate of prostate cancer, using DRE or SePSA in isolation and also in combination, in patients attending the urology clinic at Korle-bu Teaching Hospital (KBTH), Accra, Ghana.

SUBJECTS AND METHODS

The study was a prospective study involving male outpatients aged 45 years and older referred to the Korle-bu Teaching Hospital Urology Clinic and presenting for the first time. Many of the patients had been referred to the unit on account of lower urinary tract symptoms (urine retention, haematuria, infection). Others had been referred on account of an abnormal finding on DRE performed by a family physician, and/or an elevated total SePSA. The patients were assessed by one consultant urologist through a physical examination including DRE. Total SePSA assay was done in an accredited laboratory for all the patients. Patients were recruited into the study following the successful administration of informed consent. The study was approved by the Ethical and Protocol Review Committee of the University of Ghana Medical School and was carried out over a 4-month period. Detailed demographic and clinical data were collected on each patient including age, educational background, occupation, nature and duration of symptoms, and family history of prostate cancer.

Patients with abnormal DRE (abnormal being defined as the presence of hard, irregular, asymmetric, nodular or indurated areas), an elevated total SePSA or both underwent TRUS-B, performed by the urologist in an outpatient setting using a B&K ultrasound machine (Denmark), an 8.0 MHz end-firing transducer (BARD, USA) and an 18-gauge biopsy needle. A total of 12 biopsies were taken from the apex, mid-zone and the base of both sides of the prostate, with a minimum of two biopsies taken from each site. Any abnormal areas detected by DRE or TRUS, were incorporated into one of the six biopsy sites. The biopsies were fixed in 10% buffered formalin and processed into paraffin wax-embedded tissue blocks. The tissue was sectioned at a

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thickness of 5µm. Three slides, with 3 sections on each, were prepared for each of the six biopsies. Step sections were taken at 3µm intervals to ensure adequate sampling. The sections were stained with haematoxylin and eosin and examined for the presence of cancer and other histopathological changes. All slides positive for cancer were then graded using the Gleason scoring system.

Data on age, DRE findings, SePSA results and TRUS-B results were analysed using the Statistical Package for Social Sciences (SPSS) version 10, and the results presented as simple frequency tables. PSA density and cancer diagnosis rate using each method were calculated using the following formulae:

$$PSA\ density = Total\ SePSA / Gland\ volume.$$

$$Cancer\ diagnosis\ rate = (number\ of\ malignancies/number\ of\ subjects) \times 100.$$

RESULTS

A total of 150 patients were recruited into the study. The ages of the subjects ranged from 46 to 85 years with a mean age of 67.7 (SD 8.6) years. Majority of subjects (110, 73.4%) were between 60 and 79 years of age. Of the 150 subjects, 71(47.3%) were diagnosed as benign and 79(52.7%) had prostate cancer confirmed histologically. The ages of prostate cancer patients ranged between 49 and 84 years, with a mean of 68.2 (SD 8.6) years.

The recorded values of total SePSA in patients with prostate cancer showed a wide range between 8.3ng/ml and 6,305.0ng/ml with a mean total SePSA of 242.7ng/ml (SD 762.2). SePSA density values were obtained for 67 subjects out of the 79 with prostate cancer. The range was from 0.5 – 110.2, with a mean PSA density of 6.62(SD 17.5). The PSA density was found to be high (>0.15) in all patients with prostate cancer, and this finding was statistically significant ($X^2 = 5.245$, p -value=0.022).

Table 1: Combined Gleason Scores in 79 prostate cancer cases

Gleason score	Frequency	Percentage
3+2 (5)	2	2.5
3+3 (6)	14	17.7
3+4 (7)	14	17.7
3+5 (8)	5	6.3
4+2 (6)	1	1.3
4+3 (7)	8	10.1
4+4 (8)	10	12.7
4+5 (9)	8	10.1
5+3 (8)	4	5.1
5+4 (9)	7	8.9
5+5 (10)	6	7.6
Total	79	100

The lowest combined Gleason score was 5 and the highest 10 (Table 1). The mean combined Gleason score was 7.61 (SD 1.23).

Only 2 subjects with prostate cancer had low grade malignancy (Gleason score 5, 2.5%). Half of the subjects with cancer had high grade malignancy (Gleason score 8-10, 50%). The remaining 37(47%) had moderate grade tumours (Gleason score 6 & 7). There was a positive relationship between the total SePSA and the Gleason score (ie, the SePSA was high in patients with a high Gleason score). However, the association was not statistically significant. (P -value = 0.053). Table 2 shows the cancer diagnosis rates using the DRE and SePSA individually and in combination. Seventy one (71) subjects out of 110 who had abnormal features on DRE alone were found to have cancer, giving a cancer diagnosis rate of 64.5%. Thirty nine (39) subjects with abnormal DRE did not have cancer on biopsy.

Table 2: Comparison of Cancer diagnosis rates using DRE and PSA

	Group	Number	Number of malignancies On TRUS-B	Cancer diagnosis rate (%)
DRE	Normal	40	8	20.0
	Abnormal	110	71	64.5
PSA	0-4	3	0	0.0
	4-10	22	2	9.1
	>10	125	77	61.6
Normal DRE				
PSA	0-4	0	0	0.0
	4-10	10	0	0.0
	>10	30	8	26.7
Abnormal DRE				
PSA	0-4	3	0	0.0
	4-10	12	2	16.7
	>10	95	69	72.6

Table 2 also shows that for a total SePSA above 10ng/ml, 77 cancers were diagnosed out of 125 subjects, giving a cancer diagnosis rate of 61.6%. However, for SePSA between 4 and 10ng/ml, the cancer diagnosis rate was found to be 9.1%. Forty eight (48, 38.4%) subjects who had SePSA elevated above 10ng/ml did not have cancer on biopsy.

Combining the two tests, no cancers were diagnosed in subjects with a normal DRE and SePSA below 10ng/ml. For those with a normal DRE and SePSA above 10ng/ml, (30 subjects) 8 cancers were diagnosed. However, in subjects with both abnormalities on DRE and an elevated SePSA, 2/12 were diagnosed with cancer and SePSA between 4 and 10ng/ml and 69/95 were diagnosed with cancer and SePSA above 10ng/ml.

Table 3 shows that the cancer diagnosis rate using a combination of DRE and SePSA was higher (66.4%)

than using the DRE (64.5%) or SePSA (53.7%) in isolation.

Table 3: Cancer Diagnosis Rates in DRE and PSA

Variable	Total Number	Malignancies	Cancer detection rate
Abnormal DRE	110	71	64.5%
Elevated PSA>4ng/ml	147	79	53.7%
Both abnormal DRE and Elevated PSA>4ng/ml	107	71	66.4%

DISCUSSION

There has been great progress in the last few decades, in the investigation and treatment of prostate cancer. However, the basis for diagnosing early prostate cancer clinically remains dependent on DRE findings and elevated SePSA. Patients with abnormalities on DRE and/or an elevated SePSA are usually referred for TRUS-guided prostate biopsy.

The positive predictive value or cancer diagnosis rate of DRE in this study was 64.5%. Nwofor et al¹² also described a positive predictive value for DRE of 66.7%, in a study in Nigeria. The high positive predictive value in this study may be because many of the subjects in Ghana, as in Nigeria, present with high SePSA and abnormal features on DRE that suggest more advanced disease than subjects in other places where similar studies have been carried out¹³. DRE is therefore more useful in diagnosing late stage disease but its usefulness in detecting early cancers is still unclear. Researchers in other settings studying the use of digital rectal examination as a screening tool have reported values of 20% and 37%¹⁴ and in 1998 Schroder et al¹⁵ reported that DRE has a poor predictive value for detecting early prostate cancer and should be replaced with a more sensitive test. Findings from this study which assessed the use of DRE in diagnosis of prostate cancer (and not in screening), may indirectly be in agreement with this position and suggest that DRE, though not so useful in detecting early cancers, has a reasonably good predictive value in late stage disease.

No cancers were diagnosed in subjects with SePSA below 4ng/ml. This may be due to the small number of subjects (3 out of 150, 2.0%) presenting with SePSA in this range. Other studies on prostate cancer diagnosis in men with a SePSA of 4-10ng/ml show diagnosis rates of 20-32%^{16, 17}. The cancer diagnosis rate in the current study in this category of subjects was 9.1% and this low figure may again be because only 14.5% of the subjects had SePSA between 4 and 10ng/ml. The cancer diagnosis rate for

subjects with SePSA above 10ng/ml, however, was 61.8%, and this compares with the findings of Ng et al¹⁸ who reported a rate of 68% in this category of subjects in their study conducted in Sydney, Australia, and confirms that the higher the SePSA, the greater the likelihood of cancer being present. Overall, however, the cancer diagnosis rate using SePSA elevation above 4ng/ml alone was 53.7%, with an almost equal percentage of subjects with no cancer also having a SePSA above 4ng/ml. This suggests that the chances of a patient with an elevated PSA above 4ng/ml having prostate cancer, is roughly 50%. Thus, moderate elevation of SePSA may be due to benign disease of the prostate. Therefore, SePSA test results must be interpreted with caution, and patients appropriately counselled and referred for TRUS –B.

No cancers were diagnosed in subjects with normal DRE and SePSA below 10ng/ml, even though there were 10 subjects with normal DRE and SePSA in the 4–10ng/ml range. In a similar study, Ng et al¹⁸ found that 248 out of 812 subjects with normal DRE and total SePSA less than 10ng/ml had prostate cancer. The subject population they used, as in this study, was not a screening population, but rather a highly selected group who were recruited from the hospital's urology clinic. Reasons for the negative yield in this study compared with theirs may be the small sample size and the presumed late presentation of the subjects in this study.

For subjects with abnormalities on DRE, no cancers were diagnosed in those with SePSA within the conventional normal range of 0-4ng/ml. Though this finding suggests that an abnormal DRE and a SePSA of less than 4ng/ml is not likely to be due to prostate cancer, it is difficult to draw such a conclusion as there were only three (3) subjects with these characteristics in this study. In their much larger study, Ng et al¹⁸ found 26 cancers out of 98 subjects who had an abnormal DRE and SePSA below 4ng/ml. On the other hand, if a patient with an abnormal DRE also has a SePSA above 10ng/ml, then the likelihood of having cancer is much higher (72.6%) as shown in this study. Therefore a combination of abnormal DRE and elevated SePSA above 10ng/ml would be a strong indication for TRUS-biopsy.

In this study the cancer diagnosis rate using DRE alone was 64.5%; the rate using SePSA alone was 53.7%. When the two tests were combined, the cancer diagnosis rate increased to 66.4%. These figures indicate that more prostate cancers are diagnosed when DRE and SePSA are combined, and is in support of the findings of Ng et al¹⁸.

CONCLUSION

The present findings suggest that DRE has a high positive predictive value in our patients, but this is probably due to the late presentation of majority of the patients. The use of DRE in detecting early cancers

remains controversial. SePSA alone is not reliable, and results must be interpreted with caution because of significant false positive rates. Cancer diagnosis rates increase when the DRE and SePSA tests are combined. These patients should be referred for TRUS-guided biopsy of the prostate and histological confirmation.

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REFERENCES

1. Jemal A, Siegal R, Ward E, Murray T, Xu J, Smigal C et al. Cancer Statistics 2006. *CA Cancer J Clin*. 2006; 56:106-130.
2. Hayat MJ, Howlander N, Reichman ME, Edwards BK. Cancer Statistics, Trends and Multiple Primary Cancer Analyses from the Surveillance, Epidemiology and end results (SEER) Program. *Oncologist*. 2007; 12:20-37
3. Guinan P, Bush I, Ray V, Vieth R, Rao R, Bhatti R. The accuracy of the rectal examination in the diagnosis of prostate carcinoma. *N Engl J Med* 1980; 303:499-503
4. Yamamoto T, Ito K, Ohi M. Diagnostic significance of digital rectal examination and transrectal ultrasonography in males with prostate specific antigen levels of 4ng/ml or less. *Urology* 2001; 58:994-998
5. Barry MJ. Prostate-specific-antigen testing for early diagnosis of prostate cancer. *N Engl J Med*. 2001; 344:1373-1377
6. Montironi R, Mazzucheli R, Alagba F, Bostwick DG, Kronegrad A. Prostate specific antigen as a marker of prostatic disease. *Virchows Arch* 2000; 436:297-304
7. Goolsby MJ. Clinical Practice Guidelines – Use of PSA measurement in practice. *J Am Acad Nur Prac* 2001; 13:246-248
8. Anim JT, Kehinde EO, Sheikh MA, Prasad A, Mojiminiyi OA, Ali Y et al. Serum prostate specific antigen levels in middle eastern men with subclinical prostatitis. *Med Princ Pract* 2007;16:53-58
9. Chang JJ, Shinohara K, Hovey RM, Montgomery C, Presti JC Jr. Prospective evaluation of systemic sextant transition zone biopsies in large prostates for cancer detection. *Urology* 1998; 52:89-93
10. Presti J, Chang J, Bhargava V, Shinohara K. The optimal systematic prostate biopsy scheme should include 8 rather than 6 biopsies: Results of a prospective clinical trial. *J Urol* 2000; 163:163-167
11. Cupp MR, Oesterling JE. Prostate specific antigen, digital rectal examination and transrectal ultrasound: Their roles in diagnosing early cancer of the prostate. *Mayo Clin Prac* 1993; 38:297-306
12. Whittemore AS, Wu AH, Kolonel LN, John EM, Gallagher RP, Howe GR et al. Family history and prostate cancer risk in black, white and asian men in the United States and Canada. *Am J Epidemiol*. 1995; 141:732-740.
13. Schroder FH, Van der Maas P, Beemsterboer P, Kruger A.B, Hoedemaaker R, Rietbergen J et al. Evaluation of the digital rectal examination as a screening test for prostate cancer. Rotterdam section of the European Randomized Study of Screening for Prostate cancer. *J Natl Cancer Inst* 1998; 90:1817-1823
14. Nwofor AME, Oranusi CK. Cancer of the prostate: Experience at Nnewi, Southeast Nigeria. *Nig J Clin Prac*. 2004; 7:65-68
15. Gueye SM, Ziegler-Johnson CM, Friebel T, Spangler E, Jalloh M, MacBride S et al. Clinical characteristics of prostate cancer in African-Americans, American whites and Senegalese men. *Urology* 2003; 61:987-992
16. Catalona WJ, Smith DS, Ratliff TL, Dodds KM, Coplen DE, Yuan JJ et al. Measurement of prostate specific antigen in serum as a screening test for prostate cancer. *N Eng J Med* 1991; 324:1156-1161
17. Oesterling JE, Jacobsen SJ, Chute CG, Guess HA, Girman CJ, Panser LA et al. Serum prostate-specific antigen in a community based population of healthy men. Establishment of age specific reference ranges. *JAMA* 1993;270:860-864
18. Ng TK, Vasilareas D, Mitterdorfer AJ, Maher PO, Lalak A. Prostate cancer detection with digital rectal examination, prostate-specific antigen, transrectal ultrasonography and biopsy in clinical urological practice. *BJU International* 2005; 95:545-548.

SEX AND RURAL-URBAN DISPARITIES IN SELF-REPORTED CHRONIC NON-COMMUNICABLE DISEASES AND HEALTH RISKS AMONG OLDER ADULTS IN GHANA: IMPLICATIONS FOR THE NATIONAL AGING POLICY

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Abstract

Objective: Differences exist in the composition and morbidity/mortality patterns of older persons. This analysis determined sex differences and rural-urban disparities in common chronic diseases and health risks among older persons in Ghana.

Methods: This work was based on World Health Organization's (WHO) multi-country Study on global AGEing and adult health (SAGE), conducted in six countries including Ghana. Nationally representative sample of 4725 persons ≥ 50 years was involved in this analysis. Data were obtained on eight self-reported chronic diseases and analysed by sex and location.

Results: Women ≥ 50 years in rural-urban locations self-reported more ill-health than men of comparable age. Educational levels, household incomes and possession of health insurance were lower among rural residents. Alcohol and tobacco use were significantly

higher in rural locations (61% vs. 55.3%) and (29.6% vs. 20.9%) respectively, while obesity was significantly higher among urban residents (17.5% vs. 4.5%). Sex differences in prevalence of chronic conditions were statistically significant for-Angina (F:M 1.8), Arthritis (F:M 1.7), Depression (F:M 2.9), Diabetes (F:M 1.3), Hypertension (F:M 1.8) and Stroke (F:M 1.2). Urban-rural disparities were significant for chronic lung disease (1% vs. 0.4%), diabetes (6.4% vs. 2.2%), hypertension (22.8% vs. 7.3%) and stroke (4% vs 1.7%).

Conclusions: Preventive health programmes and provision and targeting of social protection (improved access to health care and pensions) should consider sex and location of vulnerable older persons as the country implements the national aging policy.

Key Words: *older adults, chronic diseases, sex differences, rural-urban differences, national ageing policy.*

Introduction

Trends in aging have been increasing in all regions of the world, including sub-Saharan Africa. The older adult population has increased steadily since 1950 in all six WHO regions of the world. In sub-Saharan Africa the projected older adult population (aged 60 years and older) will exceed 10% of the total population by 2050¹. In Ghana, the older adult population is projected to almost double from 6% in 2013 to 12% in 2050².

Globally, significant sex differences exist in the composition of the older adult population, the so-called 'feminization' of aging^{1,3-6}. Sex differences in morbidity and mortality are influenced by multiple factors, including a variety of biological and

behavioural differences between men and women. Sex differences in behaviour are linked to gendered roles such as social roles, behaviours, attitudes and psychosocial characteristics that are more common, more expected and more accepted for one sex or the other⁷⁻⁹. These differences also contribute to disparities in health outcomes: where prevalence and impacts of chronic non-communicable diseases may contribute to lower healthy life expectancy – with these differences having implications for health and quality of life at the individual level and how gender is included in policies on aging at the community level^{3, 10, 11}.

Aside sex differences in the prevalence of chronic non-communicable diseases, place of residence (rural or urban) are important. Differences in levels of awareness, access and utilization of health services vary between rural and urban locations. Population based hypertension surveys and a systematic review on hypertension have indicated rural urban differences; prevalence of hypertension was higher in urban than rural areas in Ghana^{12, 13}. A rural-urban assessment of hypertension in one of the regions of Ghana (Ashanti region) also indicated that the age-adjusted mean systolic and diastolic blood pressure levels were lower

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in rural men and women than in urban men and women¹⁴.

In Ghana, the numbers of older adults is growing, however the health and care of this segment of the population has not been accorded optimal attention¹⁵. Ghana has a national aging policy prepared in 2002 by the Ministry of Employment and Social Welfare (MESW) and launched in 2010¹⁵. The national policy recognized old age and health challenges, aging and the living environment and aging and gender as three of the seven main policy challenges confronting older persons in Ghana. The policy document suggested improving health, nutrition and well-being of older persons, improving housing and living environment of older persons and providing adequate attention to gender variations in aging among others as strategies to overcome these challenges¹⁵. The implementation of the policy is beset with many challenges including inadequate funds and budgetary allocation to support planned activities, multiple lead agencies involving different government ministries and departments, and thus limited focused leadership. In addition, the paucity of evidence-based national level data on older adults in Ghana, is an inhibitory factor to the provision of targeted interventions within limited national resources.

This analysis is based on a nation-wide survey data from the World Health Organization's (WHO) Study on global AGEing and adult health (SAGE) Wave 1 in Ghana, and is aimed at determining sex differences and rural and urban disparities in common self-reported chronic non-communicable diseases and health risks among older persons in Ghana. It is also aimed to contribute evidence based data to guide the process of implementing the national aging policy.

Methods

SAGE Wave 1 was undertaken in Ghana in a partnership between the University of Ghana's Department of Community Health, the Ministry of Health and WHO, as part of a multi-country longitudinal study to complement existing aging data sources to inform policy and programmes¹⁶. SAGE Wave 1 used nationally representative samples of persons aged 50+ years, with comparison samples of younger adults aged 18–49 years in Ghana. The face-to-face interview was conducted in Ghana (2007–08). Multistage cluster sampling strategies were used where households were classified into one of two mutually exclusive categories:

(1) All persons aged 50 years and older were selected from households classified as '50+ households'; and

(2) One person aged 18–49 years was selected from a household classified as an '18–49 household' (i.e. a household without a person \geq 50 years).

Household enumerations were carried out in the final sampling units. One household questionnaire was

completed per household where a household informant and individual respondent need not be the same individual. One individual was selected from 18–49 households, whereas for 50+ households all individuals aged 50+ were invited to complete the individual interview. Household-level analysis of weights and person-level analysis of weights were calculated, which included sample selection and a post-stratification factor. Post stratification correction techniques used the most recent population estimates provided by the Ghana Statistical Service¹⁶. Details on subject selection and instruments used for the SAGE Wave 1 survey in Ghana have been provided in the Ghana National SAGE Wave 1 Report¹⁶.

Respondents were interviewed regarding their household characteristics, socio-demographic and work history, perceived health status, risk factors and preventive health behaviours, chronic diseases and health services coverage, health care utilization, subjective well-being and quality of life, and social cohesion. Field work and data entry were undertaken between May 2007 and June 2008. SAGE was approved by the World Health Organization's Ethical Review Board as well as a national approval in Ghana. Informed consent has been obtained from all study participants. For this analysis, 4725 respondents \geq 50 years were involved (respondents 18–49 years were excluded).

Variables

Sociodemographic and socioeconomic variables included sex, age, marital status, highest educational level completed, health insurance status and household income levels. Ghana operates a social health insurance policy- the National Health Insurance Scheme- introduced in 2003 and became operational in most health public and private health facilities in 2005. There are a few private, voluntary and mutual health insurance schemes operated by health and corporate organizations in the urban centres¹⁷. Health insurance status of older persons was based on this.

Wealth or income quintiles were derived from the household ownership of durable goods, dwelling characteristics and access to services (improved water, sanitation and cooking fuel) for a total of 21 assets. Wealth levels were generated through a multi-step process, where asset ownership was converted to an asset ladder, Bayesian post-estimation method was used to generate raw continuous income estimates, and then income transformed into quintiles, Q1 to Q5; Q1 as lowest income and Q5 as highest income^{18,19}.

Health Risk indices

Tobacco use: Lifetime tobacco use was assessed with the question 'Have you ever smoked tobacco or used smokeless tobacco?' SAGE Wave 1 included other questions on the type of tobacco used (such as cigarettes, cigars, pipes, chewing tobacco, or snuff) and the pattern of tobacco consumption^{18,19}.

Alcohol use: Lifetime alcohol use was assessed with the question 'Have you ever consumed a drink that contains alcohol (such as beer, wine, spirits, etc.)?' Response options were 'Yes' or 'No, never'. In the survey both commercially available and home-brewed beverages were quantified in terms of alcohol content and quantity (i.e. a "standard drink") for comparability to other health surveys^{18,19}.

Body Mass Index (BMI): was derived from the measured weight and height of respondents.

Chronic diseases: SAGE gathered evidence on a selected range of chronic diseases typically more prevalent among older adults and that contribute to a large portion of non-communicable disease burden. In this analysis, data are presented for eight chronic diseases; angina, asthma, chronic lung disease, depression, diabetes mellitus, hypertension, osteoarthritis and stroke. The prevalence rates for these chronic disease conditions were based on responses to the question "Has a health care professional ever told you, you have...?".

Four disease conditions (angina, arthritis, asthma, and depression) have symptoms with sufficient specificity and sensitivity to improve estimation of prevalence by using established algorithms based on results from the symptom-reporting and this is described in another paper on SAGE Wave 1 by Kowal et al, 2012¹⁸. Although SAGE Wave 1 assessed measured hypertension, the focus of this analysis was on self-reported chronic disease conditions.

Data analysis

Outcomes of interest for this analysis included rural-urban disparities and sex differences in the burden of these eight chronic diseases among older adults ≥ 50 years in Ghana. Sociodemographic and socioeconomic variables analysed included age (50-59, 60-69 and 70-plus years), educational level, marital status, income levels (high-Q4 and Q5, or low-Q1, Q2 and Q3) and health insurance status (insured indicates respondent has mandatory or voluntary insurance or both and uninsured status indicates respondent has no insurance). Health risks such as alcohol use (yes or no), tobacco use (ever or never), obese (Yes or No) were assessed by location of residents (rural or urban).

Descriptive measures (frequencies, ratios, and percent) were used for the analysis and Chi-square test was used to determine significant sex differences and rural-urban disparities in the prevalence of selected chronic diseases among older persons at the 95% significance level, $p < 0.05$. Data analysis was conducted using SPSS version 21.

Results

Sociodemographic/ socioeconomic characteristics and health risks indices of older adults by location (rural and urban)

Among the 4725 older persons, 50.3% were women. Relatively higher proportions of urban residents (54.3%) were women while men formed the majority among older persons in rural locations (52.4%). Urban locations had higher proportion of younger older adults, 50-59 years (43.1% vs 37.7%) while rural locations had relatively higher proportion of the very old, 70 years and above (34.2% vs 30%).

There was a significant difference in the distribution of educational level by location; close to a third of all rural residents (62.7%) had no formal education compared to urban residents (43.2%). In addition, a higher proportion of urban residents had secondary level education or higher compared to those in rural locations.

Considering the marital status of the older persons, the currently married/ cohabiting were in the majority in both rural and urban locations. The proportion of older persons living with partners was relatively higher among rural residents (61.4% vs 53.8%), i.e. more older persons living without partners (widowed, separated or divorced) were in urban locations.

Over half of all older persons (in both locations) had low household incomes (Q1, Q2 and Q3). The proportion of older persons with low household incomes and without health insurance were significantly higher in rural locations i.e. (62.5% vs 55.3%) and (34.1% vs 44.8%) respectively.

Health risk assessment of older persons indicated significant disparities in alcohol and tobacco use by location; was higher in rural locations (61% vs 55.3% for alcohol) and (29.6% vs 20.9% for tobacco). Obesity however, was significantly higher among urban residents (17.5% vs 4.5%).

Sex differences and rural -urban disparities in prevalence of eight chronic diseases in older persons in Ghana

Table 2, demonstrates that the prevalence of all eight chronic diseases were higher among urban residents than rural residents. To illustrate this rural-urban disparity more clearly with hypertension (the most prevalent self-reported chronic condition), urban prevalence was 22.8 compared to 7.3 in rural locations. Self-reported hypertension was thus three times higher in urban than rural locations. Similarly, urban prevalence of stroke was more than double that in rural locations (4.0 vs. 1.7).

Table 1: Sociodemographic/ socioeconomic characteristics and health risks indices among persons ≥ 50 years by location ((rural and urban)), SAGE Wave 1, Ghana

Characteristics		Urban	Rural	Total N= 4725	Chi square (p-value)
Sex	Male	881 (45.7)	1466 (52.4)	2347 (49.7)	20.1 (0.001)
	Female	1045 (54.3)	1333 (47.6)	2378 (50.3)	
	Total	1926 (100)	2799 (100)	4725 (100)	
Age	50-59	829 (43.1)	1054 (37.7)	1883 (39.9)	15.2 (0.001)
	60-69	519 (26.9)	787 (28.1)	1306 (27.6)	
	70 and above	578 (30)	958 (34.2)	1536 (32.5)	
	Total	1926 (100)	2799 (100)	4725 (100)	
Educational Level	No education	832 (43.2)	1744 (62.7)	2576(54.7)	255.7 (0.001)
	Primary school completed	412 (21.3)	570 (20.4)	982 (20.8)	
	Secondary school completed	569 (29.9)	432 (15.2)	1001 (21.2)	
	Tertiary (college/university/postgraduate)	120 (6)	46 (1.7)	163 (3.3)	
	Total	1933 (100)	2792 (100)	4722 (100)	
Marital status	Never married	32 (1.5)	32 (1)	64 (1.2)	30.1 (0.001)
	Married/cohabiting	1032 (53.8)	1715 (61.4)	2747 (58.3)	
	Separated/divorced	299 (15.5)	332 (11.8)	632 (13.3)	
	widowed	562 (29.2)	721 (25.7)	1282 (27.2)	
	Total	1925 (100)	2800 (100)	4725 (100)	
Household Income	Low income	1065 (55.3)	1747 (62.5)	2812 (59.6)	24.2 (0.001)
	High income	862 (44.7)	1051 (37.5)	1913 (40.4)	
	Total	1927 (100)	2798(100)	4725 (100)	
Health insurance status	Yes (mandatory, voluntary or both)	862 (44.8)	955 (34.1)	1817 (38.5)	54.8 (0.001)
	No insurance	1063 (55.2)	1845 (65.9)	2908 (61.5)	
	Total	1925 (100)	2800 (100)	4724 (100)	
Health Risks					
Tobacco use	Yes	478 (20.9)	861 (29.6)	1329 (26)	40.8 (0.001)
	No	1500 (79.1)	1896 (70.4)	3396 (74)	
	Total	1978(100)	2747 (100)	4725 (100)	
Alcohol use	Yes	1079 (55.3)	1656 (61)	2735 (58.7)	14.0 (0.001)
	No	893 (44.7)	1097 (39)	1990 (41.3)	
	Total	1972 (100)	2753 (100)	4725 (100)	
Obesity	Yes	1549 (82.5)	2504 (95.5)	4053 (90.2)	192.2 (0.001)
	No	430 (17.5)	242 (4.5)	672 (9.8)	
	Total	1979 (100)	2746 (100)	4725 (100)	

These urban and rural disparities were statistically significant for four chronic disease conditions- chronic lung disease (1% vs. 0.4% ; p-value= 0.004), diabetes (6.4% vs 2.2%; p-value= 0.001), hypertension (22.8% vs 7.3%; p-value= 0.001) and stroke (4% vs1.7%; p-value= 0.008).

In both locations, self-reported prevalence of chronic disease were higher among females for all eight chronic diseases analysed, except chronic lung disease. Sex differences in self-reported prevalence of the chronic disease conditions were statistically significant for- Angina (F:M 1.8; p-value= 0.001), Arthritis (F:M 1.7; p-value= 0.001), Depression (F:M 2.9; p-value= 0.001), Diabetes (F:M 1.3; p-value= 0.020),

Hypertension (F:M 1.8; p-value= 0.001) and Stroke (F:M 1.2; p-value= 0.040).

The overall sex differences indicated that, depression had the highest F: M ratio of 2.9 followed by angina and hypertension (F: M of 1.8 respectively). Sex differences existed among older persons in urban as well as rural locations. In both locations females self-reported higher prevalence for all eight conditions, except chronic lung disease for urban and stroke for rural residents. Within each location, self-reported depression had the highest sex differential (F: M ratio 2.3 for urban and 3.3 for rural) i.e. relatively higher proportions of rural dwelling older women self-reported depression.

Table 2: Prevalence of chronic diseases by location (rural and urban), sex and sex ratio, among persons ≥ 50 years, SAGE Wave 1, Ghana.

Location	Sex (N=4725)	Chronic Disease prevalence (%)							
		Angina	Arthritis	Asthma	*Chronic Lung Dx	Depression	*Diabetes	*Hyper-tension	*Stroke
Urban	M	2.5	10.4	3.8	1.3	1.2	5.7	18.3	3.6
	F	4.6	15.7	4.4	0.8	2.7	7.0	27.0	4.4
	F: M	1.8	1.5	1.2	0.6	2.3	1.2	1.5	1.2
Urban Prevalence		3.6	13.2	4.1	1.0	2.0	6.4	22.8	4.0
Rural	M	2.4	9.7	3.1	0.3	0.6	1.9	4.9	1.8
	F	4.0	17.1	4.0	0.4	2.0	2.5	10.3	1.7
	F: M	1.7	1.8	1.3	1.3	3.3	1.3	2.1	0.9
Rural Prevalence		3.1	13.0	3.5	0.4	1.3	2.2	7.3	1.7
Overall F:M Ratio		1.8	1.7	1.2	0.9	2.9	1.3	1.8	1.2
Overall National Prevalence		3.3	13.1	3.7	0.6	1.6	3.9	13.6	2.7

* rural and urban differences in self-reported prevalence of these chronic disease conditions were statistically significant (Chronic lung disease p-value= 0.004, Diabetes p-value= 0.001, hypertension p-value= 0.001 and stroke p-value= 0.008) ; were higher among urban residents.

* sex differences in self-reported prevalence of these chronic disease conditions were statistically significant (Angina p-value= 0.002, Arthritis p-value= 0.001, Depression p-value= 0.001, Diabetes p-value= 0.020, Hypertension p-value= 0.001, and Stroke p-value= 0.040

Discussion

The analysis of SAGE Wave 1 data in Ghana demonstrates clear sex differences and rural-urban disparities in the prevalence of self-reported chronic non-communicable diseases among older persons in Ghana.

Women (50 years or more) self-reported more chronic non-communicable diseases than men of comparable age. This is in agreement with findings from the 2003 World Health Survey (WHS) in Ghana, where there was a higher prevalence of similar chronic conditions (angina, arthritis, asthma, diabetes and depression) among women²⁰.

Women self-reported more depression compared to men (F: M ratio of 2.3 in urban and 3.3 in rural location), this implies higher proportions of rural dwelling older women self-reported depression. SAGE Wave 1 however, did not assess the potential socioeconomic, cultural, religious and probable gender factors that could account for this. For effective implementation of interventions to improve health and social wellbeing of older adults, these factors may need further exploration.

Although the prevalence of self-reported chronic lung disease (CLD) was very low between the sexes and across locations, fewer women in urban locations self-reported CLD (F: M ratio of 0.6). These findings conform to the WHO Global burden of disease

estimates which demonstrates higher disability adjusted life years (DALYs) of depression for women and higher DALYs of CLD for men. Potential explanations for the observed higher prevalence of self-reported ill-health in women may be due to their relatively higher life expectancy, gendered patterns of assessment of personal health status, socioeconomic status, gendered roles and cultural and sociological influences^{1, 21, 22}.

As the nation's aged population increases, sex disparities in chronic conditions need to be given serious national health and social policy attention. The implication of this sex difference in self-reported ill-health for monitoring policy goals and programmes is extremely important especially regarding the strategy of bridging the gender disparities and gaps in the health of older women as outlined in the national aging policy document¹⁵.

The analysis indicated clear disparities in prevalence of some health risks, alcohol and tobacco use were more prevalent in rural locations while obesity was higher in urban locations. Urban residents had higher rates of self-reporting for all eight chronic diseases e.g. urban prevalence for hypertension was three times higher than the rural prevalence. Addo and colleagues in their review of population based hypertension surveys in Ghana from 1973 to 2009, showed the prevalence of hypertension ranged from 19.3% in rural to 54.6% in urban locations¹². A rural-

urban assessment of hypertension in one of the regions of Ghana (Ashanti region) indicated that the age-adjusted mean systolic and diastolic blood pressure levels were lower in rural men and women than in urban men and women¹⁴. Most of these studies were in specific locations and regions, however the SAGE Wave 1 is a nationwide survey data which provides empirical evidence on the rural-urban disparities in health risks and self-reported ill-health among older persons in the whole country.

Efficient use and allocation of the limited health budget targeted at these conditions, is essential if the health of the older person in Ghana is to improve.

It is important to note that, hypertension, diabetes and stroke which have dietary factors and sedentary lifestyle as strong associated risk factors²³ were more prevalent among older adults in urban locations where these risks are known to be higher.

The urban resident may be more aware with increased exposure to both print and electronic media and may have increased access to and utilization of health services compared to the rural resident. This difference may also be due to more urbanized lifestyles with increased tendency to sedentary work, easy access to transport and fast foods (energy dense diet) compared to the rural residents. Most rural Ghanaians are engaged in farming as the major occupation^{2, 24-26}, a physically taxing activity.

Health education and health promotion activities should be targeted at these risks and specific disease conditions in health facilities and within communities supported and promoted in line with the Ministry of Health's (MoH) Regenerative Health policy²⁷. The regenerative health policy of the MoH seeks to promote good eating habits, drinking adequate clean water, maintaining clean personal and environmental hygiene, regular exercises and having adequate rest²⁷. Targeted promotion of this will be beneficent to the older population.

This analysis again demonstrates rural-urban disparities in important sociodemographic and socioeconomic factors which have direct influence on the health and well being of older persons. Educational levels, household incomes and possession of health insurance which improve financial access to health care and social well-being were lower among rural residents. Improving health, nutrition, well-being, housing and living environment of older persons are important strategies outlined in the national aging policy of Ghana¹⁵. It is critical that Government's social intervention programmes such as the Livelihood empowerment against poverty (LEAP) and the national health insurance scheme (NHIS) be well targeted to identify the most vulnerable older persons especially in rural locations²⁸. LEAP is the provision of stipends from Government to support extremely poor and vulnerable households in all the regions of Ghana²⁸, while the NHIS aims to exempt persons 70 years and above from premium payments¹⁷. The targeting and

identification of these households and older persons have been difficult to attain. Government departments and agencies in health and social services should coordinate their activities to streamline the targeting of poor households. In implementing the national aging policy, efforts to provide basic living skills for older persons (those young enough, 50-59 years to work and support themselves and their families) at the local government level should be considered. A structured social intervention strategy (involving home-based and community care) to make older persons as independent as possible should be pursued.

It is indeed clear from the analysis that disparities in living arrangements exist. Relatively higher proportion of older persons live without partners (i.e. widowed and separated or divorced) in urban locations. Spousal support is pivotal to living arrangements, financial wellbeing and social relationships of older people but in divergent ways for older women and men²⁹. Widowhood represents the loss of a partner of many years who may have been the main source of companionship and support as well as a primary confidante^{30, 31}. Therefore, the widowed among older persons in both rural and urban locations may form a vulnerable group who should be included as beneficiaries of the national social intervention strategies indicated above. It is critical not to ignore urban dwelling widows during such national targeting of beneficiaries.

It is important to note also that in most developed and developing countries income and pension inequalities exist between men and women and by location; women have lower personal incomes than men in later life due mainly to smaller pensions³² with a direct consequence for risk of poverty in later life. Older persons in rural locations may not have pensions at all, since most are in the informal sector. Older men and women with relatively lower incomes may be socially disadvantaged due to their inability to perform expected gender-based social roles and may not attract social contact from family and friends^{33, 34}. Providing and improving pensions for older persons are imperative. The implementation of the New Pensions Act of the Republic of Ghana³⁵ which among others aim overall to diversify the sources and increase quantum of pensions will have beneficial effects on the health and social wellbeing of the aged in Ghana. Older persons outside the formal sector ought to be protected by other social interventions e.g. LEAP and NHIS.

Limitation: The self-report of health conditions, such as angina, depression and hypertension, is likely an underestimate of prevalence rates which is a potential limitation for this analysis³⁶. However, the WHO SAGE survey employed measures to improve estimates which are not presented here. It is important to note that per the objective of this analysis, prevalence of only self-reported hypertension (not measured blood pressure) in older adults were

considered, although SAGE Wave 1 has data on measured hypertension.

Summary: Analysis of SAGE Wave 1 data in Ghana demonstrates clear sex differences (women ≥ 50 years in rural and urban locations self-reported more chronic non-communicable diseases than men of comparable age) and rural-urban disparities in health risks and prevalence of self-reported ill-health among older persons in Ghana. Educational levels, household incomes and possession of health insurance (which improves financial access to health care and social well-being) were lower among older persons in rural locations.

As Ghana's population ages, a general trend towards increase prevalence of non-communicable diseases, and need for careful consideration of sex and rural-urban disparities in health risk in the older adult is inevitable. Preventive health programmes and provision of social protection (improved access to health care and pensions) need national attention through consideration of gender and location disparities among older adults in Ghana. Importantly, SAGE can be used to further document impact and also as a monitoring mechanism for the 2010 National Aging Policy with SAGE Ghana Wave 2 planned for 2013/14 and Wave 3 two years after this. A stitch in time saves nine!

Competing interests

The authors declare no competing interest. The views expressed in this paper are those of the authors. No official endorsement by the World Health Organization or Ministry of Health of Ghana/ Ghana Health Service is intended or should be inferred.

Authors' contributions

AE Yawson, P Dako-Gyeke, NA Hagan-Seneadza and KL Malm developed the concept, AE Yawson, G Mensah, N Minicuci, N Naidoo, S Chatterji, P Kowal and RB Biritwum are members of the WHO Multi-country SAGE Study Team involved in the conduct and analysis of the SAGE survey in Ghana. S Hewlett, BNL Calys-Tagoe, KL Malm, NA Baddo, P Martey, P Dako-Gyeke, NA Hagan-Seneadza and AE Yawson contributed to the writing and reviewing of the various sections of the manuscript. All the authors reviewed the final version of the manuscript before submission.

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References

1. United Nations Population Division, DESA. World Population Prospects: The 2010 Revision. New York: United Nations, 2011.
2. Ghana Statistical Service. 2010 National Population and Housing Census Report. Accra Published by Ghana Statistical Service, Accra, Ghana, 2011
3. Hosseinpoor AR, Williams JS, Jann B, Kowal P, Officer A, Posarac A, Chatterji S. Social determinants of sex differences in disability among older adults: a multi-country decomposition analysis using the World Health Survey. *Int J Equity Health*, 2012; 8, 11:52. doi: 10.1186/1475-9276-11-52.
4. World Health Organization. Men, ageing and health. Achieving health across the life span. Geneva: World Health Organization, 2002.
5. Mathers CD, Sadana R, Salomon JA, Murray CJL, Lopez AD. Healthy life expectancy in 191 countries, 1999. *Lancet*, 1999; 357(9269), 1685–91.
6. Hemstrom O. Biological and social conditions: hypothesis regarding mortality differentials between men and women. In: Ostlin P, Danielsson M, Diderichsen F, Harenstam A, Lindberg G, (eds.). Gender inequalities in health. Boston: Harvard University Press. 1998; pp. 195–223.
7. Davidson KW, Trudeau KJ, van Roosmalen E, Stewart M and Kirkland S. Gender as a Health Determinant and Implications for Health Education. *Health Education & Behavior*, 2006; 33 (6), 731-43.
8. Waldron I. Contributions of changing gender differences in behaviour and social roles to changing gender differences in mortality. In: Sabo D, Gordon DF (eds.). Men's health and illness.

- Thousand Oaks: Sage Publications. 1995, Pg 22-45
9. Weidner G and Messina C.R. Effects of gender-typed tasks and gender roles on cardiovascular reactivity. *Int J Behav Med*, 1995; 2, 66-82
 10. Miszkurka M, Haddad S, Langlois EV, Freeman EE, Kouanda S and Zunzunegui MV. Heavy burden of non-communicable diseases at early age and gender disparities in an adult population of Burkina Faso: world health survey. *BMC Public Health*, 2012; 10, 12:24. doi: 10.1186/1471-2458-12-24.
 11. World Health Organization. Department of Measurement and Health Information, Global Burden of Disease update, 2004. Geneva: World Health Organization. 2008.
 12. Addo J, Agyemang C, Smeeth L, de-Graft Aikins A, Edusei AK, Ogedegbe O. A review of population-based studies on hypertension in Ghana. *Ghana Med J*, 2012; 46, 4-11.
 13. Bosu WK. Epidemic of hypertension in Ghana: a systematic review. *BMC Public Health*, 2010; 10: 418. doi: 10.1186/1471-2458-10-418.
 14. Agyemang C. Rural and urban differences in blood pressure and hypertension in Ghana, *West Africa. Public Health*, 2006; 120, 525-33.
 15. Ghana National Aging Policy. Ministry of Employment and Social Welfare of Ghana 'Aging with Security and Dignity'. Published by Ministry of Employment and Social Welfare, Accra, Ghana, 2010.
 16. National Report on World Health Organization's Study on global AGEing and adult health (SAGE) in Ghana, Wave 1. University of Ghana, Department of Community Health. WHO, Switzerland, Geneva: WHO. 2013.
 17. Ghana National Health Insurance Authority. Annual Report of the National Health Insurance Scheme of Ghana, 2010. Published by the National Health Insurance Authority (NHIA), Ghana, 2011.
 18. Kowal P, Chatterji S, Naidoo N, Biritwum R, Wu Fan, Lopez Ridaura R, Maximova T, Arokiasamy P, Phaswana-Mafuya N, Williams S, Snodgrass JJ, Minicuci N, D'Este C, Peltzer K, Boerma JT, and the SAGE Collaborators. Data Resource Profile: The World Health Organization Study on global AGEing and adult health (SAGE). *Int J Epidemiol*, 2010; 1-11. doi:10.1093/ije/dys210
 19. Richard B. Biritwum, George Mensah, Nadia Minicuci, Alfred E. Yawson, Nirmala Naidoo, Somnath Chatterji and Paul Kowal. Household characteristics for older adults and study background from SAGE Ghana Wave 1. *Global Health Action* 2013; 6: 20096 - <http://dx.doi.org/10.3402/gha.v6i0.20096>
 20. National Report of the World Health Survey in Ghana. Department of Community Health, University of Ghana. World Health Survey 2002-2004. WHO, Geneva, 2004.
 21. Snow RC. Sex, gender, and vulnerability. *Global Public Health*, 2008; 3, 58-74
 22. Sundby J. A gender perspective on disability adjusted life years and the global burden of disease. Paper presented at a conference for the World Health Organization, Geneva, 1998.
 23. Cooper RS, Rotimi C. Establishing the epidemiologic basis for prevention of cardiovascular diseases in Africa. *Ethnicity and Disease*, 1993; S13-S22.
 24. Ghana Statistical Service, Ghana Health Service (GHS), and ICF Macro. Ghana Demographic and Health Survey 2008. Accra: Macro International. 2009.
 25. Ghana Statistical Service. National Population and Housing Census Report, 2000. Accra: Published by Ghana Statistical Service, Accra, Ghana, 2000.
 26. Ghana Living Standards Survey. Report of the Fifth Round, [September 2005- September 2006]. Published by Ghana Statistical Service, Accra, Ghana, 2008
 27. Ministry of Health Ghana. Annual Report, 2012. Published by the Ministry of Health, Republic of Ghana, Accra, 2013
 28. Ministry of Finance Ghana. Annual Budget Statement of the Government of the Republic of Ghana. Published by the Ministry of Finance, Accra, Ghana, 2013
 29. Arber S, Ginn J. Gender Dimensions of Age Shift. In: Lewis Johnson M, Bengsten VL Coleman PG (eds.). *The Cambridge Handbook of Age and Aging*. 2005; Chapter 6.5 pp. 527-536.
 30. Davidson K, Daly T, Arber S. Older men, social integration and organizational activities. *Social Policy and Society*, 2003; 2(2), 81-89.
 31. Askham J. Marriage relationships of older people. *Rev Clinical Gerontol*, 1994; 4(4), 261-268.
 32. Ginn J, Street D, Arber S. Women, work and pensions: international issues and prospects. Buckingham: Open University Press, 2001.
 33. Cohen S. Psychological models of the role of social support in the etiology of physical disease. *Health Psychology*, 1998; 7,269-97.
 34. Cooper H, Arber S, Fee L, Ginn J. The influence of social support and social capital on health: review and analysis of British data. London: Health Education Authority. 1999.
 35. National Pensions Act. National Pensions Act 766. Schedule Act of the Parliament of Republic of Ghana. Published by the Ghana Assembly Press, Accra, 2008
 36. Andresen E, Malmstrom TK, Miller DK, Miller JP, Wolinsky FD. Retest reliability of self-reported function, self-care and disease history. *Med Care*, 2005; 43(1), 93-97.

TWENTY-TWO YEARS OF REPAIR OF ATRIAL SEPTAL DEFECTS IN GHANA

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Abstract

Introduction: Atrial septal defects (ASDs) are one of the most common types of congenital heart disease. Repair is often necessary to forestall the various complications associated with the natural history. Surgical repair under cardiopulmonary bypass has been one of the standard modes of treatment. Much of the data available is from the rest of the world. There is not much data from the West African sub region. The purpose of this study is therefore to provide data from this sub region, guide both referring and operating clinicians in their decisions, and also to serve as comparative data for future studies. We analysed our institutional data to determine the age and sex distribution, the types of ASD and the outcome of surgical repair.

Subjects and Methods: A retrospective study was done for all patients who had surgical repair of ASD from January 1992 to December 2013 in the National Cardiothoracic Centre. The data was analysed using Microsoft excel 2010 software.

Results: There were 129 patients, 2 in the first year and 9 in the last year of the study. There were 53 (41.0%) males and 76 (59.0%) females. The mean age was

17.6 ± 14.9 years (1 – 70). The commonest age group was 1 – 10 years; 53 (41.0%), followed by 11 – 20 years; 36 (27.9%). Secundum ASDs were the commonest, 104 (80.6%), followed by primum ASDs 14 (10.9%), and sinus venosus ASDs 6 (4.7%). Large defects described as common atrium were 5 (3.9%). Autologous pericardium was used in repairing 125 (96.9%) and GORETEX® patch was used in the remaining 4 (3.1%). Thirty-three (25.7%) cases had associated cardiac anomalies that needed concomitant surgical intervention. The commonest was cleft in the anterior mitral leaflet causing severe mitral regurgitation 12 (9.3%), followed by pulmonary stenosis (PS) 11 (8.5%). There was an early mortality of 2 (1.6%). No other significant complication was encountered.

Conclusion: Surgical repair of ASDs in this sub region has been going on for over two decades now, with excellent outcomes. Patients with ASDs must be offered repair as soon as possible to forestall the serious complications that may follow unrepaired ASDs

Key Words: Atrial septal defect, surgical repair, outcome

Introduction

Atrial septal defects (ASDs) are one of the most common types of congenital heart disease. Though they commonly occur as isolated lesions, they may also occur as part of other major cardiovascular anomalies. Atrial septal defects are commonly classified according to their anatomic location in the interatrial septum into four main types. The commonest type is the ostium secundum ASD, comprising about 80%. The remaining 20% comprises the ostium primum, sinus venosus and coronary sinus ASDs. Occasionally, when the septal defect is so wide that there is just a thin rim of tissue between the two atria, the condition is called a common atrium. Repair of ASDs is often necessary to forestall the various complications associated with it,

like chronic flooding of the lungs leading to frequent chest infections, pulmonary hypertension and

Eisenmenger's syndrome. After the age of 2 years, ASDs rarely close spontaneously. The recommended age for repair therefore is 3 – 5 years¹. Surgical repair under cardiopulmonary bypass has been one of the standard modes of treatment, with a very good outcome¹⁻⁴. The other modes of treatment currently are the minimally invasive thoracoscopic approach and the transcatheter percutaneous device approach. Much of the data available is from the rest of the world. And since there is not much data on these from the West African sub region, we analysed our institutional data to determine the age and sex distribution, types of ASD and the outcome of surgical repair. This data covers a 22-year period.

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Conflict of Interest: None declared

Subjects and Methods

We carried out a retrospective study of all the patients who had surgical repair of ASDs from January 1992 to December 2013. The setting was the National Cardiothoracic Centre, Korle-Bu Teaching Hospital, Accra. The source of the data was the theatre records and the patients' case notes. The data was analysed using Microsoft excel 2010 software.

Results

A total of 129 patients were enlisted in the study, 2 in the first year of the study and 9 in the last year. There were 53 (41.0%) males and 76 (59.0%) females. The mean age was 17.6 ± 14.9 years (1 – 70), with a median of 12 years and a mode of 6 years. The commonest age group was 1 – 10 years; 53 (41.0%), followed by 11 – 20 years; 36 (27.9%). The age distribution is shown in Fig. 1.

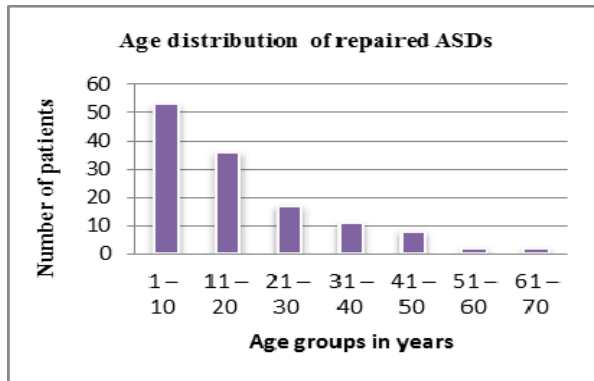


Figure 1. Age distribution of repaired ASDs

Most of the ASDs were secundum type 104 (80.6%), followed by the primum type 14 (10.9%), then the sinus venosus type 6 (4.7%). Large defects described as common atrium were 5 (3.9%). Autologous pericardium was used in repairing 125 (96.9%) and GORETEX® patch was used in the remaining 4 (3.1%). Thirty-three (25.7%) cases had associated cardiac anomalies that needed concomitant surgical intervention. The commonest was cleft in the anterior mitral leaflet causing mitral regurgitation 12 (9.3%), followed by pulmonary stenosis (PS) 11 (8.5%), shown in Table 1. There were 2 (1.6%) early mortalities. No other significant complication was encountered.

Table 1. Associated cardiac conditions that needed concomitant surgery

Associated condition	No.	Percentage	Surgery performed for it
MR 2 ^o cleft AML	12	9.3%	Mitral valve repair
PS	11	8.5%	RVOT Widening
PDA	4	3.1%	Ligation
PS+PDA	2	1.6%	Ligation + RVOT Widening
PAPVC	2	1.6%	Baffle of PVs into the LA
Cor triatriatum	1	0.8%	Excision of the septating membrane
Constrictive pericarditis	1	0.8%	Pericardiectomy
Total	33	25.7%	

MR: Mitral Regurgitation. AML: Anterior Mitral Leaflet. PS: Pulmonary Stenosis. PAPVC: Partial Anomalous Pulmonary Venous Connection. RVOT: Right ventricular outflow tract. PV: Pulmonary vein. LA: Left Atrium.

Discussion

There has been a gradual increase in the number of patients who have had surgical repair over the years. There were 2 in the first year of the study, and 9 in the last year. This is probably due to the increase in the population of the nation and also due to the increased awareness of the referring doctors of the existence of such a facility. The male: female ratio was 1: 1.4, confirming that ASD is slightly more common in females¹. The commonest age group was 1 – 10 years, followed by the 11 – 20 years age group (Fig. 1). These two groups comprise 68.9%. The age distribution gradually tapers until the 61 – 70 age group. It is not surprising to see adults with ASDs since the pathophysiology is slightly more favourable than other congenital lesions like VSDs, for example, thereby allowing surgical repair even at those older ages. The oldest patient was 70 years old and the youngest was 1 year old. Both were females with secundum ASDs. Not surprisingly, most of the ASDs were the secundum type 104 (80.6%), followed by the primum type 14 (10.9%), and the sinus venosus type 6 (4.7%). The common atriums were 5 (3.9%). This finding is very similar to other reported series in the literature where secundum ASDs comprise the majority, followed by primum, sinus venosus, and rarely the unroofed coronary sinus types^{1,2,5,6}. All the ASDs were repaired through a median sternotomy, except one which was performed through a right anterolateral thoracotomy. They were all performed under cardiopulmonary bypass. Most of them 125 (96.9%) were repaired with 0.6% glutaraldehyde treated autologous pericardium. The remaining 4 (3.1%) were repaired with GORETEX® patch. This was when the pericardium did not look healthy or was inadequate due to a very large defect. Newer methods of repairing ASDs such as the minimally invasive thoracoscopic approach^{4,7-12} and transcatheter percutaneous device closure have also been described recently with very good outcomes^{13,14}. The minimally invasive thoracoscopic approach also requires cardiopulmonary bypass, but uses a much smaller incision. This is a 6 – 8 cm anterior thoracotomy incision below the right breast in the 5th or 6th intercostal space. It has superior cosmesis and less morbidity but it is technically more difficult to do. The transcatheter percutaneous device closure is the latest approach in ASD closure. It is done in the catheterization laboratory by the interventional cardiologist. It involves making a needle puncture in the groin to get access to the femoral vein, inserting a catheter through it, advancing it through the inferior

vena cava to the right atrium where the occluding device is deployed across the defect to close it. Its advantage is that it does not need general anaesthesia, a big incision, nor cardiopulmonary bypass. Its disadvantage is that not all ASDs can be closed safely through the transcatheter approach. These are secundum ASDs without a good septal margin, primum ASDs, sinus venosus ASDs and coronary sinus ASDs.

In this study, the associated conditions that had the respective surgeries is shown in Table 1. The commonest associated lesion was a cleft in the anterior mitral leaflet 12 (9.3%). These were in primum ASDs. The second commonest associated lesion was pulmonary stenosis 11 (8.5%). Pulmonary stenosis as the commonest associated finding has been reported as 10%¹. There were 2 (1.6%) early mortalities. The first was in a 1½ -year old syndromic child who died intra-operatively. The cause could not be determined. The other was a 6-year old child with pulmonary hypertension. Echocardiography estimated a pulmonary artery systolic pressure of 65mmHg. Right heart catheterisation was not done at the time. She did well intra-operatively but had sudden cardiac arrest on the 5th post-operative day, and cardiopulmonary resuscitation was not successful. Long term complications after ASD repair in adults like atrial fibrillation, pulmonary hypertension and heart failure have not been analysed in this study because of the limitations of this study.

Limitations of the Study

Not all of the patients' case notes were available. This made it difficult to reliably estimate the medium and long term outcomes.

Conclusion

Surgical repair of ASDs in this sub region has been on-going for over two decades now, with excellent outcomes. Patients with ASDs must be offered surgical repair as soon as possible to forestall the serious complications that may follow unrepaired ASDs.

References

1. Baue AE, Geha AS, Hammond GL, Laks H, Naunheim KS. Glenn's Thoracic and cardiovascular surgery. 6th edition. Connecticut. Appleton & Lange. 1996:67:1115-1121
2. Kouchoukos NT, Blackstone EH, Doty BD, Hanley FL, Karp RB. Kirklin/Barratt-Boyes cardiac surgery. 3rd edition. Philadelphia. Elsevier. 2003:16:715-747
3. Murphy JG, Gersh BJ, McGoon MD, Mair DD, Porter CJ, Ilstrup DM, McGoon DC, Puga FJ, Kirklin JW, Danielson GK. Long-term outcome after surgical repair of isolated atrial septal defect. Follow-up at 27 to 32 years. *N Engl J Med*. 1990;13:323:1645-50.
4. Ryan WH, Cheirif J, Dewey TM, Prince SL, Mack MJ. Safety and efficacy of minimally invasive atrial septal defect closure. *Ann Thorac Surg*. 2003;75:1532-4.
5. Perloff JK, Marelli AJ. Perloff's clinical recognition of congenital heart disease. 6th edition. Philadelphia, Elsevier Saunders. 2012:15: 212-266
6. Soltoski PR, Karamanoukian HL, Salerno TA. Cardiac surgery secrets. 2nd edition. Philadelphia. Hanley and Belfus. 2004:2: 4-10
7. Lancaster LL, Mavroudis C, Rees AH, Slater AD, Ganzel BL, Gray LA Jr. Surgical approach to atrial septal defect in the female. Right thoracotomy versus sternotomy. *Am Surg*. 1990; 56:218-21.
8. Casselman FP, Dom H, De Bruyne B, Vermeulen Y, Vanermen H. Thoracoscopic ASD closure is a reliable supplement for percutaneous treatment. *Heart*. 2005; 91:791-4.
9. Ak K, Aybek T, Wimmer-Greinecker G, Ozaslan F, Bakhtiary F, Moritz A, Dogan S. Evolution of surgical techniques for atrial septal defect repair in adults: a 10-year single-institution experience. *J Thorac Cardiovasc Surg*. 2007;134:757-64.
10. Vistarini N, Aiello M, Mattiucci G, Alloni A, Cattadori B, Tinelli C, Pellegrini C, D'Armini AM, Viganò M. Port-access minimally invasive surgery for atrial septal defects: a 10-year single-center experience in 166 patients. *J Thorac Cardiovasc Surg*. 2010;139:139-45.
11. Ma ZS, Dong MF, Yin QY, Feng ZY, Wang LX. Totally thoracoscopic repair of atrial septal defect without robotic assistance: a single-center experience. *J Thorac Cardiovasc Surg*. 2011;141:1380-3.
12. Chu MW, Losenno KL, Fox SA, Adams C, Al-Habib H, Guo R, Menkis AH, Kiai B. Clinical outcomes of minimally invasive endoscopic and conventional sternotomy approaches for atrial septal defect repair. *Can J Surg*. 2014;57:E75-81.
13. Chessa M, Carminati M, Butera G, Bini RM, Drago M, Rosti L, Giamberti A, Pomè G, Bossone E, Frigiola A. Early and late complications associated with transcatheter occlusion of secundum atrial septal defect. *J Am Coll Cardiol*. 2002;20; 39:1061-5.
14. Post MC, Suttrop MJ, Jaarsma W, Plokker HW. Comparison of outcome and complications using different types of devices for percutaneous closure of a secundum atrial septal defect in adults: a single-center experience. *Catheter Cardiovasc Interv*. 2006;67:438-43.

PREDICTORS OF PRE-ECLAMPSIA: A HOSPITAL BASED STUDY IN ACCRA, GHANA

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Abstract

Introduction: Pre-eclampsia is a medical condition which develops after 20 weeks of pregnancy, where blood pressure is elevated to 140/90mm/Hg or more, with significant amounts of protein in the urine. It is a pre-cursor of eclampsia and leads to increased morbidity and mortality in the affected mother and fetus or baby. The only cure for pre-eclampsia involves delivery of the placenta. Pre-eclampsia is asymptomatic and difficult to predict in the first trimester of pregnancy.

Methods: This was a case control study done at the Police Hospital in Accra, using secondary data which were antenatal clinic records from 1st January 2008 to 31st December 2010. We sought to determine the number of deliveries complicated with pre-eclampsia, the proportion of deliveries complicated by pre-eclampsia, and risk factors of pre-eclampsia.

Results: The proportion of deliveries complicated by pre-eclampsia was 2.5%. We found no association between pre-eclampsia and season of delivery, maternal blood group, history of previous abortions, maternal infections of syphilis, HIV and Hepatitis B. We found maternal age of 25 years and above, parity of one and systolic blood pressure of 130mm/Hg or more at booking were statistically significant predictors of pre-eclampsia.

Conclusion: These three variables could be used to select pregnant women in the first 20 weeks of pregnancy for focused surveillance, and as a tool for selecting women for early referral for specialist care. We however recommend larger studies with the addition of lifestyle variables in further studies.

Key Words: Pre-eclampsia, Eclampsia, Ghana, maternal death, Pre-cursor

Introduction

Pre-eclampsia (PE) is the pre-cursor of eclampsia, a pregnancy-specific syndrome characterized by new-onset hypertension and proteinuria, occurring usually after 20 weeks' gestation. It is associated with high maternal mortality and morbidity as well as risk of fetal perinatal death, preterm birth, and intrauterine growth restriction¹. However it is asymptomatic and difficult to predict in the early stages of pregnancy. As a result, most cases are not detected early and are seen at health facilities in severe PE or eclampsia stage, which most of the time is difficult to treat or manage. The absence of screening tools makes diagnosis at an early stage of pregnancy in some antenatal clinic (ANC) settings difficult.

Although blood pressure (BP) elevation is the most visible sign of the disease, it involves generalized damage to the maternal endothelium, kidneys, and liver, with the release of vaso-constrictive factors being secondary to the original damage. There is currently no known treatment for pre-eclampsia and ultimate

treatment involves delivery of the placenta².

Pre-eclampsia is diagnosed when a pregnant woman develops high BP (two separate readings taken at least four hours apart of 140/90 mm/Hg or more). Also, laboratory values for PE include; proteinuria of >300 mg/24h, urine dipstick >1+, and protein/creatinine ratio >0.3³. A 24-hour urine protein analysis remains the criterion standard for proteinuria diagnosis. Alternatively, greater than 1+ protein on a dipstick analysis on a random sample is sufficient to make the diagnosis of proteinuria. A rise in baseline BP of 30mm/Hg systolic or 15mm/Hg diastolic, while not meeting the absolute criteria of 140/90mm/Hg is also considered important.

In the United States of America, PE is believed to be responsible for 15% of premature deliveries⁴ and 17.6% of maternal deaths^{5, 6}. In Ghana there was an estimated 11,166 cases of pre-eclampsia in 2003⁷. Despite its impact on maternal and child health, efforts to predict and prevent PE have been disappointing. Numerous strategies including the use of vitamin C and E supplementation have been shown to be of little benefit⁸.

The Ghana Ministry of Health's five year Programme of work (POW) from 2002 to 2006, of reducing maternal mortality ratio (MMR) from 214 to 150 per 100,000 live births (figures based on health institutional data only) by 2006 was not achieved⁹. The rate of reduction in MMR is so low that Ghana is unlikely to achieve the Millennium Development Goal

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5 which calls for a 75% reduction in the MMR of 1990, by 2015¹⁰. There is the need to develop a simple, low cost tool to identify women who are more likely to develop pre-eclampsia before the 20th week of pregnancy. This tool could be used in rural settings, clinics, and maternity homes and also serve as a guide for early referral to prevent progression to eclampsia, and thus reduce maternal morbidity and mortality from this cause.

Worldwide, approximately five to eight percent of pregnancies (over 6.6 million women) are affected by PE every year¹¹. Over 90% of these occur in developing countries. In 2008, there were 953 institutional maternal deaths in Ghana, with 168 of these deaths due to eclampsia¹². Since PE resolves postpartum, premature delivery of the baby may be essential to safeguard the mother's life. Up to a third of infants born of pre-eclamptic pregnancies are affected by intrauterine growth restriction¹³.

Consequently, many of the infants born of pre-eclamptic pregnancies require costly support in special care baby units. The burden of pre-eclampsia on health care resources is therefore substantial.

As PE remains a serious and poorly understood complication of pregnancy, there is the need to identify epidemiological and clinical risk factors to predict it before it threatens the survival of both mother and fetus. The study of risk factors and the underlying evidence base can be used to assess risk of pre-eclampsia at antenatal booking¹⁴.

Objectives

Our study objectives were:

- To determine the number of pre-eclampsia cases,
- To determine the proportion of deliveries complicated by pre-eclampsia,
- To determine risk factors of pre-eclampsia among pregnant women delivering at the Police Hospital

Methods

Study area

This study was conducted at the Police Hospital situated at Cantonments in the La Dade Kotopon Municipal Area in Accra in 2011. The Police Hospital was established for Police personnel and their dependents in 1976. However, since 1980, it has opened its doors to civilians as well and currently over 80% of out-patient attendees are civilians. In 2009, there were 1,638 deliveries. The following year however, there was a marginal decline of deliveries to 1,535. The department of Obstetrics and Gynaecology is headed by an experienced Consultant. Consistently, eclampsia has been one of the leading causes of maternal mortality in the hospital.

Study design

This was an unmatched case - control study, with the use of secondary data extracted from ANC cards and other available medical records to determine possible predictors of pre-eclampsia before the 20th week of pregnancy, with independent variables at the first visit or booking at the ANC.

Study population

The study population was pregnant women that delivered at the Police Hospital, from 1st January 2008 to 31st December, 2010.

Cases

A case was a pregnant woman with PE that delivered at the Police Hospital in Accra, from 1st January 2008 to 31st December 2010, irrespective of disease progression or outcome.

Controls

A control was a pregnant woman without PE that delivered at the Police Hospital, from 1st January 2008 to 31st December 2010.

Exclusion criteria

Pregnant woman that delivered with pre-existing hypertension, diabetes, renal disease, or previous history of PE were excluded from the study. Any pregnant woman, presenting at first visit at the ANC after the 20th week of gestation was also excluded from the study. Some controls were found to be having BPs higher than 130/90 on their first visit. However they were subsequently found not to be hypertensive. They were therefore not excluded from the study.

Sample size and sampling procedure

The sample size for the study was 200, with 100 cases and 100 controls. We used the Fleiss equation for analytic studies to calculate the sample size.

Selection of cases and controls

We found 115 women that delivered with complications of PE over the three year period, out of which 103 fit our case definition. 100 of the 103 were selected as cases by simple random sampling. In selecting the controls, a total number of 4,637 records arranged according to their dates of first ANC attendance for the three year period was obtained. This number was divided by 100, which was the sample size of controls. Every 46th record was thus selected to be part of the study. The selection of the first ANC card was done by cutting 46 identical pieces of paper, and then numbering them from 1 to 46. They were then folded identically and put in a bowl and thoroughly mixed. A blindfolded assistant picked the first piece of paper with the number 13 written on it. We therefore started picking the controls from the 13th record and picked the rest at intervals of 46. This was repeated till the 100 controls were retrieved.

Data capture and analysis

Data entry sheets were used to capture data from the ANC attendance cards and folders of patients.

Where necessary, medical officers involved in management were consulted for clarifications. Quality control checks were performed for completeness, internal consistency and accuracy of data collected. Univariate analysis was done by running frequencies, percentages, and means. Bivariate analysis was performed with the use of odds ratio to compare categorical variables. Student's t-test was used for quantitative variables. Multivariate analysis involved the use of binary logistic regression to show the relationship between binary dependent and independent variables. Data was analyzed with Epi info version 3.5.2 and SPSS version 17.

Results

The number of deliveries complicated by pre-eclampsia for the study period was 115, with total number of deliveries being 4,637. The proportion of pre-eclampsia cases to total deliveries was 2.5%, over the three - year period. The breakdown per year is shown in Table 1.

Table 1: Pre-eclampsia cases from 2008 to 2010.

Year	Number of mothers with pre-eclampsia	Number of deliveries	Proportion of pre-eclampsia/deliveries
2008	29	1488	0.020 (2%)
2009	35	1614	0.021 (2.1%)
2010	51	1535	0.033 (3.3%)
Total	115	4637	0.025 (2.5%)

As shown in Table 2, most of the cases (38%) were in the 25 to 29 age group. Among the controls however, majority were in the 30 to 34 age group (33%).

Table 2: Distribution of cases and controls by age groups

Age group (years)	Frequency		Percent	
	Case N=100	Control N=100	Case N=100	Control N=100
<15	0	2	0	2
15-19	3	4	3	4
20-24	9	21	9	21
25-29	38	26	38	26
30-34	30	33	30	33
35-39	14	9	14	9
40-44	6	5	6	5

The minimum and maximum ages among the cases and controls were 19 and 42, and 13 and 39 respectively. The mean age of the cases were 29.5 ± 5.2 , and 29.8 ± 5.3 for the controls respectively. The difference between the mean of the ages was however not statistically significant ($p > 0.05$).

Majority were Christians (85%) with the rest being Muslims, whilst most of the mothers (97%) were married. A total of 186 (93%) records had values for blood group. Majority of the mothers 88 (47.3%) were of blood group O, blood group B were 50 (26.9%), blood group A were 43(23.1%), and blood group AB were 5 (2.7%), as shown in Figure 1.

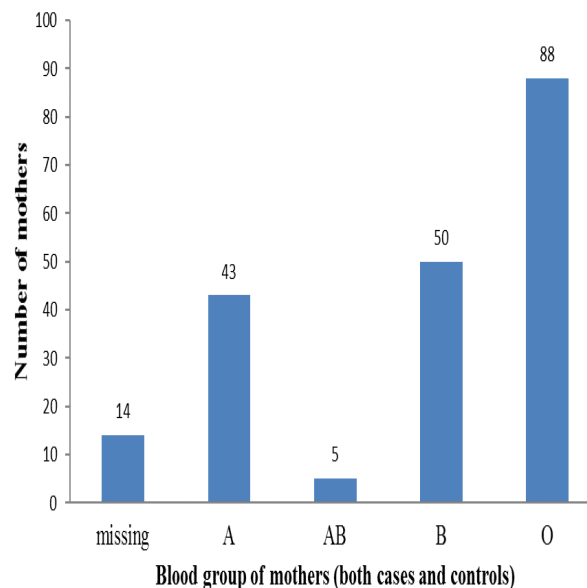


Figure 1: Shows maternal blood groups of mothers for both cases and controls

The minimum and maximum systolic blood pressure (SBP) values among cases were 85 and 190 mmHg. The mean Systolic BP (SBP), of cases and controls were 123.5 ± 18.9 and 112.1 ± 10.8 respectively. The mean SBP was significantly higher among the cases ($p < 0.001$). The minimum and maximum diastolic blood pressures (DBP) among cases were 50 and 130, and among controls were 50 and 100 respectively. The mean DBP among cases and controls were 78.8 ± 15.7 and 68.5 ± 8.9 . Cases were found to show significantly higher DBP than controls ($p < 0.001$).

Mean number of previous deliveries for the cases and controls were 1.1 ± 1.0 and 1.0 ± 0.9 respectively and showed no statistically significant difference ($p > 0.05$). Mean number of previous pregnancies for cases and controls were 2.5 ± 1.4 and 2.3 ± 1.1 respectively and also showed no statistically significant difference ($p > 0.05$). Twenty four percent of cases had a previous history of abortions, as against 11% of controls. Previous history of abortions appeared to be associated with an increased risk of PE. This finding was statistically significant (OR 2.56, 95% CI 1.18-5.55, $p=0.0246$). Eighty-nine percent of cases were 25 years or above as compared to 77% of controls. Maternal age of 25 years and above appeared to increase the risk of

PE. This finding was also statistically significant (OR 2.42, 95% CI 1.11-5.28, $p < 0.05$). Being married was not associated with PE (OR 3.12, 95% CI 0.62-15.89, $p > 0.05$). A parity of 1 appeared to be protective against PE (OR 0.5 95% CI 0.27-0.91 $p < 0.05$). Forty-two percent of cases were nulliparous as compared to 28% of controls. Nulliparity appeared to increase the risk of

pre-eclampsia, though this finding was not statistically significant (OR 1.86, 95% CI 1.03-3.35 $p > 0.05$).

Fifty one percent of cases and 55% of controls delivered in the wet season. There were no significant difference between the cases and controls concerning season of delivery (OR 0.85, 95%CI 0.49-1.48, $p > 0.05$).

Table 3: Risk factors of pre-eclampsia

Parameter	Cases		Controls		Odds ratio (OR)	Confidence interval (CI)	P value
	Number	%	Number	%			
Sickling positive	13	13	19	19	0.64	0.29-1.37	0.2470
Sickling negative	87	87	79	79			
Rhesus positive	85	85	80	80	1.42	0.68-2.96	0.3509
Rhesus negative	15	15	20	20			
Hep B Positive	3	3	6	6	0.5	0.12-2.06	0.4983
Hep B negative	97	97	94	94			
Syphilis reactive	1	1	0	0	-	-	-
No syphilis	99	99	100	100			
HIV pos	1	1	0	0	-	-	-
HIV neg	99	99	100	100			
Systolic BP ≥ 130	44	44	11	11	6.38	3.03-13.33	<0.0001
Systolic BP < 130	56	56	89	89			
Diastolic BP ≥ 90	55	55	27	27	3.31	1.83-5.97	<0.0001
Diastolic BP < 90	45	45	73	73			

We found no statistically significant association between maternal blood group and pre-eclampsia.

At the beginning of the study those with previous history of hypertension were excluded. However some records were found with high booking blood pressures but subsequent BP checks proved they were not hypertensive and were not medicated for that. Such records were included in the study.

As seen in Table 3, SBP of 130mm/Hg or more increased the risk of PE (OR 6.38 95% CI 3.03-13.33 $p < 0.0001$), as a DBP of 90mm/Hg or more (OR 3.31, 95% CI 1.83-5.97 $p < 0.0001$). These findings were statistically significant.

Thirteen percent of cases were sickling positive, as compared with 19% of controls.

Positive sickling status appeared to be protective but this was not statistically significant (OR 0.64, 95% CI 0.29-1.37, $p > 0.05$). Rhesus positive state appeared to be associated with PE, but this was not statistically significant (OR 1.42, 95% CI 0.68-2.96, $p > 0.05$). There was no statistically significant association between PE and maternal infections of hepatitis B, syphilis and HIV.

A multivariate model was constructed for variables that showed a statistically significant association with PE in the univariate analysis.

Table 4: Multivariate analysis of significant variables

Variable	B	S.E	Wald	OR	P value	95% CI
Age	0.895	0.434	4.265	2.448	0.039	1.047 - 5.726
Previous abortions	0.813	0.435	3.496	2.255	0.062	.961- 5.290
Systolic BP	1.264	0.537	5.545	3.540	0.019	1.236 - 10.14
Diastolic BP	0.762	0.604	1.590	2.142	0.207	0.656 - 7.000
Parity	- 0.777	0.347	5.009	0.460	0.025	0.233 - 0.908

These variables were maternal age, previous abortions, parity, SBP and DBP. The results of the multivariate analysis are presented in table 4. Out of the five variables, only three were found to be significant. These were maternal age, parity, and systolic blood pressure.

Discussions

We set out to determine possible predictors of pre-eclampsia before clinical manifestation, which is before the 20th week of pregnancy, with information on the antenatal cards of pregnant women at the first antenatal visit. This first visit must have taken place before the 20th week of pregnancy to be included in the study.

We found the proportion of deliveries complicated by pre-eclampsia for the three year period was 2.5%. This figure falls within the range of 1.5 to 4.2% found elsewhere^{15, 16, 17, 18}. Our finding however contrasted with a study done at the Korle - Bu Teaching Hospital¹⁹, which reported prevalence of pre-eclampsia to be 7.03%. This is not surprising, since the Korle-Bu Teaching Hospital is the largest referral center in Ghana with complicated cases referred to it. Though several studies had sought to establish a link between maternal infection and pre-eclampsia with inconsistent results^{20, 21} we found no association of pre-eclampsia and maternal infection with HIV, Hepatitis B virus, and syphilis. We found no significant association between pre-eclampsia and season of delivery. This finding is consistent with earlier studies in Iran²². Our finding however contrasts with other findings from South Africa²³ which reported that pre-eclampsia occurs more frequently in winter and Nigeria²⁴, where it peaked in the rainy season.

Again we found no statistically significant association between maternal blood group and pre-eclampsia. This finding is in agreement with a study in the United Kingdom²⁵ but however contrasts with a

population based study in Finland²⁶ that found that Blood group AB was associated with an increase in the risk for pre-eclampsia. We found that previous history of abortion was 2.6 times more likely to be associated with pre-eclampsia. This was not sustained in the multivariate analysis. Such associations were however found in other studies^{27, 8}. Maternal age of 25 and above was associated with increased risk of pre-eclampsia in our study which was confirmed in multivariate analysis. Other studies did not show this association^{14, 29}. We found no statistically significant association between PE and nulliparity which is in disagreement with other studies which found nulliparity to be significantly associated with increased risk of PE¹⁴.

In our study, first booking systolic BP of 130 mm/Hg or more significantly increased the risk of pre-eclampsia. This was sustained in multivariate analysis. This finding is in disagreement with a study that reported that a raised diastolic BP at booking increased the risk of pre-eclampsia¹⁴.

Limitations of the study

Since there was no qualitative arm of this study, mothers were neither examined nor interviewed so we could not study the effect of lifestyle variables on pre-eclampsia. Since this study was done in only one health facility with a relatively small sample size, the findings may not therefore be extrapolated on the general population. Male partner's variables could also not be assessed since these were not available on the records.

Conclusion

We analyzed data from the ANC of the Police hospital from 1st January 2008 to 31st December 2010, with the objective to determine the number of pre-eclampsia cases, the proportion of deliveries complicated by pre-eclampsia, and risk factors of pre-eclampsia.

The total number of deliveries for the period was 4,637 and the number of deliveries complicated by preeclampsia was 115. The proportion of deliveries complicated by PE was 2.5%.

We found in multivariate analysis that, maternal age of 25 years and above, previous parity of one, and systolic blood pressure of 130 mm/Hg or more at booking were statistically significant predictors of pre-eclampsia.

Recommendations

These findings of this study could be used to predict pre-eclampsia among pregnant women at first antenatal clinic attendance. This will enable the selection of these pregnant women for focused surveillance of pre-eclampsia and therefore lead to early detection and management to prevent adverse outcomes.

We recommend further studies should have a qualitative component and involve a larger sample size, preferably from more health facilities.

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References

- Sibai B, Dekker G, Kupferminc M (2005). Pre-eclampsia. *Lancet* 365 (9461):785-799.
- Silasi M, Cohen B, Karumanchi SA, Rana S (2010). Abnormal placentation, angiogenic factors, and the pathogenesis of preeclampsia. *Obstet Gynecol Clin North Am.* 37 (2):239-253.
- Report of the National High Blood Pressure Education Program Working Group on High Blood Pressure in Pregnancy. (2000). *Am J Obstet Gynecol.* 183 (1):S1-S22.
- Goldenberg RL, Rouse DJ (1998). Prevention of premature birth. *N Engl J Med.* 339 (5): 313-320.
- Koonin LM, Mackay AP, Berg CJ (1997). Pregnancy-related mortality surveillance-United States, 1987-1990. *MMWR CDC Surveill Summ.* 46 (4): 17-36.
- Berg JC, Jeani C, William MC, Sara JW (2003). Pregnancy-Related Mortality in the United States, 1991-1997. *Obstet Gynecol.* 101: 287-296.
- Statistics By Country For Preeclampsia Available at <http://www.cureresearch.com/p/preeclampsia/stats-country.htm> Accessed on 3-11-10
- Poston L, Briley AL, Seed PT, Kelly FJ, Shennan AH (2006). Vitamin C and vitamin E in pregnant women at risk for pre-eclampsia (VIP trial): Randomised placebo-controlled trial. *Lancet.* 367(9517):1145-1154.
- Ghana Health Sector Programme of Work 2002-2006. Independent Review of POW-2006, June 2007, Page 21.
- United Nations Development programme (UNDP). 2003. Indicators for monitoring the millennium development goals: definitions, rationale, concepts, and sources. New York: United Nations.
- Landau R, Irion O (2005). Recent data on the physiopathology of pre-eclampsia and recommendations for treatment. *Rev Med Suisse* 1(4): 290-25.
- Reproductive and Child Health Division, Ghana Health Service Annual Report, 2008, Page 13.
- Walker JJ (2000) Pre-eclampsia. *Lancet* 356: 1260-1265.
- Duckitt K, Harrington D (2005). Risk factors for pre-eclampsia at antenatal booking: systematic review of controlled studies. *BMJ.* 2005 Mar 12; 330 (7491): 565. Epub 2005 Mar 2.
- Silva LM, Coolman M, Steegers EAP, Jaddoe VWV, Moll HA, Hofman A, Mackenbach J P, Raat H (2008). Low socioeconomic status is a risk factor for pre-eclampsia: the Generation R Study, *J Hypertens* 26:1200-1208.
- Villar J, Carroli G, Wojdyla D, Abalos E, Giordano D, Ba'aqueel H (2006). The World Health Organization Antenatal Care Trial Research Group. Pre-eclampsia, gestational hypertension and intrauterine growth restriction, related or independent conditions? *Am J Obstet Gynecol* 194: 921-931.
- Klemmensen AK, Olsen SF, Østerdal ML, Tabor A (2007). Validity of pre-eclampsia-related diagnoses recorded in a national hospital registry and in a postpartum interview of the women, *Am J Epidemiol.* 166: 117-124.
- Roberts CL, Algert CS, Morris JM, Ford JB, Henderson-Smart DJ (2005). Hypertensive disorders in pregnancy: a population-based study, *MJA* 182: 332-335.
- Obed SA, Aniteye P (2006). Birth weight and Ponderal index in Pre-Eclampsia; A comparative study. *Ghana Med J.* 40: 8-13.
- Frank KA, Buchmann EJ, Schackis RC (2004). Does Human Immunodeficiency Virus Infection Protect Against Pre-eclampsia-Eclampsia? *Obstetrics & Gynecology.* 104 (2): 238-242.
- Conde-Agudelo A, Villar J, Lindheimer M (2008). Maternal infection and risk of pre-eclampsia: systematic review and metaanalysis. *Am J Obstet Gynecol.* 198 (1): 7-22.
- Soroori ZZ, Gharami SH, Faraji R (2007). Seasonal variation of the onset of preeclampsia and eclampsia. *Journal of Research in Medical Sciences;* 12(4): 198-202.
- Immink A, Scherjon S, Wolterbeek R, Steyn DW (2008). Seasonal influence on the admittance of pre-eclampsia patients in Tygerberg Hospital. *Acta Obstet Gynecol Scand.* 87 (1): 36-42.
- Okafor UV, Ezegwui HU (2010). Cesarean delivery in pre-eclampsia and seasonal variation in a tropical rainforest belt. *Journal of postgraduate medicine* 56 (1): 21-23.
- Clark P, Wu O (2008). ABO (H) blood groups and pre-eclampsia. A systematic review and meta-analysis. *Thromb Haemost.* 100 (3): 469-74.
- Hiltunen ML, Laivuori H, Rautanen A, Kaaja R, Kere J, Krusius T, Paunio M, Rasi V (2009). Blood group AB and factor V Leiden as risk factors for pre-eclampsia: A population-based nested case-control study *Thrombosis Research.* 124 (2): 167-173.
- Xiong XU, Fraser WD, Demianczuk NN (2002). History of abortion, preterm, term birth, and risk of pre-eclampsia: A population-based study. *American Journal of Obstetrics & Gynecology* 187 (4) 1013-1018.
- Trogstad L, Per M, Skjærven R, Stoltenberg C (2008). Previous abortions and risk of pre-eclampsia. *Int J Epidemiol.* 37(6): 1333-1340.

29. Shamsi U, Hatcher J, Shamsi A, Nadeem Z, Zeeshan Q, Saleem S (2010). A multi centre matched case control study of risk factors for pre-eclampsia in healthy women in Pakistan. *BMC Women's Health* 10: 14.
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THE ASSOCIATION BETWEEN CHRONIC KIDNEY DISEASE, HYPERURICAEMIA AND PROTEINURIA IN ADULT PATIENTS ATTENDING OUTPATIENT CLINICS IN BANJUL, THE GAMBIA

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Abstract

Background: Chronic kidney disease (CKD) is manifested by irreversible worsening renal function and is associated with proteinuria and hyperuricaemia.

Objective: To determine the prevalence of CKD, hyperuricaemia and proteinuria and explore the relationship between CKD, hyperuricaemia and proteinuria among outpatients in Banjul, The Gambia.

Design: Prospective cross-sectional study

Setting: Outpatient clinics of Edward Francis Small Teaching Hospital and Medical Research Council Laboratories in Banjul.

Methods: Two hundred and eight consecutive patients with hypertension on treatment and 108 non-hypertensive patients aged over 25 years were enrolled. A questionnaire was filled and anthropometric measurements were taken. An oral glucose tolerance test was done. Serum uric acid and creatinine were determined from venous blood samples and proteinuria was determined by urine dipsticks. The estimated glomerular filtration rate (GFR) was calculated using

the Cockcroft and Gault equation. CKD was defined and classified by The National Kidney Foundation's Kidney Diseases Outcomes Quality Initiative guidelines.

Results: The results of 300 participants were included in this analysis. The prevalence of hyperuricaemia was 36%, proteinuria 25% and CKD 41% (10.7% of participants had Stage 1, 6.7% Stage 2, 21.7% Stage 3, 1.3% Stage 4 and 0.3% Stage 5). The mean uric acid was 0.33 (0.13) mmol/L, mean creatinine 88.1 (54.1) μ mol/L and mean GFR was 103.2 (80.2) ml/min/1.73 m². There was a strong and significant association between hyperuricaemia, proteinuria and CKD among these participants before and even after controlling for age, sex, hypertension and diabetes mellitus.

Conclusion: The prevalence of CKD, hyperuricaemia and proteinuria in patients attending clinics in Banjul was high. There was a strong and significant association between CKD, hyperuricaemia and proteinuria.

Key Words: Systemic Hypertension, Chronic Kidney Disease, Renal Failure, Hyperuricaemia, Proteinuria

Introduction

Chronic kidney disease (CKD) is one of the major causes of mortality and morbidity in the developing as well as the developed world¹⁻⁴. The incidence and prevalence of CKD is on the increase and this is especially as a result of the epidemic of hypertension and type II diabetes mellitus (DM) which is occurring worldwide and is consequently fueling this increase^{5,6}. There is therefore the need for increased screening and early detection of renal disease generally but especially among hypertensive and DM patients. This is especially crucial in sub-Saharan Africa where there are very few facilities for treatment of CKD and treatment costs are prohibitive^{7,8}. The current guidelines recommend screening for CKD using the estimated glomerular filtration rate (GFR) after

determining serum creatinine levels⁹.

CKD is defined as irreversible, substantial and long-standing loss of renal function. Albuminuria defined as urine albumin-creatinine ratio ≥ 30 mg/g, is a diagnostic component of CKD¹⁰. This is particularly in diabetic nephropathy which in the incipient phase is characterised by microalbuminuria (30-300 mg/day) and is followed by the phase of overt proteinuria (>300 mg/day). In DM patients, microalbuminuria is now known as the earliest marker of diabetic nephropathy and is currently the recommended screening test¹¹. In hypertensive patients with and without DM, microalbuminuria is a risk factor for hypertensive target end organ damage including kidney disease and is associated with progression to end-stage renal disease¹². Microalbuminuria is also a marker of increased risk for the development of hypertension in normotensives¹³. CKD is thus associated with proteinuria and examining urine for protein is important in screening for CKD.

Hyperuricaemia is associated with confirmed risk factors for CKD such as DM, hypertension and metabolic syndrome¹⁴. Severe renal failure of any aetiology may be associated with hyperuricaemia and may in the rare instance result in gout. In certain

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instances the severe renal failure is the cause of the hyperuricaemia while the reverse is also true in other instances. There are some studies which have shown an association between hyperuricaemia and CKD while others have demonstrated otherwise. Wang et al found hyperuricaemia to be a risk factor for CKD in a cohort study involving 94,422 Taiwanese participants¹⁴. In another Taiwanese study See et al found only a weak association between hyperuricaemia and renal function while hyperuricaemia was strongly associated with metabolic syndrome¹⁵. The prevalence of gout has been shown to increase with progressing stages of CKD in the US population from National Health and Nutrition Examination Surveys data¹⁶.

There are very few published studies on renal function, CKD, uric acid and urine protein in The Gambia. From a 1996-97 community study conducted in Banjul and Farafenni, van der Sande et al reported mean uric acid and creatinine as well as prevalence of high creatinine and uric acid as part of various reports¹⁷⁻¹⁹. Recently de la Cruz and others have reported on 69 terminal CKD patients they screened for haemodialysis in Banjul. Mean creatinine for these patients was 1425.6 (366.1) $\mu\text{mol/L}$ and 13% had proteinuria.

As part of our study to determine the relationship between left ventricular hypertrophy and insulin resistance, we determined creatinine, uric acid and urine protein in hypertensive and non-hypertensive Gambians who were seen at outpatient clinics^{20,21}. Our main objective for this current study was to determine the prevalence of CKD, hyperuricaemia and proteinuria and also explore the relationship between CKD, hyperuricaemia and proteinuria among patients attending outpatient clinics in Banjul, The Gambia.

Materials and Methods

This was a prospective cross sectional study conducted from January to May 2000. The participants were recruited from the Edward Francis Small Teaching Hospital (EFSTH), Banjul and Medical Research Council (MRC) Laboratories, Fajara, The Gambia. Patients with systemic hypertension who were seen at the hypertension clinic of EFSTH were consecutively recruited into the study. At the Gate Clinic of the MRC Laboratories, patients who reported with minor infectious diseases who had no cardiovascular disease or DM who in addition did not have hypertension were recruited as the non-hypertensives. The exclusion criteria for this study were severe inter-current illnesses, systemic or metabolic diseases, cardiovascular disease (excluding hypertension) or labile hypertension and morbid obesity ($\text{BMI} > 35 \text{ kg} / \text{m}^2$). Known cases of DM were excluded from the study but those who were diagnosed after undergoing an oral glucose tolerance test (OGTT) were included.

A field worker administered a questionnaire using the appropriate local language and this was followed

by a physical examination undertaken by one physician. The weight of participants wearing light clothes and without footwear was measured using an electric scale (Secca[†] 770, CMS London). Height was measured to the nearest 0.5 cm after participants have removed their footwear and head gear or cap using standardised stadiometers. A plastic tape measure was used to record hip and waist circumferences to the nearest 0.5 cm. The blood pressure was measured on the left arm using a digital blood pressure machine (Omron[†] HOM – 705 CP, Japan). Three readings were taken and the mean of the later two readings was used in the analysis²².

The participants after recruitment reported back the following morning after an overnight fast for urine examination, blood sampling, electrocardiogram and echocardiogram. The patient first collected about 10 to 20mls of urine in a sterile wide-necked leak proof urine specimen container. This was immediately tested for urine protein with Albustix urine dipsticks (Bayer AG, Germany). The results were read and recorded as no proteinuria, 1+ proteinuria, 2+ proteinuria or 3+ proteinuria. Venous blood samples were then collected and analysed for uric acid and creatinine at the MRC Biochemistry Laboratory using a centrifugal biochemical analyzer (Cobas Fara, Roche, UK). Afterwards an OGTT was performed utilising 75g anhydrous glucose in 300 - 350 ml of water. The glucose levels on a fasting, 30 min and 120 min samples were determined immediately upon taking the samples using a Haemocue analyser (Haemocue AB, Sweden). The complete results of the OGTT is in the process of being reported in another article but the results were used in classifying the participants into those with and without DM.

The following definitions were adopted for this study. Hypertension was defined as systolic blood pressure ≥ 140 and / or diastolic blood pressure $\geq 90 \text{ mmHg}$ in subjects who are not taking antihypertensive medication²³. Overall Obesity was defined as Body Mass Index ($\text{BMI} \geq 30 \text{ kg} / \text{m}^2$) while Central Obesity or High Waist Hip Ratio (WHR) was defined as $\text{WHR} > 0.9$ for males and > 0.8 for females²⁴. And DM was defined as fasting venous plasma glucose ($\text{FPG} \geq 7.0 \text{ mmol/L}$) and or 2h post glucose capillary whole blood $\geq 11.1 \text{ mmol/L}$ ^{25,26}. Hyperuricaemia was defined as uric acid level $\geq 0.36 \text{ mmol/L}$ in females and $\geq 0.42 \text{ mmol/L}$ for males²⁷. Proteinuria was defined as any proteinuria on urine dipstick and this included 1+ proteinuria, 2+ proteinuria and 3+ proteinuria.

The estimated glomerular filtration rate (GFR) was calculated using the Cockcroft and Gault equation; $\text{GFR} (\text{ml}/\text{min}/1.73 \text{ m}^2) = 1.23 (140 - \text{age}) \times \text{weight} (\text{kg}) / \text{Plasma creatinine} (\mu\text{mol/l})$ for males and $\text{GFR} (\text{ml}/\text{min}/1.73 \text{ m}^2) = 1.04 (140 - \text{age}) \times \text{weight} (\text{kg}) / \text{Plasma creatinine} (\mu\text{mol/l})$ for females²⁸. The National

Kidney Foundation's Kidney Diseases Outcomes Quality Initiative (NKF KDOQI) guidelines were used in defining and classifying CKD. This classification is based on GFR and the presence or absence of kidney damage. The reduced GFR and or kidney damage must be present for more than 90 days to establish chronicity. In the absence of past data on GFR or markers of kidney damage, chronicity is inferred from clinical presumption of kidney disease for more than 3 months. Based on this assumption CKD was classified into Stage 1 GFR > 90 ml/min/1.73 m² and albuminuria, Stage 2 GFR 60 - 89 ml/min/1.73 m² and albuminuria, Stage 3 GFR 30 - 59 ml/min/1.73 m², Stage 4 GFR 15 - 29 ml/min/1.73 m² and Stage 5 GFR < 15 ml/min/1.73 m² 29.

The data was analysed using Stata version 8.0 statistical package and Microsoft Excel 2007. The mean and standard deviation were calculated for continuous variables, and were compared using the Student t-test. Percentages were calculated for discrete variables and these were compared using Pearson Chi-square test. The participants were classified further into normotensives with and without DM and hypertensives with and without DM and these four subgroups were captioned as the clinical group. CKD was classified into stage 1 to stage 5 and proteinuria into four subgroups (Group 0 – No proteinuria, Group 1 – 1+ proteinuria, Group 2 – 2+ proteinuria and Group 3 – 3+ proteinuria). One-way analysis of variance was used in the analysis of the continuous variables in the different subgroups of the clinical group, CKD and proteinuria. The results of FPG, GFR and creatinine were not normally distributed so a logarithmic transformation was done and this was used in all further analysis using univariate and multivariate linear and logistic regression. For the purpose of logistic regression analysis proteinuria was reclassified into two subgroups, no proteinuria and any proteinuria. P-values of less than 0.05 were taken as statistically significant.

The study was approved by The Gambia Government / MRC Ethical Committee. All the participants gave a formal consent by signing or thumb printing an informed consent form after careful consideration and explanation.

Results

From outpatient clinics 208 consecutive patients (138 females) with systemic hypertension on treatment and 108 non-hypertensive patients (69 females) were enrolled for our initial study^{20,21} but only 300 (194 hypertension, 198 females) of these patients were included in this analysis. Ten participants had no results for both creatinine and uric acid while 6 had no urine protein results and were therefore excluded from the analysis. The mean (\pm standard deviation (sd)) age of the participants was 53.5 (12.0) years. Table 1 show the characteristics of hypertension and normotensive patients.

Table 1: The clinical characteristics of the participants

	Hypertension (n=194)	Normal (n=106)	All (n=300)	P
Parameter				
	Number (%)	Number (%)	Number (%)	
Sex				
Male	63 (32.5)	39 (36.8)	102 (34.0)	0.45
Female	131 (67.5)	67 (63.2)	198 (66.0)	0.45
Age range (years)	27 – 85	30 – 80	27 - 85	
BMI \geq 30	60 (30.9)	18 (17.0)	78 (26.0)	< 0.01
High WHR	153 (78.9)	62 (58.5)	215 (71.7)	< 0.001
DM	35 (18.0)	8 (7.6)	43 (14.3)	0.01
	Mean \pm sd	Mean \pm sd	Mean \pm sd	
Age (years)	55.2 (11.6)	50.5 (12.1)	53.5 (12.0)	< 0.01
Weight (kg)	73.5 (16.2)	66.3 (13.3)	71.0 (15.6)	< 0.001
Height (m)	1.64 (0.08)	1.65 (0.09)	1.64 (0.08)	0.44
BMI (kg/m ²)	27.5 (6.2)	24.6 (5.4)	26.5 (6.1)	< 0.001
Waist circumference (cm)	95.4 (12.5)	87.8 (11.9)	92.7 (12.8)	< 0.0001
Hip circumference (cm)	106.9 (12.8)	103.2 (12.0)	105.6 (12.6)	0.02
Waist-Hip ratio	0.89 (0.06)	0.85 (0.07)	0.88 (0.06)	< 0.0001
SBP (mmHg)	147.9 (27.1)	115.9 (13.1)	136.6 (27.7)	< 0.0001
DBP (mmHg)	88.8 (13.5)	72.0 (8.4)	82.9 (14.4)	< 0.0001

They were similar in the proportion of males and females but there were significant differences in all the other parameters with the hypertension patients having significantly higher values. Mean FPG was 5.7 (2.4) mmol/L, mean uric acid 0.33 (0.13) mmol/L, mean creatinine 88.1 (54.1) μ mol/L and mean GFR was 103.2 (80.2) ml/min/1.73 m² (Table 2). Mean FPG was similar in the normal and hypertension groups while it was significantly higher in the DM and DM - hypertension groups. There were significant differences in the mean uric acid in the different clinical groups, the highest in the DM - hypertension group and the lowest in the normal group. The

differences in creatinine and GFR in the clinical groups were not statistically significant.

Table 3 shows the GFR group of participants by the degree of proteinuria. There were no significant differences in the various GFR groups except the 30 – 59 ml/min/1.73 m² which included the only 2 participants with proteinuria of 3+. In the GFR group of > 90 ml/min/1.73 m², there were 28 participants with proteinuria of 1+ and 4 participants with 2+ and these 32 (10.7%) were classified as CKD stage 1 while in the 60 – 89 ml/min/1.73 m² group there were 16 participants with proteinuria 1+ and 4 with 2+ (20 (6.7%)) who were classified as CKD stage 2. Sixty five (21.7%) participants were classified as CKD stage 3, 4 (1.3%) as CKD stage 4 and 1 (0.3%) as CKD stage 5. Overall 122 (40.7%) of the study population had CKD. One hundred and seven (35.7%) of participants had hyperuricaemia and 76 (25.3%) had some degree of proteinuria. Table 4 shows that the patients with hypertension with and without DM had significantly higher prevalence of hyperuricaemia. There were no significant differences in the prevalence of CKD in the different clinical groups while proteinuria was significantly common in the hypertension and DM - hypertension groups. The 4 participants with stage 4 and the single participant with stage 5 CKD were all hypertension patients. From Table 5 there were several significant associations between the various variables. The clinical group was significantly associated with hyperuricaemia, uric acid and proteinuria but not with CKD, creatinine and GFR.

Hyperuricaemia was significantly associated with CKD, creatinine and GFR but not with proteinuria. CKD was associated with proteinuria and uric acid while proteinuria was significantly associated with uric acid and creatinine but not with GFR.

The results from the univariate linear regression analysis (Table 6) were similar but not identical to the results from Table 5. There were significant association between uric acid and proteinuria, log of creatinine and log of GFR. Log of creatinine and log of GFR were both significantly associated with proteinuria, hyperuricaemia and uric acid. All these associations were still significant after controlling for age, sex, hypertension and DM in multivariate analysis (Table 7). Table 8 shows the results of univariate logistic regression analysis with proteinuria and hyperuricaemia as the outcome variables. Proteinuria was significantly associated with uric acid, log of creatinine and log of GFR but the relationship with hyperuricaemia was not up to statistical significance. There were significant association between hyperuricaemia and log of creatinine and log of GFR but the association with proteinuria was not statistically significant. After controlling for age, sex, hypertension and DM, the association between proteinuria and both uric acid and hyperuricaemia were not significant. The association between proteinuria and log of creatinine and log of GFR on one hand and the association between hyperuricaemia and log of creatinine and log of GFR on the other hand were both significant in multivariate analysis.

Table 2: Mean FPG, uric acid, creatinine and GFR by clinical group

	Normal (n=98)	DM (n=8)	Hypertension (n=159)	DM-Hypertension (n=35)	All (n=300)	P
FPG (mmol/L)	5.1 (0.6)	12.4 (9.4)	5.2 (0.7)	8.0 (3.3)	5.7 (2.4)	< 0.0001
Uric Acid (mmol/L)	0.29 (0.09)	0.35 (0.16)	0.35 (0.13)	0.39 (0.18)	0.33 (0.13)	< 0.001
Creatinine (µmol/L)	80.4 (19.6)	89.9 (22.4)	92.3 (64.1)	90.5 (72.3)	88.1 (54.1)	0.39
GFR (ml/min/1.73 m ²)	90.4 (32.1)	86.0 (31.5)	107.3 (99.7)	124.6 (78.0)	103.2 (80.2)	0.12

Table 3: GFR group by proteinuria

Proteinuria \ GFR	0 (n=224)	1+ (n=62)	2+ (n=12)	3+ (n=2)	All (n=300)	P
> 90	116 (51.8)	28 (45.2)	4 (33.3)	0 (0)	148 (49.3)	0.25
60 - 89	62 (27.7)	16 (25.8)	4 (33.3)	0 (0)	82 (27.3)	0.79
30 - 59	44 (19.6)	15 (24.2)	4 (33.3)	2 (100.0)	65 (21.7)	0.03
15 - 29	2 (0.9)	2 (3.2)	0 (0)	0 (0)	4 (1.3)	0.53
< 15	0 (0)	1 (1.6)	0 (0)	0 (0)	1 (0.3)	0.28

Table 4: Prevalence of hyperuricaemia, CKD and proteinuria by clinical group

	Normal (n=98)	DM (n=8)	Hypertension (n=159)	DM+ Hypertension (n=35)	All (n=300)	P
Hyperuricaemia	14 (14.3)	3 (37.5)	74 (46.5)	16 (45.7)	107 (35.7)	< 0.001
CKD						
Stage 1	4 (4.1)	0 (0.0)	20 (12.6)	8 (22.9)	32 (10.7)	0.10
Stage 2	5 (5.1)	0 (0.0)	11 (6.9)	4 (11.4)	20 (6.7)	0.52
Stage 3	17 (17.4)	3 (37.5)	39 (24.5)	6 (17.1)	65 (21.7)	0.33
Stage 4	0 (0)	0 (0)	3 (1.9)	1 (2.9)	4 (1.3)	0.49
Stage 5	0 (0)	0 (0)	1 (0.6)	0 (0)	1 (0.3)	0.83
All Stages	26 (26.5)	3 (37.5)	74 (46.5)	19 (54.3)	122 (40.7)	< 0.01
Proteinuria						
0	87 (88.8)	8 (100)	110 (69.2)	19 (54.3)	224 (74.7)	< 0.0001
1	9 (9.2)	0 (0)	40 (25.2)	13 (37.1)	62 (20.7)	< 0.001
2	2 (2.0)	0 (0)	7 (4.4)	3 (8.6)	12 (4.0)	0.35
3	0 (0)	0 (0)	2 (1.3)	0 (0)	2 (0.7)	0.62

Table 5: The association between clinical group, hyperuricaemia, CKD and proteinuria and various variables

	Clinical Group	Hyperuricaemia	CKD	Proteinuria
	P	P	P	P
Sex	0.24 (χ^2)	0.88 (χ^2)	< 0.001 (χ^2)	0.39 (χ^2)
BMI \geq 30	0.02 (χ^2)	0.30 (χ^2)	0.03 (χ^2)	0.49 (χ^2)
High WHR	< 0.01 (χ^2)	0.86 (χ^2)	< 0.01 (χ^2)	0.35 (χ^2)
Hypertension	N/A	< 0.001 (χ^2)	< 0.01 (χ^2)	< 0.001 (χ^2)
DM	N/A	0.21 (χ^2)	0.42 (χ^2)	0.20 (χ^2)
Clinical group	N/A	< 0.001 (χ^2)	0.07 (χ^2)	< 0.01 (χ^2)
Hyperuricaemia	< 0.001 (χ^2)	N/A	< 0.001 (χ^2)	0.07 (χ^2)
CKD	0.07 (χ^2)	< 0.001 (χ^2)	N/A	< 0.001 (χ^2)
Proteinuria	< 0.01 (χ^2)	0.07 (χ^2)	< 0.001 (χ^2)	N/A
Age	< 0.01 (F)	0.03 (t)	< 0.001 (F)	0.03 (F)
Weight	< 0.001 (F)	< 0.001 (t)	< 0.0001 (F)	0.22 (F)
Height	0.72 (F)	0.60 (t)	0.10 (F)	0.06 (F)
BMI	< 0.001 (F)	< 0.01 (t)	< 0.0001 (F)	0.08 (F)
Waist circumference	< 0.0001 (F)	< 0.001 (t)	< 0.0001 (F)	0.09 (F)
Hip circumference	0.02 (F)	< 0.01 (t)	< 0.0001 (F)	0.16 (F)
Waist-Hip ratio	< 0.0001 (F)	0.04 (t)	0.96 (F)	0.41 (F)
SBP	< 0.0001 (F)	< 0.001 (t)	< 0.001 (F)	< 0.0001 (F)
DBP	< 0.0001 (F)	< 0.01 (t)	0.02 (F)	< 0.0001 (F)
FPG	< 0.0001 (F)	0.09 (t)	0.92 (F)	0.80 (F)
Uric Acid	< 0.001 (F)	N/A	< 0.0001 (F)	< 0.01 (F)
Creatinine	0.39 (F)	< 0.0001 (t)	< 0.0001 (F)	< 0.01 (F)
GFR	0.12 (F)	< 0.001 (t)	N/A	0.36 (F)

Table 6: Univariate analysis with uric acid, log of creatinine and log of GFR as the outcome variable

	Uric Acid		Creatinine		GFR	
	r	P	r	P	r	P
Sex	-0.026	0.10	-0.133	0.02	0.343	< 0.001
BMI \geq 30	0.029	0.09	-0.030	0.61	0.400	< 0.001
High WHR	-0.006	0.74	-0.113	0.05	0.301	< 0.001
Hypertension	0.064	< 0.001	0.005	0.93	0.048	0.47
DM	0.053	0.01	-0.042	0.57	0.141	0.13
Proteinuria	0.050	< 0.01	0.154	< 0.01	-0.169	0.02
Hyperuricaemia	N/A	N/A	0.402	< 0.001	-0.351	< 0.001
Age	0.002	< 0.001	0.003	0.12	-0.015	< 0.001
Weight	0.001	< 0.01	-0.002	0.30	0.016	< 0.001
Height	0.128	0.17	0.261	0.41	-0.138	0.73
BMI	0.003	0.02	-0.005	0.21	0.039	< 0.001
Waist circumference	0.002	< 0.01	-0.003	0.13	0.018	< 0.001
Hip circumference	0.001	0.08	-0.003	0.11	0.020	< 0.001
Waist-Hip ratio	0.276	0.02	-0.067	0.87	0.262	0.60
SBP	0.001	< 0.001	0.002	< 0.01	-0.004	< 0.01
DBP	0.002	< 0.001	0.003	0.07	-0.002	0.41
FPG	0.070	0.02	0.021	0.84	-0.003	0.98
Uric Acid	N/A	N/A	2.288	< 0.001	-2.293	< 0.001
Creatinine	0.194	< 0.001	N/A	N/A	N/A	N/A
GFR	-0.126	< 0.001	N/A	N/A	N/A	N/A

Table 7: Multivariate analysis with uric acid, log of creatinine and log of GFR as the outcome variable adjusting for age, sex, DM and hypertension

	Uric Acid		Creatinine		GFR	
	r	P	r	P	r	P
Proteinuria	0.033	0.05	0.176	< 0.01	-0.222	< 0.01
Hyperuricaemia	N/A	N/A	0.436	< 0.001	-0.373	< 0.001
Uric acid	N/A	N/A	2.459	< 0.001	-2.251	< 0.001
Creatinine	0.191	< 0.001	N/A	N/A	N/A	N/A
GFR	-0.137	< 0.001	N/A	N/A	N/A	N/A

Table 8: Univariate analysis with proteinuria and hyperuricaemia as the outcome variable

	Proteinuria			Hyperuricaemia		
	OR	CI	P	OR	CI	P
Sex	1.48	0.48-2.63	0.18	0.96	0.58-1.58	0.88
BMI \geq 30	1.22	0.68-2.18	0.50	1.82	1.08-3.09	0.03
High WHR	1.38	0.75-2.51	0.30	0.95	0.56-1.61	0.86
Hypertension	4.35	2.18-8.69	< 0.001	4.53	2.51-8.18	< 0.001
DM	1.95	0.98-3.85	0.06	1.52	0.79-2.93	0.21
Proteinuria	N/A	N/A	N/A	1.67	0.98-2.85	0.06
Hyperuricaemia	1.67	0.98-2.85	0.06	N/A	N/A	N/A
Age	1.01	0.99-1.03	0.27	1.02	1.00-1.04	0.03
Weight	1.00	0.98-1.01	0.83	1.03	1.01-1.04	< 0.01
Height	0.03	0.00-0.73	0.03	2.20	0.12-39.71	0.59
BMI	1.02	0.98-1.06	0.41	1.07	1.02-1.11	< 0.01
Waist circumference	1.01	0.99-1.03	0.55	1.03	1.01-1.06	< 0.01
Hip circumference	1.00	0.98-1.02	0.93	1.03	1.01-1.05	< 0.01
Waist-Hip ratio	7.58	0.13-443.83	0.33	50.48	1.15-2224.93	0.04
SBP	1.02	1.01-1.03	< 0.001	1.02	1.01-1.03	< 0.001
DBP	1.04	1.02-1.06	< 0.001	1.03	1.01-1.05	< 0.01
FPG	2.16	0.83-5.66	0.12	2.15	0.85-5.47	0.11
Uric Acid	18.17	2.42-136.53	< 0.01	N/A	N/A	N/A
Creatinine	2.21	1.20-4.08	0.01	22.07	8.69-56.05	< 0.001
GFR	0.57	0.35-0.93	0.02	0.26	0.15-0.44	< 0.001

Table 9: Multivariate analysis with proteinuria and hyperuricaemia as the outcome variable adjusting for age, sex, DM and hypertension

	Proteinuria			Hyperuricaemia		
	OR	CI	P	OR	CI	P
Proteinuria	N/A	N/A	N/A	1.17	0.66-2.06	0.59
Hyperuricaemia	1.18	0.67-2.07	0.57	N/A	N/A	N/A
Uric Acid	6.65	0.82-53.94	0.08	N/A	N/A	N/A
Creatinine	2.19	1.20-3.99	0.01	26.67	9.73-73.13	< 0.001
GFR	0.45	0.26-0.78	< 0.01	0.19	0.10-0.36	< 0.001

Discussion

This study has shown that the prevalence of hyperuricaemia in these participants was 36%, that of proteinuria was 25% and CKD 41% though stages 4 and 5 CKD was only 2%. The mean uric acid was 0.33 (0.13) mmol/L, mean creatinine 88.1 (54.1) μ mol/L and mean GFR was 103.2 (80.2) ml/min/1.73 m². There was also a strong and significant association between uric acid, proteinuria and CKD among these subjects before and even after controlling for age, sex, hypertension and DM.

In the previous Gambian study the following results were obtained, mean uric acid 0.32 (0.08) mmol/L in urban men, 0.25 (0.07) mmol/L in urban women, 0.30 (0.06) mmol/L in rural men and 0.21 (0.06) mmol/L in rural women. Mean creatinine was 82.6 (49.8) μ mol/L in urban men, 66.0 (37.4) μ mol/L in urban women, 79.8 (18.6) μ mol/L in rural men and

60.7 (21.4) μ mol/L in rural women. The prevalence of hyperuricaemia was as follows; 7.4% in urban men, 8.5% in urban women, 1.4% in rural men and 1.3% in rural women while the prevalence of elevated creatinine (defined as creatinine \geq 90 μ mol/L for women or \geq 100 μ mol/L for men) were 15.3%, 8.2%, 13.0% and 1.4% for urban men, urban women, rural men and rural women respectively. These mean levels and prevalence levels for the whole population were in all instances lower than that of the hypertensives. In the hypertensive population mean uric acid was 0.36 (0.10) mmol/L in urban men, 0.29 (0.10) mmol/L in urban women, 0.35 (0.07) mmol/L in rural men and 0.23 (0.07) mmol/L in rural women while mean creatinine was 95.0 (88.4) μ mol/L in urban men, 80.5 (73.4) μ mol/L in urban women, 95.6 (18.4) μ mol/L in rural men and 71.8 (32.9) μ mol/L in rural women. The prevalence of hyperuricaemia among the hypertensives

was 18.4%, 28.1%, 6.8% and 6.9% while the prevalence of elevated creatinine was 22.2%, 10.9%, 27.0% and 6.9% for urban men, urban women, rural men and rural women respectively¹⁷⁻¹⁹. These results are similar but not identical to the findings of our present study and further comparison by formal statistical testing is also not possible.

Serum uric acid concentration is maintained through the synthesis and excretion of urate with approximately 70% of uric acid being excreted from the kidneys. Creatinine has been shown to have a strong influence on uric acid levels by Choe et al.³⁰ while an independent relationship of creatinine and uric acid has been reported by Rathmann and others³¹. Hyperuricaemia has been shown to be an independent risk factor for renal dysfunction in the normal population as well as in patients with CKD, DM and hypertension³²⁻⁴¹. In animal studies, hyperuricaemia has been shown to be associated with the development of mild renal disease which is characterised by mild proteinuria⁴². Other animal studies have also shown that hyperuricaemia may not only cause new onset renal disease but may worsen already existent renal disease³⁹. Clinical studies have demonstrated that lowering uric acid in renal disease patients with asymptomatic hyperuricaemia with allopurinol led to less and slower progression of the renal disease⁴³. Other studies have also shown that cessation of allopurinol treatment in patients with CKD resulted in a significant deterioration of renal function^{44,45}. The results of our study have shown this association between uric acid and renal function, before and after adjusting for age, sex, hypertension and DM.

Hyperuricaemia therefore has a direct effect on renal function and an indirect effect on urine protein through its effect on the kidneys. Several studies on DM patients have illustrated this relationship between hyperuricaemia and proteinuria. In a study of type 2 DM patients, a significant association was found between hyperuricaemia and serum creatinine and eGFR. In the same study serum uric acid levels was positively correlated to the urinary albumin-creatinine ratio and this relationship remained significant after adjusting for eGFR⁴⁶. Tseng also found serum uric acid to be independently correlated to urinary albumin-creatinine ratio in type 2 DM Taiwanese patients³⁴ while Fukui et al demonstrated the same association in Japanese men with type 2 DM⁴⁷. In Italy Bo et al found uric acid to be associated independently with macroalbuminuria⁴⁸ while Bruno et al established this independent association with both micro- and macroalbuminuria⁴⁹. In type 1 DM, the level of uric acid early in the course of diabetes was demonstrated to be independently and significantly associated with later development of persistent macroalbuminuria but not persistent microalbuminuria³⁷. In our study the association between uric acid and proteinuria was significant in univariate but not in multivariate analysis controlling for age, sex, hypertension and DM.

Most of these DM studies have also demonstrated the relationship between renal function and proteinuria. Eghan and others found creatinine and blood urea nitrogen to be significantly higher in type 2 DM patients with microalbuminuria in Kumasi, Ghana⁵⁰. In Dar es Salaam, Tanzania, Lutale et al found serum creatinine to be independently associated with urine albumin concentration measured as average albumin excretion rate in multiple linear regression analysis⁵¹. The relationship between microalbuminuria and renal function has also been demonstrated in normotensives and hypertension patients with hypertensive target end organ damage⁵². There was a significant association between renal function and proteinuria in these Gambians we studied with urine protein detected with urine dipsticks.

The major strength of our study is that this is one of the few renal studies which have been undertaken in The Gambia. There has been no previous study in The Gambia which sought to determine the relationship between renal function, uric acid and proteinuria. This study has shown a strong association between renal function, proteinuria and uric acid in these participants. Potential limitations of this study include the hospital based cross sectional design which is fraught with biases such as proximity and selection biases. Also instead of only measuring urine protein by using urine dipsticks the ideal would have been to determine the urinary albumin-creatinine ratio or albumin excretion rate on a morning urinary sample or a 24-hour urine sample. However since this was not the primary objective of the original study this was not measured. Further, some participants may have produced a first morning urine sample since the participants were seen early in the morning for the urine examination, but we did not inquire to confirm this neither did we take any measures to rule out ambulatory or orthostatic proteinuria. There is therefore the need generally for more cardiovascular studies, preferably large community based studies in The Gambia and specifically ones that would explore further the relationship between renal function, uric acid and proteinuria.

Conclusion

The prevalence of hyperuricaemia, proteinuria and CKD was high in The Gambia. There was a significant association between renal function, uric acid and proteinuria in these participants and this supports the suggestion that hyperuricaemia may have a direct effect on renal function and therefore indirectly on proteinuria.

References

1. Aitkins RC. The epidemiology of chronic kidney disease. *Kidney Int Suppl* 2005; 94: S14-S18.
2. El Nahas AM, Bello AK. Chronic kidney disease: the global challenge. *Lancet* 2005; 365: 331-340.

3. Naicker S. End-stage renal disease in sub-Saharan and South Africa. *Kidney Int Suppl* 2003; 83: S119-S122.
4. Mate-Kole MO, Affram RK. Presentation and clinical course of End-stage renal failure in Ghana. A preliminary prospective study. *Ghana Med. J* 1990; 24: 164-168.
5. Plange-Rhule J, Phillips R, Acheampong JW, Saggarr-Malik AK, Cappuccio FP, Eastwood JB. Hypertension and renal failure in Kumasi, Ghana. *J Hum Hypertens*. 1999 Jan; 13:37-40.
6. Haroun MK, Jaar BG, Hoffman SC, et al. Risk factors for chronic kidney disease: a prospective study of 23,534 men and women in Washington County, Maryland. *J Am Soc Nephrol* 2003; 14: 2934-2941.
7. Agaba EI, Lopez A, Ma I, Martinez R, Tzamaloukas, Vanderjagt DJ, Glew RH, Tzamaloukas AH. Chronic haemodialysis in a Nigerian teaching hospital: practice and costs. *Int J Artif Organs* 2003; 26: 991-995.
8. Fogazzi GB, Attolou V, Kadiri S, Fenili D, Priuli F. A nephrological program in Benin and Togo, West Africa. *Kidney Int Suppl* 2003; 83: S56-S60
9. European Best Practice Guidelines Expert Group on Hemodialysis, European Renal Association. *Nephrol Dial Transplant*. 2002; 17 Suppl 7:72-87.
10. Saydah SH, Pavkov ME, Zhang C, Lacher DA, Eberhardt MS, Burrows NR, Narva AS, Eggers PW, Williams DE. Albuminuria prevalence in first morning void compared with previous random urine from adults in the national health and nutrition examination survey, 2009-2010. *Clin Chem*. 2013 Apr; 59(4):675-83. doi: 10.1373/clinchem.2012.195644. Epub 2013 Jan 11.
11. National Institute for Health and Clinical Excellence. CG 66. Type 2 Diabetes: full guidelines. London: NICE, 2008. Accesseed 24 March 2014.
12. Wachtell K, Ibsen H, Olsen MH, Borch-Johnsen K, Lindholm LH, Mogensen CE, Dahlöf B, Devereux RB, Beevers G, de Faire U, Fyhrquist F, Julius S, Kjeldsen SE, Kristianson K, Lederballe-Pedersen O, Nieminen MS, Okin PM, Omvik P, Oparil S, Wedel H, Snapinn SM, Aurup P. Albuminuria and cardiovascular risk in hypertensive patients with left ventricular hypertrophy: the LIFE study. *Ann Intern Med*. 2003; 139:901 – 906.
13. Hillege HL, Janssen WM, Bak AA, Diercks GF, Grobbee DE, Crijs HJ, Van Gilst WH, De Zeeuw D, De Jong PE; Prevend Study Group. Microalbuminuria is common, also in a nondiabetic, nonhypertensive population, and an independent indicator of cardiovascular risk factors and cardiovascular morbidity. *J Intern Med*. 2001; 249:519 – 526.
14. Wang S, Shu Z, Tao Q, Yu C, Zhan S, Li L. Uric acid and incident chronic kidney disease in a large health check-up population in Taiwan. *Nephrology (Carlton)*. 2011 Nov; 16(8):767-76.
15. See LC, Kuo CF, Chuang FH, Li HY, Chen YM, Chen HW, Yu KH. Serum uric acid is independently associated with metabolic syndrome in subjects with and without a low estimated glomerular filtration rate. *J Rheumatol*. 2009; 36:1691-8. doi: 10.3899/jrheum.081199. Epub 2009 Jun 16.
16. Juraschek SP, Kovell LC, Miller ER 3rd, Gelber AC. Association of kidney disease with prevalent gout in the United States in 1988-1994 and 2007-2010. *Semin Arthritis Rheum*. 2013 Jan 8. pii: S0049-0172(12)00266-1
17. van der Sande MA, Walraven GE, Milligan PJ, Banya WA, Ceesay SM, Nyan OA, McAdam KP. Family history: an opportunity for early interventions and improved control of hypertension, obesity and diabetes. *Bull World Health Organ*. 2001; 79:321-8.
18. van der Sande MA, Ceesay SM, Milligan PJ, Nyan OA, Banya WA, Prentice A, McAdam KP, Walraven GE. Obesity and undernutrition and cardiovascular risk factors in rural and urban Gambian communities. *Am J Public Health*. 2001;91:1641-4.
19. van der Sande MA, Milligan PJ, Nyan OA, Rowley JT, Banya WA, Ceesay SM, Dolmans WM, Thien T, McAdam KP, Walraven GE. Blood pressure patterns and cardiovascular risk factors in rural and urban gambian communities. *J Hum Hypertens*. 2000; 14:489-96.
20. Nkum BC, Nyan O, Corrah T, Ankrah TC, Allen S, Micah FB., et al. Resting electrocardiographic and echocardiographic findings in an urban community in the Gambia. *Journal of Science and Technology*. 2009; 29:130-140.
21. Nkum BC, Micah FB, Ankrah TC Nyan O. Left ventricular hypertrophy and insulin resistance in adults from an urban community in The Gambia: cross-sectional study. *PLoS One*. 2014 4; 9:e93606.
22. Mayet J, Shahi M, Foale RA, Poulter NR, Sever PS, McG Thom SA. Racial differences in cardiac structure and function in essential hypertension. *Br Med J*. 1994; 308:1011-1014.
23. Chalmers J, MacMahon S, Mancia G, Whitworth J, Beilin L, Hansson L et al. 1999 World Health Organization-International Society of Hypertension Guidelines for the management of hypertension. Guidelines sub-committee of the World Health Organization. *Clin Exp Hypertens*. 1999 Jul-Aug; 21(5-6):1009-60.
24. Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults--The Evidence Report. National

- Institutes of Health. *Obes Res*. 1998 Sep; 6 Suppl 2:51S-209S.
25. Alberti KG, Zimmet PZ. Definition, diagnosis and classification of diabetes mellitus and its complications. Part 1: diagnosis and classification of diabetes mellitus provisional report of a WHO consultation. *Diabet Med*. 1998; 15:539-53.
 26. World Health Organisation. Definition and diagnosis diabetes mellitus and intermediate hyperglycaemia: Report of a WHO/IDF Consultation, Geneva, Switzerland. 2006.
 27. Harmonisation of Reference Intervals Pathology Harmony Group, Clinical Biochemistry Outcomes, January 2011. Retrieved 24 March 2014.
 28. Cockcroft DW, Gault MH. Prediction of creatinine clearance from serum creatinine. *Nephron* 1976; 16: 31–41.
 29. Anonymous. Kidney Disease Outcome Quality Initiative. Clinical Practice Guidelines for Chronic Kidney Disease: Evaluation, Classification and Stratification. *Am J Kidney Dis* 2002; 39 Suppl 1: S1-S246.
 30. Choe JY, Park SH, Kim JY, Shin IH, Kim SK: Change in serum uric acid between baseline and 1-year follow-up and its associated factors in male subjects. *Clin Rheumatol* 2008, 27:483–489
 31. Rathmann W, Haastert B, Icks A, Giani G, Roseman JM: Ten-year change in serum uric acid and its relation to changes in other metabolic risk factors in young black and white adults: the CARDIA study. *Eur J Epidemiol* 2007, 22:439-445.
 32. Iseki K, Oshiro S, Tozawa M, Iseki C, Ikemiya Y, Takishita S: Significance of hyperuricemia on the early detection of renal failure in a cohort of screened subjects. *Hypertens Res* 24: 691-697, 2001
 33. Segura J, Campo C, Ruilope LM: How relevant and frequent is the presence of mild renal insufficiency in essential hypertension? *J Clin Hypertens* 4: 332-336, 2002
 34. Tseng CH: Correlation of uric acid and urinary albumin excretion rate in patients with type 2 diabetes mellitus in Taiwan. *Kidney Int* 68: 796–801, 2005
 35. Ohno I, Hosoya T, Gomi H, Ichida K, Okabe H, Hikita M: Serum uric acid and renal prognosis in patients with IgA nephropathy. *Nephron* 87: 333–339, 2001
 36. Feig DI, Mazzali M, Kang DH, Nakagawa T, Price K, Kannelis J, Johnson RJ. Serum uric acid: a risk factor and a target for treatment? *J Am Soc Nephrol*. 2006; 17(4 Suppl 2):S69-73.
 37. Hovind P, Rossing P, Johnson RJ, Parving HH. Serum uric acid as a new player in the development of diabetic nephropathy. *J Ren Nutr*. 2011;21:124-7. doi: 10.1053/j.jrn.2010.10.024.
 38. Johnson RJ, Kang DH, Feig D, Kivlighn S, Kanellis J, Watanabe S, Tuttle KR, Rodriguez-Iturbe B, Herrera-Acosta J, Mazzali M. Is there a pathogenetic role for uric acid in hypertension and cardiovascular and renal disease? *Hypertension*. 2003; 41(6):1183-90. Epub 2003 Apr 21.
 39. Kang DH, Nakagawa T, Feng L, Watanabe S, Han L, Mazzali M, Truong L, Harris R, Johnson RJ. A role for uric acid in the progression of renal disease. *J Am Soc Nephrol*. 2002; 13:2888-97.
 40. Heinig M, Johnson RJ. Role of uric acid in hypertension, renal disease, and metabolic syndrome. *Cleve Clin J Med*. 2006; 73:1059-64.
 41. Cirillo P, Sato W, Reungjui S, Heinig M, Gersch M, Sautin Y, Nakagawa T, Johnson RJ. Uric acid, the metabolic syndrome, and renal disease. *J Am Soc Nephrol*. 2006; 17:S165-8.
 42. Nakagawa T, Mazzali M, Kang DH, Kanellis J, Watanabe S, Sanchez-Lozada LG, Rodriguez-Iturbe B, Herrera-Acosta J, Johnson RJ. Hyperuricemia causes glomerular hypertrophy in the rat. *Am J Nephrol*. 2003; 23:2-7.
 43. Siu YP, Leung KT, Tong MK, Kwan TH. Use of allopurinol in slowing the progression of renal disease through its ability to lower serum uric acid level. *Am J Kidney Dis*. 2006; 47:51-9.
 44. Kanbay M, Ozkara A, Selcoki Y, Isik B, Turgut F, Bavbek N, Uz E, Akcay A, Yigitoglu R, Covic A. Effect of treatment of hyperuricemia with allopurinol on blood pressure, creatinine clearance, and proteinuria in patients with normal renal functions. *Int Urol Nephrol*. 2007; 39:1227-33. Epub 2007 Aug 15.
 45. Talaat KM, el-Sheikh AR. The effect of mild hyperuricemia on urinary transforming growth factor beta and the progression of chronic kidney disease. *Am J Nephrol*. 2007; 27(5):435-40. Epub 2007 Jul 4.
 46. Bonakdaran S, Hami M, Shakeri MT. Hyperuricemia and albuminuria in patients with type 2 diabetes mellitus. *Iran J Kidney Dis*. 2011; 5(1):21-4.
 47. Fukui M, Tanaka M, Shiraishi E, Harusato I, Hosoda H, Asano M, Kadono M, Hasegawa G, Yoshikawa T, Nakamura N. Serum uric acid is associated with microalbuminuria and subclinical atherosclerosis in men with type 2 diabetes mellitus. *Metabolism*. 2008 May; 57(5):625-9. doi:10.1016/j.metabol.2007.12.005.
 48. Bo S, Cavallo-Perin P, Gentile L, Repetti E, Pagano G. Hypouricemia and hyperuricemia in type 2 diabetes: two different phenotypes. *Eur J Clin Invest*. 2001 Apr; 31(4):318-21.
 49. Bruno G, Cavallo-Perin P, Bargero G, Borra M, Calvi V, D'Errico N, Deambrogio P, Pagano G. Prevalence and risk factors for micro- and macroalbuminuria in an Italian population-based cohort of NIDDM subjects. *Diabetes Care*. 1996 Jan; 19(1):43-7.

50. Eghan BA Jr, Frempong MT, Adjei-Poku M. Prevalence and predictors of microalbuminuria in patients with diabetes mellitus: a cross-sectional observational study in Kumasi, Ghana. *Ethn Dis.* 2007 autumn; 17(4):726-30.
51. Lutale JJ, Thordarson H, Abbas ZG, Vetvik K. Microalbuminuria among Type 1 and Type 2 Diabetic patients of African origin in Dar Es Salaam, Tanzania. *BMC Nephrol.* 2007 Jan 15;8:2.
52. Halimi J-M, Hadjadj S, Aboyans V, Allaert F-A, Artigou J-Y, Beaufils M, et al. Microalbuminuria and urinary albumin excretion: French clinical practice guidelines. *Diabetes Metab.* 2007; 33(4):303 – 309.
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SPECIAL ARTICLES

IMPACT OF FREE MATERNAL CARE POLICY ON MATERNAL AND CHILD HEALTH INDICATORS IN GHANA

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Abstract

Background: Maternal and child mortality remain major global public health challenges. Majority of the world's maternal mortality occur in low-income countries including Ghana, where financial barriers make maternal healthcare inaccessible to many women during obstetric emergencies, resulting in avoidable maternal deaths. Ghana implemented a free maternal care policy nation-wide in 2008 to provide pregnant women antenatal, delivery and postnatal care in public, and accredited private healthcare facilities. This work assesses the impact of the policy on selected Maternal and Child Health (MCH) indicators in Ghana.

Methods: Literature on financial barriers to maternal healthcare in Low Income Countries (LICs) was reviewed. WHO databases were searched for MCH indicators for Ghana from 2000-2011, aggregated and trends analysed. Additional data was obtained from Maamobi Polyclinic, Koforidua Regional Hospital (KRH), and the Korle Bu Teaching Hospitals (KBTH). These were statistically analysed for trends to assess the policy's impact on these indicators.

Results: Over four years of implementation, average antenatal coverage increased by 2%, skilled birth attendance 11%; contraceptive prevalence unchanged and unmet need for contraception rose marginally. Under-5 mortality declined by 22%. KBTH recorded increased antenatal (ANC) attendance and decreased annual deliveries that were non-significant. Maternal Mortality Rate (MMR) increased by 89/100,000LB; Caesarean section (C/S) rate rose by 5.5%, fresh still birth (FSB) rate increased and Neonatal intensive care unit (NICU) admissions surged 21%. KRH recorded significant increases in deliveries by 2114; C/S rate by 3% while MMR reduced by 0.56% (562/100,000LB). However, the FSB proportion increased by 13%, ANC attendance reduced by 567, annual deliveries rose by 300, C/S rate and FSB increased by 3% and 11% respectively per year at the Maamobi Polyclinic.

Conclusion: Encouraging trends were observed in the MCH indicators attributable to the policy. Increasing FSB rates indicate inadequate care quality especially intra-partum monitoring possibly due to over-stretched staff and facilities from rising patient loads.

Key Words: Free maternal care, Policy, Impact

Introduction

Progress towards the Millennium Development Goals (MDGs) in maternal and child health has been slow in many developing countries including Ghana. According to the World Health Organisation (WHO), about 99% of maternal deaths annually happened in developing countries¹. The cost of a single maternal death to society remains extremely high, necessitating every effort to prevent these avoidable losses. The inability of most women to access prenatal and timely emergency obstetric care (EmOC) remains one major challenge in addressing the burden of maternal mortality worldwide. Timely availability of EmOC has been identified as one important modality for preventing maternal deaths².

Severe pregnancy-related complications leading to disability and long-term illness occur in over 15million women annually, implying that for every mortality, a lot more women suffer severe morbidity. The high numbers of maternal deaths in Sub-Saharan Africa are due to poor maternal health and inadequate care³. UNICEF estimates 15% of childbirths to have complications requiring emergency obstetric care which should readily be available and accessible to all women needing it³. Most maternal deaths occur among women who lack financial access to skilled providers⁵. Patients also suffer delays after arriving at the healthcare facility when prompt life-saving emergency obstetric treatment is not readily available^{4,3}. Substantial socioeconomic disparities have been observed in access to professional delivery care in many low and middle income countries, suggesting a clear poverty gradient to maternal mortality^{6,7}.

Unfortunately however, access to healthcare among women in developing countries is limited by poverty, women inequality, low status, as well as society's attitude towards women and their needs³. Unaffordable healthcare bills remain a major barrier to utilization of maternal and child healthcare services,

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and has adversely affected progress towards attaining the MDGs⁸.

Over the past two decades, charging user fees for primary health care services including maternal and delivery care has been controversial and debated. The World Bank and donor governments have promoted community financing through user charges as part of sector reforms. These user charges potentially give a perceived value to the services provided, and also deter un-necessary demand; and further provide incentives for staff; remove the hidden, unofficial charges by unscrupulous health workers or parallel markets; and ultimately increase use by improving service quality. Practically, many studies have confirmed that demand-for-services by the poor and the vulnerable populations e.g. mothers and children, is not price-inelastic¹⁴. Appreciable declines in the patronage of antenatal care, maternal care, child health, and sexually transmitted diseases have been reported following introduction of user charges. Such reductions in demand were greatest among the most socioeconomically deprived populations.

Major constituents of this financial barrier include cost of paying for medical treatments and transport cost to reach health care facility, which in a Tanzanian study has been estimated to be as high as 50% of the total delivery-related cost. Another component is the financial cost of the days of work (wages) lost to an accompanying relation of the woman to the health facility¹⁵. Healthcare financing is a central determinant of access to skilled delivery care. General tax financing, coupled with national policies for universal coverage have been linked to high service coverage and utilization, and low maternal mortality in Sri Lanka, Malaysia and Kerala, India¹⁵. A recent cross-national study of LICs found a higher proportion of government financing of health was associated with greater utilization of skilled birth attendants. Out-of-pocket payments for delivery services constitute a barrier and inflict substantial financial repercussions on households including women having to borrow money or sell valuable household items to bear these catastrophic healthcare costs¹⁵.

Various interventions have yielded varying levels of success in efforts to remove the financial barriers to skilled delivery services and improve access to emergency obstetric care. These include the “midwife in every village” program and “Jamskesmas” in Indonesia;¹⁶ cash transfers for families with pregnant woman and children in Indonesia¹⁹; public-private partnership for EmOC to women from households below the poverty line in Gujarat, India where the state covered out-of-pocket costs incurred on travel to healthcare facility plus financial support to one accompanying relation’s lost wages¹⁷. In Tanzania, the fee exemption policy introduced in 2001 achieved no improvement in skilled birth attendance because 53.6% of their delivery-related cost was spent on transport which the policy did not cover¹⁵. The use of vouchers

that strengthened demand side for reproductive health services (RHS) by selected families in Kenya, Uganda, Bangladesh and Cambodia resulted in increased utilization of RHS, care quality and population health outcomes²¹.

Background on Ghana and Healthcare Financing in Ghana

In Ghana, healthcare financing has journeyed from free healthcare in the first republic, through the ‘Cash and Carry’ system in the 1980s, till the late 1990s when the move to a national health insurance system started with pilots in two district mutual insurance schemes in the Dangbe East and Nkoranza districts. By 2003 the National Health Insurance Scheme (NHIS) was established and rolled out.

Before the NHIS, ANC was theoretically free but such exemption running alongside a cash and carry system, gave rise to widespread illegal and under-the-table payments. Many pregnant women therefore had to pay for obstetric care, and their inability to afford this denied them access to skilled care.

Additionally, increasing inequalities in access to skilled birth attendance remained a major concern in Ghana. The absolute differences between the bottom and top quintiles in terms of skilled birth attendance increased from 60% in 1993 to 68% in 1998 and nearly 70% in 2003. These compared with an average poor-rich gap of 42% for a selection of sub-Saharan African countries.¹⁰ In order to uniformly improve access to skilled birth attendance and reduce maternal mortality, the policy exempting women attending health facilities from paying delivery care fees was considered^{10, 11}. This policy was piloted in 2003, in the four poorest regions of Ghana - Upper East, Upper West, Northern and Central regions¹⁰. An evaluation of the pilot by Witter et al¹¹ found that delivery exemptions in Ghana can be effective and cost-effective, and that despite being universal in application, they benefitted the poor. It subsequently concluded that the concept was a bold, timely and supported by existing evidence. Realization of the policy’s potential to increase skilled birth attendance however, fundamentally depended on effective implementation¹² Another review of the policy’s impact on institutional deliveries in the Central Region revealed that the delivery-related MMR decreased from 445 to 381 per 100,000LB ($p=0.458$). It concluded that the delivery-related institutional maternal mortality did not appear to have been significantly affected after about one year of policy implementation¹⁰.

In the year 2005, the national MMR in Ghana was an alarming 560 per 100,000LB putting Ghana in the global category of countries with high burden of maternal mortality¹¹. In response, the Health minister declared the situation as a national emergency and highlighted the need for greater prioritisation of reproductive health services. By 2007, an independent review indicated that the proportion of deliveries attended by skilled health staff was at a low 35%;

institutional maternal mortality rates and the proportion of births attended by traditional birth attendants were increasing¹¹.

These posed a huge threat to the Ghana's progress in improving maternal health and reducing child mortality to achieve the targets of the MDGs. The National Health Insurance running quite smoothly and there was increasing availability of funding globally to support initiatives that sought to reduce financial barriers to maternal healthcare. The nation-wide universal free medical care for all pregnant women in Ghana was therefore implemented in May 2008¹¹. The policy allows for all pregnant women at any public, mission or NHIS-accredited private health facility to get registered and receive free comprehensive antenatal, delivery, and postnatal care. It also covers routine diagnostics and EmOC at the primary, secondary and tertiary levels of the Ghanaian healthcare system¹³. Since implementation, the policy has had no comprehensive assessment to evaluate its effectiveness in order to recommend its continuation or modification.

This paper therefore assessed changes recorded in key MCH indicators attributable to this policy, and in a way tested the hypothesis that the free maternal care policy has produced improvements in the selected MCH indicators in Ghana.

Objectives

- To describe and analyze trends in available data on the key indicators of Maternal and Child Health such as maternal mortality rate, supervised delivery rate, contraceptive prevalence rate, and neonatal mortality rate from 2001 to 2011
- To examine for and highlight changes in the indicators following the introduction of the policy and make appropriate recommendations.

Methodology

We conducted a literature search to review existing literature on similar evaluations of free maternal care policies implemented in developing countries including Africa. This comprised a key word search with truncation on the databases of Popline, EMBASE and PubMed, including Medline using keywords: matern* impact; pregnancy impact; antenatal care; delivery impact; neonatal impact; free matern* care; developing countr*; which were searched separately and then linked together. No time frame was included. There was also a title search for articles and publications on "impact of free maternal care policy in developing countries" on Google Scholar database. These yielded over a thousand five hundred and thirty peer-reviewed article publications, editorials and commentaries on the impact of the free maternal care policy on maternal and

neonatal health indicators in developing countries. The search was restricted to only publications in English language. Additional search of the websites of world health and international development, population and health agencies like www.who.int; www.unicef.org; www.worldbank.org; and www.unfpa.org was done to obtain more publications and later, data on the selected indicators obtained for description and analysis.

The list of publications was then reviewed and those which did not focus on free maternal healthcare or service in a low-income country excluded. Five publications which assessed aspects of Ghana's policy were also reviewed.

Existing national data on maternal and child health indicators for Ghana was extracted for the period 1990 – 2010, and aggregated, covering:

Antenatal coverage, Caesarean section rate, Maternal Mortality Ratio, Contraceptive prevalence rate, Unmet need for contraception, skilled birth attendance rate, Neonatal Infant mortality rates.

Facility based data was then extracted in June-July 2012 from the annual returns and routine data reports of three conveniently sampled facilities in Ghana namely Maamobi polyclinic (primary); Koforidua Regional Hospital (secondary) and the Korle Bu Teaching Hospital (tertiary) from the period 2001 to 2011. Maamobi polyclinic is a primary healthcare facility located in a densely populated sub-urban location in Accra. This facility has a 46 bed maternity unit with one operating theatre, one labour ward, and manned by two Specialist Obstetricians, two medical officers and 30 midwives and nurses. Koforidua Regional Hospital (KRH) is a secondary facility that serves as both primary and referral centre for all hospitals and clinics in the Eastern Region of Ghana. It has a maternity unit with 94 beds, one labour ward with 10 delivery suites and manned by one Consultant Obstetrician Gynaecologist, 2 Residents in training, 2 Medical Officers, 4-6 House Officers, 65 Midwives and Nurses, and 12 Nursing Assistants. Korle Bu Teaching Hospital is the leading teaching facility in Ghana with over 2000 bed capacity and a busy maternity unit that receives complicated referrals from all over the country into its 375 bed block that has two labour wards with a total of 18 delivery suites and 3 operating theatres; manned by 264 Nurses and Midwives. There are 12 Consultants, 10 Specialists, 22 Residents, and 27 House Officers. The unit also houses the hospital's Neonatal Intensive Care Unit (NICU) with 40-baby capacity, 32 Nurses and 4 Nursing Assistants, 4 House Officers, 5 Residents and one Consultant Neonatologist.

These data were then appropriately aggregated and presented in separate tabular and graphical forms, observed trends were described and statistically analysed to determine whether the changes occurring in the selected indicator after the policy was implemented are significant based on the t – distribution test and 95% confidence interval calculations. This was based

on the difference in the means of the indicators before and after the policy intervention.

Results

Nationally, there was an average increase of 1.8% (95%CI: -3 to 7) in the national antenatal care coverage after the policy was implemented (p=0.5). There was an 11% (p=0.02) increase recorded in skilled birth attendance following the policy implementation. The contraceptive prevalence remained unchanged alongside a marginal rise in the unmet need for contraception from 34 to 35% in 2010. Under-five mortality rate (U5MR) declined by a non-significant 22 deaths per 1000 births. Available Neonatal Mortality rates (NMR) were 43/1000LB and 30/1000LB in 2003 and 2008 respectively; with Infant Mortality rates (IMR) 64/100LB and 50/1000LB in 2003 and 2008 respectively. There was a clear decrease in MMR from 550 through 450, to 350/100000LB in 2000, 2005 and 2008 respectively.

Trends in national MCH indicators from 200-2011

Table 1: Trends in the national ANC Coverage (%); Women having 4 ANC visit; Skilled Birth(%); MMR per 100000LBs, Contraceptive Prevalence(%); Unmet need for Contraception(15-49years)%; Lifetime Risk of maternal death(1 in); NMR; IMR; U5MR(per 1000LB)

Indicator	2000	2003	2005	2008	2010
ANC Coverage (%)	88	92	92	95	90
At Least 4 ANC Visits	62	69	77	78	78
Skilled Birth Attendance (%)	44	47	50	59	57
MMR per 100,000LB	550		440		350
Contraceptive Prevalence (%)	22	25	17	24	24
Unmet need for contraception (15-49) %	34		34	35	35
Lifetime risk of Maternal death 1 in	39		51		68
Neonatal Mortality Rate / 1000Birth		43		30	
Infant Mortality Rate / 1000Births		64		50	
Under 5 Mortality Rate / 1000Births	99	111	86	79	74

At the Korle Bu Teaching Hospital (figure-1 and table-2), ANC attendance increased by 643 (p > 0.5). Annual deliveries decreased by a non-significant 295 (p > 0.5) over three years of policy implementation. MMR rose

by 89/100,000LB (95%CI: -97, 275; P > 0.5). The absolute number of Caesareans rose by 602 (p=0.05); but the C/S rate increased by 5.5% (p=0.1). Still birth rate reduced by 1%, but the proportion of fresh still births saw a lower decline of 0.9% (p > 0.5). There was a 21% increase (n=354) (p=0.1) in NICU admissions following the policy implementation. The NICU receives referrals from nation-wide

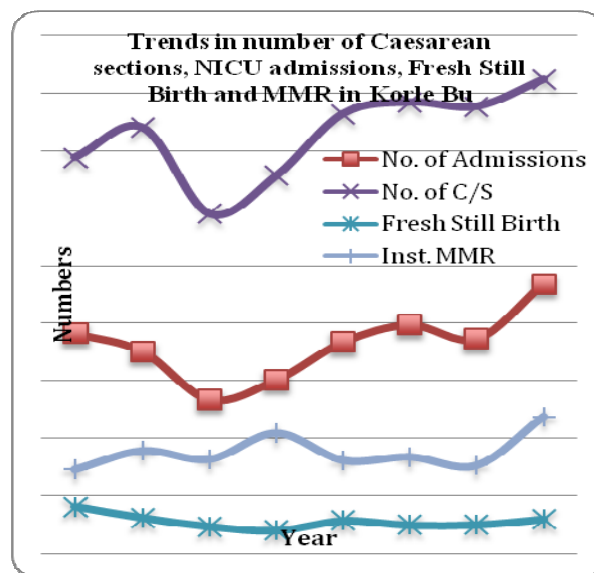


Figure1: Patterns in selected indicators at the KBTH over the period 2001 – 2011: C/S increased by 602; MMR slightly increased by 89/100,000LB; Proportion of FSB in still birth rates marginally increased by 1%; and NICU admissions increased by 354 cases following the policy implementation.

Antenatal care, deliveries and still births at KBTH

Table2: Trends in ANC Registration, Attendance; Still Births; Fresh Still Births and Annual Deliveries at the KBTH 2001-2011

Year	New ANC Registrants	ANC Attendance	Still Births Rate (%)	Proportion of Fresh Still Births (%)	Annual Deliveries
2001	17346	46439	6.5	57	12631
2002	15147	41420	5.8	53	12502
2003	13467	33773	6.8	53	11484
2004	14194	33848	6.2	54	12060
2005	11060	35690	5.5	47	12159
2006	11409	27092	6.3	52	7261
2007	11225	29187	5.3	51	7559
2008	13273	32390	5.3	54	9994
2009	13413	43032	4.6	51	10673
2010	16557	35795	4.8	48	10882
2011	13155	32749	5.4	53	10503

Data from the Koforidua Regional Hospital (Table-3 and Figure-2) showed a near 100% rise (n=3530) in ANC attendance by the third year of the policy (p=0.05). Similarly, annual deliveries increased by a significant 2114 babies (95%CI: 1362 – 2866; p < 0.001). C/S rates fell by 2.8% following the policy (p > 0.5); and a significant decline of 0.56% (562/100,000LB) in MMR (p = 0.05). Although still birth rates marginally decreased by 1.5%, the proportion of FSB recorded had increased by 13.3% (p=0.05) after three years of the free delivery policy.

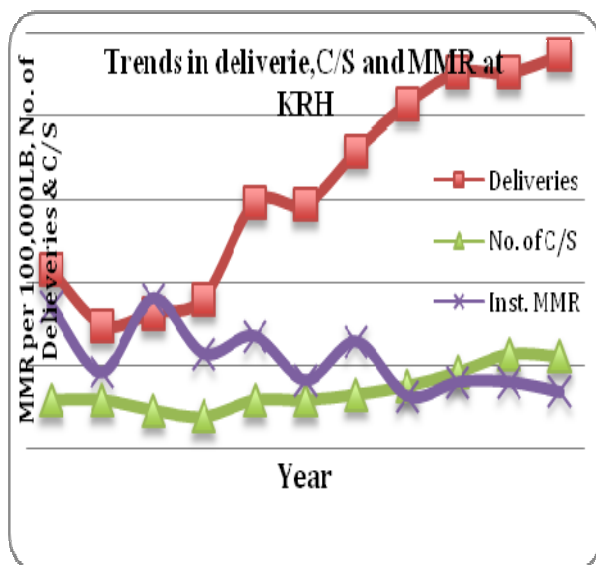


Figure 2: Pattern of Annual Deliveries; Caesarean section and Maternal Mortality at the KRH from 2001-2011.

Antenatal care at Koforidua regional hospital

Table 3: Trends in ANC Registration, At Least 4 Visits, and Attendance at the KRH from 2006-2011

Year	New ANC Registrants	At Least 4 ANC Visits	ANC Attendance
2006	301	621	1691
2007	980	1655	6384
2008	1403	1234	7417
2009	1238	1261	6425
2010	1372	1096	7899
2011	1451	1751	8530

From Fig.3, ANC attendance at the Maamobi policlinic reduced by 567 (p > 0.5); but annual deliveries increased by 300 (p=0.2). Similarly, the C/S rates rose by 3.1% (p=0.1). Again, the proportion of fresh still births component of still births rose by a significant 11% (95% CI: 4 – 18%), (p=0.02).

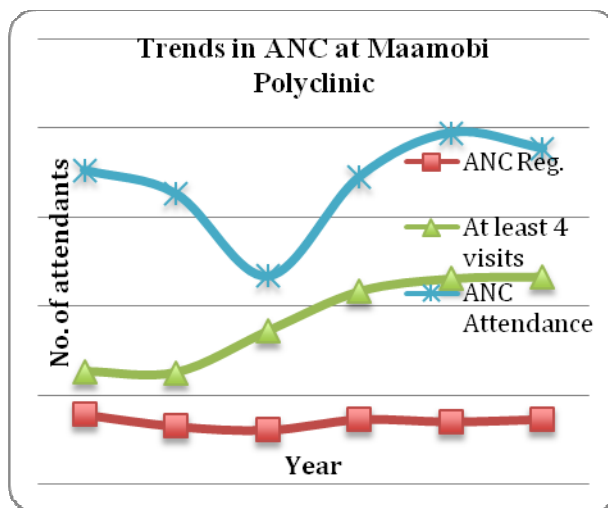


Figure3: Pattern in ANC Registration; At least 4 Visits, and Attendance at the Maamobi Polyclinic 2006-2011

There was a near four-fold increase in the number of Manual Vacuum Aspirations (MVAs) done for women presenting with incomplete abortions in the densely populated catchment area of the Maamobi Polyclinic following the policy implementation. The policy does not cover this service.

Discussion

Some improvement has been recorded in the national trends of ANC coverage, albeit statistically non-significant. The significant 11% increase in the percentage of births assisted by skilled health professionals may be attributable to the policy. Both increases particularly peaked at 95% and 59% respectively in 2008, the year of the policy implementation nationwide. These peaks may be explained by the sudden removal of the otherwise prohibitive financial barriers to both antenatal care and facility delivery. Many women were now able to access the available maternal care they have been in need of but unable to access earlier due to financial and other constraints. The 5% drop in ANC coverage and 2% in the skilled birth attendance rate in the policy’s third year may be the indirect reflections of the increased burden of clinical workload that the surge in patient numbers had imposed on the healthcare staff and facilities. These healthcare staff received no added remuneration for the extra work done, and similarly the existing facilities had seen no expansion in space or medical logistics to cope with the anticipated increase in patient turn-out. As a result, and consistent with findings in previous studies³, the overburdened staff became unfriendly in attitude and so the quality of care they provided waned, and these may have discouraged pregnant women from patronising ANC and delivery services at the facilities¹⁵. This pattern notwithstanding, Ghana’s skilled birth attendance rate still remained

above the UNFPA estimation of 34% in low income countries. In Ghana, the skilled birth attendance steadily increased from 44% in 2000 to 57% in 2010, accompanied by a declining MMR from 550 to 350/100,000LB over same period. In addition to the overall encouraging effects of the policy in three years, the over 60% literacy level rate in Ghanaian women (which according to UNICEF increased dramatically from 2005-2010) could be a major contributor to the ANC utilization and SBA rates. This is because a woman's education level has been found to have a strong influence on (maternal) healthcare utilization³⁰. The national contraceptive prevalence rate among the 15-49-year olds did not show any remarkable change over the period; and the unmet need for contraception marginally increased. This trend may be the result of the policy's provisions not covering family planning services despite available evidence indicating that women delivering in hospitals are increasingly welcoming the opportunity to delay their next pregnancy³¹.

Two years following the policy, there were 30% and 22% decline in the Neonatal Mortality and Infant Mortality Rates; however due to the lack of annual measurements of these indicators, detailed analysis of their trend was limited. The U5MR declined by 14%, meaning that 22 deaths per 1000 births were prevented over the period. This pattern while indicating progress towards the MDGs of reducing U5MR by two-thirds, also further emphasise the importance of national efforts and policies to improve perinatal morbidity and mortality³². Fenton et al³³ were correct in emphasizing that to further reduce infant mortality and U5MR, problems during birth and the first week of life must also be addressed.

The declaration in 2008 by the Ghanaian Minister of Health that maternal mortality was a national emergency had been a major 'focusing event' which further induced support and willingness amongst the major stakeholders for the implementation of the policy, including the media in creating the needed awareness. The government of Ghana subsequently received £2.8million in support of its free maternal care intervention from the British government¹¹

At the Korle Bu Teaching Hospital, slight decrease in ANC attendance and annual deliveries was found, with an accompanying rise in C/S rate by over 5.5% annually. There was a rise in MMR, though not statistically significant, possibly from increases in complicated and moribund obstetric emergencies seen at Korle Bu. This trend was however different from the experience from a similar policy piloted in Nigeria, where the C/S rate dropped slightly during the period of the free maternal care. The Nigerian finding was explained that there was timely availability of intervention to avert delays that would otherwise result in the need for the C/S³. In the KBTH, patient load increased from ANC registration, labour cases and other referred obstetric emergencies from various parts

of the country following the policy. Whether there was a commensurate increase in the staffing at the unit, physical expansion in equipment and logistics, or any incentive package for the staff here is beyond the scope of this report. However it is likely that, delays arose in the timeliness of care in the facility, resulting in many more complications possibly from poor labour monitoring, that consequently required Caesarean sections. Extrapolating from findings in the Enugu state pilot, "there was suboptimal care during this period of free maternal care, e.g. women who needed emergency caesarean sections had to wait for hours for the theatre to be free if there was already ongoing surgery, irrespective of the indication"³.

The decrease in ANC attendance and increase in C/S rate, MMR, and still births between 2010 and 2011 may indicate facilities were overstretched and so were the staff, that they were getting unfriendly to clients who therefore got discouraged to utilize the services; and subsequently, patient outcomes were getting poor for mothers and their babies. Even though most causes of maternal mortality are acute and unpredictable, some of the important causes and risk factors can be detected prenatally and managed to lower the woman's risk of maternal death if quality ANC is available and utilized. This could alternatively partially explain the rise in MMR as the total ANC attendance and delivery at this hospital waned³⁴.

There was a decrease in still birth rates after implementing the policy, but the proportion of fresh still births which decreased marginally from 50% in 2007 to 48% in 2010, rose again to 52% by 2011. This made an overall average 1% increase in the FSB proportion of still births after policy; the average percentage also persistently remained higher than the WHO recommended 10% FSB component of stillbirths. This usually reflects the fall in the quality of intrapartum care as the removal of financial barrier brought more clients to the tertiary facility. Fresh still births are usually due to intrapartum causes. The quality of care during pregnancy and delivery have been shown to contribute to maternal and neonatal mortality; the reason why poor care quality in many developing countries have led to high maternal and perinatal mortality rates³³. Initial gains in decreasing MMR in Korle Bu were probably becoming nullified by workload and increase care demand at the maternity unit.

The Koforidua Regional Hospital reported significant increases in ANC attendance, deliveries and a non-significant decline in C/S rate alongside declining maternal mortality and still birth rates with increase in FSB percentages of still births. This secondary facility, received referrals from all over the Eastern region of Ghana; as well as providing primary care. Although the policy gave rise to marked increases in patient load, the hospital's output was still remarkable from the results obtained. One significant factor that may have contributed to this heartwarming

trend was the posting of a Consultant Obstetrician Gynaecologist to the facility to coincide with the rolling out of the policy. Additionally, Residents training in Obstetrics did regular six-month postings in this facility, thus, providing the much needed clinical care to the rising load of clients so that the professional staff was not as over-burdened as the case in the Korle Bu Teaching Hospital. It is also very likely that the quality of care was optimal and clients were probably relatively satisfied, which is why they kept coming and the numbers kept rising, since suboptimal care has been linked to decreased utilization and increased perinatal mortality³⁵. The increases in other operative vaginal deliveries like vacuum extraction further affirm the improved availability of skilled care when needed at the facility.

At the primary care facility, the greatest improvements across all the indicators assessed were recorded in the year immediately following the policy. This included the instrument-assisted vaginal deliveries and C/S rates; with a remarkable zero maternal death. It is likely the sudden awareness of free care among a population with needs for care, as depicted by the numbers of incomplete abortions recorded that required MVAs may explain this trend. Also a possible contributing factor to these remarkable results could be the location of this facility in a densely populated neighborhood, with skilled staff and care available round the clock. There was a marginal rise in total still births, with another significant increase in the proportion of fresh still births; indicating the increase client load may have caused some pregnant women to suffer delays in receiving care at the facility or not receiving adequate intrapartum monitoring, resulting in these adverse perinatal outcomes. This is consistent with Unicef's emphasis on addressing the health worker crises which it described as being critical to the improvement of maternal and newborn health especially in Africa and Asia³⁶. It is likely that with improvements in ANC attendance, most of the prenatally detectable maternal complications and risk factors may have been picked up timely and managed to avert mortality. The near four-fold increase in the average number of manual vacuum aspirations performed for patients with incomplete abortion at this primary facility, while reflecting a situation of rising unmet need for contraception, also further brings into focus the need to consider its coverage under the scheme's care package.

Overall there are still more potential gains to be made in reducing maternal and child mortality in Ghana through increased skilled birth attendance for instance, aside the fee exemption policy for deliveries nationwide. As was found during an evaluation of the 'midwife in every village' programme in Indonesia by Laurel H. et al⁶, socio-economic inequalities in professional attendance at birth were reduced by the policy, but the gap in access to potentially life-saving emergency obstetric care widened. All these therefore

underscore the importance of understanding the barriers to accessing emergency obstetric care and of the ways to overcome them, especially among the poor⁶.

Conclusion

There have been remarkable improvements in some of the selected MCH indicators over three years of implementation, attributable to the free maternal care policy nation-wide from 2008. Across primary, secondary and tertiary levels of healthcare, still birth rates have been variable, but the proportion of fresh still births have consistently increased to varying extents following this policy.

Contraceptive prevalence rate remained relatively unchanged while unmet need for contraception increased to indicate the potential gains Ghana could make if the policy covered family planning services. Overall, the policy holds prospects to accelerate progress towards the MDGs 4 and 5 if implemented properly with adequate pre-implementation preparations to address the challenges of additional workload so that care quality is not compromised, and to ensure sustainable improvements in all indicators in low-income countries.

References

1. Brugha R, Pritze-Aliassime S. Promoting safe motherhood through the private sector in low- and middle-income countries. *Bulletin of the World Health Organization* [Internet]. 2003 81:616–23. Available from: <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2572515&tool=pmcentrez&rendertype=abstract>
2. Richard F, Witter S, de Brouwere V. Innovative approaches to reducing financial barriers to obstetric care in low-income countries. *American journal of public health* [Internet]. 2010 Oct [cited 2012 Aug 19];100(10):1845–52. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/20724689>
3. Ezugwu EC, Onah H, Iyoke CA EF. Obstetric outcome following free maternal care at Enugu State University Teaching Hospital (ESUTH), Parklane, Enugu, South-eastern Nigeria. *J Obstet Gynaecol. J Obstet Gynaecol.*; 2011; 31(5):*J Obstet Gynaecol.* 2011;31(5):409–12.
4. Borghi J, Ensor T, Neupane BD, Tiwari S. Financial implications of skilled attendance at delivery in Nepal. *Tropical medicine & international health: TM & IH* [Internet]. 2006 Feb [cited 2012 Aug 15]; 11(2):228–37. Available from:<http://www.ncbi.nlm.nih.gov/pubmed/16451348>
5. Prata N, Graff M, Graves a, Potts M. Avoidable maternal deaths: three ways to help now. *Global public health* [Internet]. 2009 Jan [cited 2012 Aug

- 16];4(6):575–87. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/19326279>
6. Hatt L. Did the strategy of skilled attendance at birth reach the poor in Indonesia? *Bulletin of the World Health Organization* [Internet]. 2007 Oct 1 [cited 2012 Aug 15];85(10):774–82. Available from: <http://www.who.int/bulletin/volumes/85/10/06-033472.pdf>
 7. Nahar S, Costello a. The hidden cost of “free” maternity care in Dhaka, Bangladesh. *Health policy and planning* [Internet]. 1998 Dec;13(4):417–22. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/10346033>
 8. Okafor II, Obi SN UE. Impact of Free Maternal and Child Healthcare programme on maternal and neonatal healthcare outcome in Enugu State of Nigeria. *a/Local/Temp/3/pubmed_result.txt* 1. *Niger J Med.* 2011 Oct-Dec;20(4):441-3. 2011;20(4):22288319.
 9. National Health Insurance Authority. The Road to Ghana’s Healthcare Financing - From Nkrumah to Health Insurance [Internet]. www.nhis.gov.gh. 2012 [cited 2012 Jun 16]. Available from: <http://nhis.gov.gh>
 10. Bosu W, Bell JS, Armar-Klemesu M, Tornui JA. Effect of delivery care user fee exemption policy on institutional maternal deaths in the central and volta regions of Ghana. *Ghana medical journal* [Internet]. 2007 Sep;41(3):118–24. Available from: <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2279091&tool=pmcentrez&rendertype=abstract>
 11. Witter S, Adjei S, Armar-Klemesu M, Graham W. Providing free maternal health care: ten lessons from an evaluation of the national delivery exemption policy in Ghana. *Global health action* [Internet]. 2009 Jan [cited 2012 Aug 15];2:1–5. Available from: <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2779941&tool=pmcentrez&rendertype=abstract>
 12. Witter S, Armar-klemesu M, Dieng T. National fee exemption schemes for deliveries: comparing the recent experiences of Ghana and Senegal. *Studies in HSO&P.* 2008; 24:167–98.
 13. MOH. Implementation Guidelines For Financing Free Delivery Through NHIS. 2008 ;(June):1–4.
 14. Costello A. Should mother and child health services in developing countries be free? Anthony Costello. *BMJ (Clinical research ed.)* [Internet]. 1997 Mar 29;314(7085):925–8. Available from: <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2126378&tool=pmcentrez&rendertype=abstract>
 15. Kruk ME, Mbaruku G, Rockers PC, Galea S. User fee exemptions are not enough: out-of-pocket payments for “free” delivery services in rural Tanzania. *Tropical medicine & international health : TM & IH* [Internet]. 2008 Dec [cited 2012 Aug 15]; 13(12):1442–51. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/18983268>
 16. The World Bank/Bank Dunia. “. . . and then she died’ Indonesia Maternal Health Assessment. 2010; 22 - 46.
 17. Bhat R, Mavalankar DV, Singh PV, Singh N. Maternal healthcare financing: Gujarat’s Chiranjeevi Scheme and its beneficiaries. *Journal of health, population, and nutrition* [Internet]. 2009 Apr;27(2):249–58. Available from: <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2761781&tool=pmcentrez&rendertype=abstract>
 18. Royat S. The PNPM Generasi : Conditional Cash Transfer for Poor people Driven by Community For Better Health and Education In Indonesia. :1–15. Available from: <http://www.socialsecurityextension.org/gimi/gess/RessFileDownload.do;jsessionid=6f1519d7f3>
 19. ILO. Initiatives In South East Asia Indonesia : Conditional Cash Transfer To The Poor [Internet]. Available from: <http://www.ilo.org/public/english/region/asro/bangkok/events/sis/download/paper22.pdf>
 20. Bellows NM, Bellows BW, Warren C. Systematic Review: the use of vouchers for reproductive health services in developing countries: systematic review. *Tropical medicine & international health : TM & IH* [Internet]. 2011 Jan [cited 2012 Aug 15];16(1):84–96. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/21044235>
 21. Ensor T, Ronoh J. Effective financing of maternal health services: a review of the literature. *Health policy (Amsterdam, Netherlands)* [Internet]. 2005 Dec [cited 2012 Jul 28];75(1):49–58. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/16298228>
 22. Ridde V, Diarra A. A process evaluation of user fees abolition for pregnant women and children under five years in two districts in Niger (West Africa). *BMC health services research* [Internet]. 2009 Jan [cited 2012 Aug 15];9:89. Available from: <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=2698841&tool=pmcentrez&rendertype=abstract>
 23. GSS. Demographic and Health Survey 2008 Report. Ghana. 2008; 56 - 147.
 24. GHS. Reproductive Health Strategic Plan [Internet]. 2007. Available from: www.ghs.org
 25. WHO. Ghana Factsheets of Health Statistics 2010. 2010.
 26. GHS. Ghana Health Service Annual Report. 2010; 28 -39.
 27. WHO, UNICEF, UNFPA W. Maternal mortality in 1990-2010 WHO, UNICEF, UNFPA, The World Bank and UN Population Division Maternal Mortality Estimation Inter-Agency Group Ghana

- Maternal mortality in 1990-2010 WHO, UNICEF, UNFPA, The World Bank and UN Population Division Maternal Mortality. 2010 p. 2010.
28. The WORLD BANK. Trends in Maternal Mortality : 1990 to 2010. 2010.
 29. WHO. WHO country statistics for Ghana [Internet]. 2003 p. 13-15. Available from: www.who.int
 30. Chakraborty N. Determinants of the use of maternal health services in rural Bangladesh. Health Promotion International [Internet]. 2003 Dec 1 [cited 2012 Jul 15]; 18(4):327–37. Available from: <http://www.heapro.oupjournals.org/cgi/doi/10.1093/heapro/dag414>
 31. Laureen Lopez, Grimes D, Szpir M. Immediate Postpartum Insertion of an IUD is Safe and Effective. Global Health Technical Briefs. 2001;6-7.
 32. Unicef. Tracking Progress in Maternal, Newborn & Child Survival the 2008 Report Newborn & Child Survival the 2008 Report. 2008; 167-221.
 33. Fenton PM, Tadesse E. Reducing perinatal and maternal mortality in the world: major challenges. BJOG : an international journal of obstetrics and gynaecology [Internet]. 2000 Jun;107(6):831–2. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/10847245>
 34. Carroli G, Rooney C, Villar J. How effective is antenatal care in preventing maternal mortality and serious morbidity? An overview of the evidence. Paediatric and perinatal epidemiology [Internet]. 2001 Jan;15 Suppl 1:1–42. Available from: <http://www.ncbi.nlm.nih.gov/pubmed/11243499>
 35. Cameron B. Outcomes in rural obstetrics. *Aust J Rural Health*. 1998;6:46–51.
 36. Unicef. THE STATE OF THE WORLD'S CHILDREN 2009 Maternal and. 2009.
 37. Biritwum R. Promoting and monitoring safe motherhood in Ghana. *Ghana medical journal* [Internet]. 2006 Sep; 40(3):78–9. Available from: <http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1790854&tool=pmcentrez&rendertype=abstract>
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CONSENT TO MEDICAL TREATMENT: WHAT ABOUT THE ADULT PATIENT WHO IS INCAPABLE OF PROVIDING A VALID CONSENT?

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Introduction

Consent to medical treatment is a principle that is increasingly gaining attention in health care systems across the world. For consent to be valid, five essential elements must exist. The patient must have the mental 'capacity' to provide consent, he or she must receive adequate and accurate information, understand the information disclosed, make a decision voluntarily and without coercion, and then authorize the treatment¹.

In an article on 'consent to medical treatment' in a previous edition of this journal, I outlined how the Ghanaian courts may resolve consent related information disclosure disputes². That article dealt with the provision of information to the adult patient who has the mental capacity to provide lawful consent to medical treatment. A likely question that an interested reader of that article may ask is; 'what are the legal provisions in Ghana for treating patients who lack the capacity to provide lawful consent to medical treatment as a result of factors such as a young age^{3,4}, disease⁵, severe brain or mental illness or incapacity such as occurs in severe dementia, severe learning disabilities⁶, and being unconscious?'^{7,8}.

The purpose of this article is to attempt to answer such a question. The scope of the article is limited to the management of the adult patient without the mental capacity to consent to medical treatment, which for the purpose of this article, in Ghana, refers to any individual aged 18 years or older who lacks the mental capacity.

The legal provisions and considerations for obtaining consent for treating the 'minor' will be dealt with in a separate article.

A changing world and consent to medical treatment

The world is increasingly becoming smaller as interactions between people from different parts of the

world increase. Issues that previously posed no problems to medical practice in Ghana are beginning to, and may continue to pose dilemmas as they become dilemmas for medical practitioners in other countries, and also as medical litigation increases in Ghana^{9, 10}. It is interesting to note that until nearly thirty years ago the question of the legality of treating patients who lack mental capacity to consent to medical treatment was not an issue that many gave much thought about even in some developed countries. Doctors and family simply went ahead and treated such patients on the basis of what the doctor thought best¹¹. Now, with increased recognition by society of individual freedoms and liberty¹², the question of who provides consent for the patient who lacks the mental capacity has presented legal disputes to the courts in some countries¹³. It is likely to do the same in Ghana sooner or later. It is important, therefore that the doctor is aware of the legal provisions in Ghana for treating such patients. Although this article provides general guidance to the doctor on treating the patient without capacity to consent to medical treatment, it is not meant to replace legal advice on the issue if required.

Legal provisions for treating patients who lack mental capacity in Ghana

Currently, in Ghana, Article 30 of the 1992 Constitution of the Republic of Ghana is the legislation that makes provision for consent to medical treatment in the adult patient without the ability to provide his or her own consent. Article 30 of the 1992 Constitution states that:

'A person who by reason of sickness or any other cause is unable to give his consent shall not be deprived by any other person of medical treatment...by reason only of religious or other beliefs'.

This legislation recognizes that there may be situations where individuals may lack the capacity to provide their own 'consent' to medical treatment. What it fails to do, however, is provide details on how the determination of the inability to provide 'consent' should be made, other than say that the individual is unable because of sickness. If confirmed that the patient indeed lacks the capacity to provide his or her own 'consent' to medical treatment, the legislation does not say who provides 'consent' on behalf of the patient. It has also not taken into account the fact that

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sometimes treatments may not be in a patient's 'best interest', and does not, therefore, provide for how the 'best interest' of a patient should be determined.

As the law stands, any dispute involving the authorization of treatment in a patient without the mental capacity to provide his or her own consent in Ghana is likely to be resolved in case law. Issues such as; whether or not the patient possesses the appropriate mental capacity to provide his or her own consent, how the determination of 'capacity' is made, who provides consent if the patient is deemed to lack 'capacity' and what constitutes the patient's 'best interest' may be raised in the courts.

The Patient Charter¹⁴ and the Code of Ethics of the Ghana Health Service¹⁵ make some provision on the matter which the courts may interpret in the context of a particular situation. Although The Patient Charter of the Ghana Health Service provides that 'recognized' and 'accredited' individuals can make decisions on behalf of other adults who are incapable of making their own decisions, it does not provide guidance on how the 'recognized' and 'accredited' individuals should make those decisions, other than that the decisions must be in the interest of the patient. When dealing with issues without specific clarification in legislation or precedents in Ghanaian law, Ghanaian judges just like judges in other common law jurisdictions refer to precedents from other common law jurisdictions such as the UK, South Africa, Australia, USA, and Canada¹⁶ in making their rulings.

What principle should guide decision-making for the patient without mental capacity?

The principles that have been applied to make healthcare decisions for adults without capacity elsewhere include 'best interest'¹⁷, and substituted judgment¹⁸. Best interest requires that decision makers consider the overall welfare of the patient and make a decision in accordance with that, whereas 'substituted judgment' requires an attempt at ascertaining what the patient would have chosen were he or she able to make the decision himself or herself¹⁹. Interestingly in the determination of 'best interest', there is a requirement to make a reasonable effort to ascertain as far as possible what the patient without 'capacity' would have chosen or done.

In the UK, the law on consent to medical treatment is such that no other person, unless granted such powers by the court can provide lawful consent for another adult, although health care professionals can provide treatment to such a patient in the patient's 'best interest'^{21 22}. In that jurisdiction not even the next of kin or other family members, can provide lawful consent for the medical treatment of an adult who is incapable of providing his or her own consent to medical treatment. The Mental Capacity Act 2005 in England and Wales, and the Adults with Incapacity (Scotland) Act 2000 currently provide regulation on the

issue and guidance to doctors and others as to how to go about providing lawful treatment to patients who lack the capacity to provide their own consent to medical treatment in a way that respects the patient's autonomy and dignity. In other countries, such as Canada^{23, 24, 25, 26, 27} and some states in the USA²⁸, the law allows the next of kin and recognized others such as spouses and other family members to make decisions on behalf of adults who lack mental capacity to do so. In these jurisdictions the substitute decision maker is expected to make the decision for the patient based on the principle of substituted judgment or if they are unaware of what the patient would want in the particular situation, to make the decision based on the 'best interest' of the patient. The Health Care Consent Act, together with similar Provincial Acts, regulate the provision of care to patients who lack the capacity to do so and provide guidance for health care and other professionals in this area in Canada.

An important development in health care law in Ghana is the enactment of The Mental Health Act 2012²⁹. This Act provides regulation for the management of patients with mental illnesses in a way that respects the autonomy and dignity of individuals with mental illnesses. It makes provision for treating mental illness in patients with mental illness without the capacity to provide consent. It provides for compulsory detention and treatment if the patient is deemed to lack the capacity to consent to treatment of his or her mental illness. It does not however, make provision for treating conditions other than mental illnesses, such as a surgical operation, in mentally ill patients who lack the capacity to provide consent for such treatment. Many mentally ill patients do not lack the mental capacity to provide consent for their medical treatment. A number of patients who lack the mental capacity to consent to medical treatment do not suffer from a mental illness. The Mental Health Act 2012 although very useful for managing mental illness, does not provide the solution for dealing with patients without a mental illness who lack the capacity to provide their own consent to medical treatment.

It may be time for the Ghanaian parliament to consider a Mental Capacity Act in Ghana or a similar Act to provide regulation and guidance on the matter. Until such time that such a legislation is enacted it may be prudent to revise the Patient Charter of the Ghana Health Service to include detailed guidance on how to go about the management of the patient who lacks the mental capacity to provide a valid consent to medical treatment. Alternatively, the Medical and Dental Council of Ghana could produce a guidance document on the issue.

Determining the lack of mental capacity

As there is no specific guidance in the law on how mental capacity is determined in Ghana in relation to 'consent to medical treatment' one has to look to how other countries with similar jurisdictions to the

Ghanaian legal jurisdiction determine mental capacity in relation to consent to medical treatment. In other common law jurisdictions such as the UK and Canada, a person is said to have capacity to consent to medical treatment if he or she is capable of understanding the information relevant to making a decision about the treatment and able to appreciate the reasonably foreseeable consequences of a decision or a lack of a decision. He or she must be able to weigh the relevant information and thus the competing factors in the process of arriving at his or her decision to accept or refuse treatment. A person lacks capacity in relation to a matter if at the material time he or she is unable to make a decision for him or herself in relation to the matter because of an impairment of or disturbance in the functioning of the mind or brain. A person is unable to make a decision for himself or herself if he or she is unable to understand the information relevant to the decision, to retain the information, to use or weigh that information as part of the process of making the decision, or to communicate his or her decision (whether by talking, using sign language or any other means)³⁰. Generally, a doctor may assume that a patient is capable of providing his or her own consent to medical treatment unless there are reasonable grounds to believe otherwise. Reasonable grounds could be something in the patient's history or behaviour that would make the doctor question the patient's capacity to consent. It is however important to note that an unwise decision by the patient does not necessarily imply lack of capacity to consent³¹. In general, if a patient knows who and where he or she is, what medical intervention is being proposed, and the consequences of the decision he or she is being asked to make, it is safe to assume that he or she has capacity³². Illiteracy and a language barrier per se do not imply a lack of capacity. It is also important to note that the lack of capacity may be temporary or permanent. It is not static and it is specific to the situation or treatment in question. It can change over time and be different depending on the nature and complexity of the specific treatment decision. What is determined in relation to consent to medical treatment is whether the patient has the ability to understand the nature and effect of the particular treatment being proposed, and not whether 'globally' he or she has the capacity to make decisions.

The Patient Charter of the Ghana Health Service which requires that patients are provided with adequate and accurate information about their health condition and their consent obtained prior to treatment, also provides that other 'accredited' and 'recognized' individuals can lawfully authorize treatment on behalf of another adult patient who for whatever reason is unable to provide consent to medical treatment, provided that the treatment is in the patient's interest.

Determining a patient's best interest

Best interest in relation to consent to medical treatment is very difficult to determine because it is not limited only to 'medical best interest'. It is generally accepted that the doctor may proceed to treat a patient without the capacity to consent to treatment in an emergency situation in the 'patient's best interest', which in an emergency situation is often to save the patient's life or prevent the patient from coming to serious harm. Best interest in the non-emergency situation however is not always easy to determine. For example, although it is a crime in Ghanaian law to have sexual relations with a lunatic, criminals do exist and women with severe mental disability could be impregnated. The family of a patient with mental disability who are concerned about her getting pregnant may present her to a doctor to get her sterilized to potentially stop her becoming pregnant³³. An argument could be raised as to whether surgical sterilization is the best course of action and whether that is in the best interest of this patient. The dilemma then is how should the 'best interest' decision in this instance be made?

The Mental Capacity Act 2005 in England and Wales, and The Adult with Incapacity (Scotland) Act 2000 provide very helpful principles on how to determine a patient's best interest, which in my view and in the view of the Medical Protection Society of the UK³⁴ are almost universally applicable, regardless of one's country. It is these principles that I outline next.

The key focus in best interest determination, as has been alluded to by the Medical Protection Society in the UK, is that the focus should be on what the patient will consider his or her best interest, not what the doctor would consider his or her best interest if he or she were in the same position. In trying to determine the 'best interest' of a patient, the doctor should encourage as much as possible the patient without capacity to take part in the decision making and make reasonable efforts to improve the patient's capacity to do so. He or she should try as much as possible to identify all the things that the patient would take into account if he or she were making the decision himself or herself. The doctor should try to ascertain as much as possible the views of the patient which may include his past and present wishes and feelings which may have been expressed either in writing, behaviour or habits, any beliefs and values such as religious, cultural, political or moral beliefs or any other factors that would be likely to influence the decision in question if the patient were making the decision for himself or herself. The doctor should not make any assumptions about a patient's 'best interest' simply on the basis of the person's age, appearance, condition or behaviour.

The doctor should not make assumptions about the person's quality of life and he or she should not be motivated in any way by a desire to bring about the death of the patient. The doctor needs to consider whether the patient is likely to regain capacity at any stage, such as after initial treatment, and if so whether the other decisions can wait until then in order to give the patient the opportunity to make it or at least participate in it. If it is practical and appropriate to do so, the doctor should consult other relevant people who know or have an interest in the patient for their views on the patient's best interest. The doctor may also seek information from them about the patient's feelings, wishes, beliefs and values. In particular, the doctor may consult any person previously named by the patient as someone to be consulted on the issue in question or similar issues. He or she may also consult anyone engaged in caring for the patient, close relatives, friends and anyone with an interest in or powers to intervene in the welfare of the patient. Anybody appointed by a court to make decisions on behalf of the patient may also be consulted. When consulting, the doctor should remember that the patient still has a right to keep his affairs private and therefore it will not be right to share every piece of information with everyone.

The decision or treatment should be the most effective option that is least restrictive of the patient's rights. If the patient has never been competent and his wishes and feelings are unknown the 'best interest' may be assumed to be the same as that of the 'reasonable person'. In addition the proposed intervention must be necessary and beneficial to the patient, and must be the minimum necessary to achieve the purpose.

If substitute decision makers are making the decision on behalf of the patient, as the Patient Charter of the Ghana Health Service allows, their decision making should follow the same pattern as outlined above and the doctor may need to guide them as they make the decision to ensure that the patient's best interest is served.

Conclusion

In conclusion, to treat a patient without the mental capacity to consent to medical treatment, the doctor needs to make a determination that the patient lacks the capacity. Then he or she should request a 'recognized' or 'accredited individual' to make a decision for the patient, whilst ensuring that the decision is in the patient's best interest. The doctor may go ahead and treat the patient in the patient's best interest without consent in an emergency.

A patient lacks the mental capacity to consent to treatment if he or she is unable to understand, retain and weigh the information provided to him or her in coming to a decision, or is unable to communicate his or her decision. Acting in a patient's best interest means making a reasonable effort to ensure that

proceeding with the proposed treatment is what the patient would like if he or she were able to make the decision himself for herself. Best interest also requires that the proposed procedure is beneficial to and consistent with the overall welfare of the patient. It also requires that the procedure is the minimum necessary to achieve the purpose of the treatment.

References

1. Mason J K and Laurie G T 'Mason and McCall Smith's Law and Medical Ethics' (New York; Oxford University Press 2011, 8th edition) chapter 4
2. Adwedaa E, 'Consent to medical treatment: A doctor's view of how the Ghanaian courts may resolve consent related information disclosure disputes', *Postgrad Med J Ghana*. September 2014; 3: 98-102
3. Glass v United Kingdom [2004] 39 EHRR 341
4. Gillick v West Norfolk and Wisbech Area Health Authority [1985] 3 All ER 402
5. In the Estate of Park [1954] P 112
6. F v West Berkshire Health Authority [1989] 2 All ER 545
7. Murray v McMurchy [1949] 2 DLR 442
8. Mitchell J, 'A fundamental problem of consent' *British Medical Journal* 1995; 310: 43
9. Elizabeth Vaah v Lister Hospital and Fertility Centre (Suit No. HRCM 69/10), Fast Track Court, High Street, Accra, Ghana
10. Norman I D et al, 'The Constitutional Mandate for Judge-Made-Law and Judicial Activism: A Case Study of the Matter of Elizabeth Vaah v The Lister Hospital and Fertility Centre' *The Open Ethics Journal* 6 (2012)1-7
11. Brazier M, Cove E, *Medicine, Patients and the Law* (Fifth edition) (New York, USA: Penguin Group, 1987) pp150
12. Schloendorff v Society of New York Hospital (NY 1914) 105 NE 92
13. T v T [1998] 1 All ER 613
14. The Patient Charter of the Ghana Health Service. Available at <http://www.ghanahealthservice.org> last accessed on May 21, 2015
15. Code of Ethics of the Ghana Health Service. Available at <http://www.ghanahealthservice.org> last accessed on May 21, 2015
16. Edwin A K, 'Don't Lie but Don't Tell the Whole Truth: The Therapeutic Privilege- Is it Ever Justified?' *Ghana Med J* 2008; 42(4): 156-161
17. Mental Capacity Act 2005 (England and Wales) Available at <http://www.legislation.gov.uk/ukpga/2005/9/contents>
18. Health Care Consent Act, 1996; S.O. 1996, Chapter 2, Schedule A available at http://www.e-laws.gov.on.ca/html/statutes/english/elaws_statute_s_96h02_e.htm
19. In Re Quinlan 355 A 2d 664 (NJ, 1976)

20. Donnelly M, 'Best Interest, Patient Participation and The Mental Capacity Act 2005' Medical Law Review 2009; 17: 1
 21. F v West Berkshire Health Authority [1989] 2 All ER 545
 22. Adults with Incapacity (Scotland) Act 2000 available at <http://www.legislation.gov.uk/asp/2000/4/contents>
 23. The Vulnerable Persons Living with a Mental Disability Act (Manitoba C.C.S.M. c. V90) available at http://www.web2.gov.mb.ca/laws/statutes/ccsm/_pdf.php?cap=v90
 24. The Adult Guardianship and Co-decision-making Act (Chapter A-5.3 of the Statutes of Saskatchewan 2000) available at <http://www.publications.gov.sk.ca/details.cfm?p=392>
 25. Representation Agreement Act British Columbia, RSBC 1996 Chapter 405 available at http://www.bclaws.ca/civix/document/id/complete/statreg/96405_01
 26. Health Care (Consent) and Care Facility (Admissions) Act British Columbia, RSBC 1996 Chapter 181 available at http://www.bclaws.ca/Recon/document/ID/freeside/00_96181_01
 27. Substitute Decisions Act Ontario, S. O. 1992 Chapter 30 available at http://www.elaws.gov.on.ca/html/statutes/english/e_lasstatutes_92s30_e.htm
 28. Respondent Michael Schiavo's Opposition to Application for Injunction Case No: 04A-825, 24 March 2005 (Supreme Court, USA)
 29. Mental Health Act 846 of 2012 Ghana
 30. NHS Trust v T [2004] EWHC 1279
 31. Re B [2002] 2 All ER 449
 32. College of Physicians and Surgeons of Ontario document on Consent to Medical Treatment. Available at <http://www.cpso.on.ca/policies-publications/policy/consent-to-medical-treatment>
 33. Re S (Adult Patient: Sterilization) [2000] 3 WLR 1288
 34. An MPS essential guide to Consent document of the Medical protection Society, UK available at <http://www.medicalprotection.org/uk/guide-to-consent-in-the-uk/determining-the-patients-best-interests>
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CASE REPORT

NICOLAU SYNDROME: A CASE REPORT OF A RARE DEBILITATING COMPLICATION FOLLOWING INTRAMUSCULAR INJECTION OF PENICILLIN

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Summary

Objective: Nicolau Syndrome is a rare, debilitating and sometimes fatal complication resulting from the administration of parenteral medication. It is associated with administration of a variety of medications. It causes a local aseptic ischaemic necrosis of the muscles, subcutaneous tissues and skin. It may be associated with neurological deficits and organ failure. The administration of parenteral medication is common in our health institutions, but this complication seems to be unknown. We would like to increase awareness about its existence and the preventive measures to take in order to minimize its occurrence as it can be associated with significant morbidity and even mortality.

Case report and interventions: A young man presented with sudden onset of severe pain in the right buttock and the whole right lower limb with associated paralysis after an intramuscular injection of penicillin.

He developed a darkened patch of skin at the site of the injection on the right buttock and also on the right leg anteriorly. This progressed to necrosis of the skin, subcutaneous tissue and muscles and osteomyelitis of the right tibia. He also presented with acute renal failure. After serial debridement, wound dressing and antibiotic treatment the wound healed with extensive scarring, the osteomyelitis resolved and the neurological deficit improved leaving a foot drop. His renal function normalized after several sessions of haemodialysis.

Conclusion: Administration of parenteral medication can be complicated by debilitating conditions such as Nicolau Syndrome. No specific treatment exists, so it is best prevented by taking the necessary precautions during administration of parenteral medication when indicated.

Key Words: Nicolau Syndrome, Intramuscular injections, prevention

Introduction

Parenteral medications are very frequently administered in our health facilities, but they can be associated with complications. Nicolau Syndrome, also known as embolia cutis medicamentosa or livedoid dermatitis, was first described in the 1920's. It is a rare debilitating complication following administration of parenteral medication, particularly via the intramuscular route. It is a local aseptic cutaneous, soft tissue and sometimes muscular ischaemic necrosis at the site of administration of parenteral medication. It may have devastating complications such as paralysis, limb gangrene, neurological deficits, sepsis, organ failure and even death. Therefore it has significant morbidity, mortality and medicolegal implications. We present a case of a young man with Nicolau Syndrome following intramuscular injection (IM) injection of penicillin. This was complicated with osteomyelitis of the tibia, a neurological deficit of foot drop, and acute

renal failure.

Case Report

A 27-year old male was referred to the surgical emergency unit with a 5-day history of a swollen painful right buttock and lower limb with inability to walk. He had presented to a private clinic 5 days prior to presentation at the surgical emergency unit and had been given an IM injection of penicillin on account of a diagnosis of syphilis. This diagnosis was made based on a 1-month history of recurrent urethral discharge and 1-week history of oral ulcers and dysuria. His Venereal Disease Research Laboratory (VDRL) test and Treponema Pallidum hemagglutination assay (TPHA) were both positive. He developed severe pain immediately after the injection was given into the right buttock. This was associated with paralysis of the limb almost immediately necessitating subsequent admission at the clinic on that same day. He was found to have developed a darkened patch of skin with blisters at the injection site and on the leg a few days later, and so was referred to our facility on the 5th day after the incident. On presentation to the surgical emergency unit five days after the onset of the symptoms, he was acutely ill, moderately pale and afebrile. His cardiovascular system was stable, chest was clinically clear and abdomen was unremarkable. His right buttock and entire right lower limb was

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swollen, moderately tense and had differential warmth. There was a darkened patch of skin with blisters over the leg anteriorly (figure 1a), and another of about 10 by 8 cm, with blisters over the posterolateral aspect of the right buttock (figure 1b). Power in the right lower limb was 1/5 with associated right foot drop and loss of sensation. Peripheral pulses were not palpable probably due to the edema but capillary refill was less than 2 seconds. Initial diagnoses entertained included intraarterial injection with ischaemia, compartment syndrome, sciatic nerve injury and cellulitis.



Figure: 1a & 1b

On admission, he was noticed to be passing dark urine but in adequate volumes of more than 1L over 24hours. His haemoglobin count was 7.0g/dl, white blood cell count $14.68 \times 10^9/L$ and platelets 88×10^9 . His sodium level was 127 mmol/l, potassium 5.3 mmol/l, urea 42.5 mmol/l and creatinine 819 mmol/l. His liver function tests and clotting profile were normal. His Human Immunodeficiency Virus (HIV) test was negative. Doppler ultrasound of the right lower limb showed heterogenous echopattern with loss of muscle fibres with normal venous system. He was started on intravenous fluids, intravenous penicillin and cloxacillin and subcutaneous enoxaparine. He had 2 sessions of haemodialysis on account of deteriorating renal function. As the differential diagnoses did not all fit the patient's presentation, a literature search was done and the diagnosis of Nicolau Syndrome arrived at. Over the next few days, the pain and swelling reduced, and power improved, but he had a residual foot drop for which he had physiotherapy and was fitted with a foot brace. The renal function also improved. X-rays of the tibia and fibula showed localized osteomyelitis of the tibia for which he was treated with clindamycin for 6 weeks. The darkened patches over the gluteal region and leg became necrotic. He therefore had serial

debridement of the necrotic tissues (figure 2), with wound dressing. He was referred for plastic surgery assessment for grafting of the skin defect, but declined to have the procedure and eventually the wound healed by secondary intention (figure 3) in 3 months.

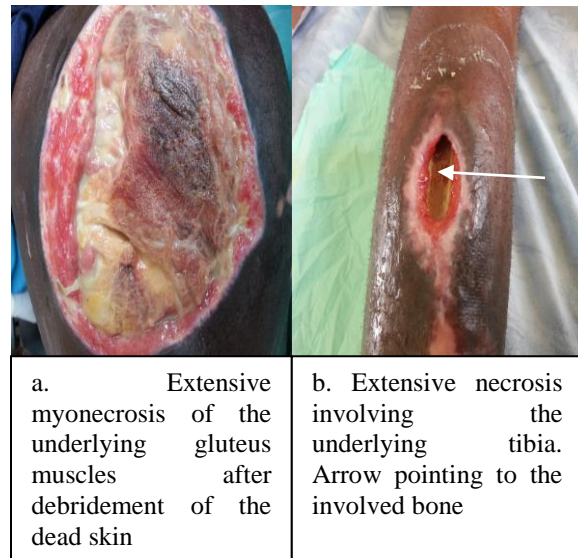


Figure: 2a & 2b



Figure: 3a & 3b

Discussion

Nicolau Syndrome, also known as *embolia cutis medicamentosa* or *livedoid dermatitis*, was first described in 1924 by Freudenthal¹ and has since been reported worldwide in association with administration of a wide variety of parenteral medications particularly intramuscular medications^{2,3,4}. More than 25 different drugs have been reported as causing this syndrome¹. Table 1 shows some reported causes of Nicolau Syndrome³⁻²³. Cases have also been reported in association with intra or periarticular injections¹⁷ of steroids as well as intravenous²² and subcutaneous injections²³.

It is a local aseptic cutaneous, soft tissue and sometimes muscular ischaemic necrosis. Pathogenesis

of Nicolau Syndrome is unclear but is likely to be of vascular origin^{1,2}. Intra-arterial, peri-arterial or

Table 1. Causes of some reported cases of Nicolau Syndrome

Drug	Route of administration	Site of administration
Diclofenac ^{3,5-13}	IM	Buttock, Thigh
Ketoprofen ^{8,10}	IM	Thigh, Buttock
Piroxicam ¹⁴	IA	Ankle
Ketorolac ¹⁵	IM	Buttock
Glucocorticoid ¹⁶	IA	Ankle
Paramethasone ¹⁷	IA	Shoulder
Cortivazol ¹⁷	IA	Shoulder
Hydrocortisone ¹⁷	IA	Shoulder
Penicillin ^{13,18-21}	IM	Thigh, Buttock
Meperidine ¹⁰	IM	Buttock
Cyanocobalamin ¹³	IM	Buttock
Polidocanol ²²	IV	Achilles tendon
Glatiramer acetate ²³	SC	Buttock
Hexavalent vaccine ⁴	IM	Thigh, arm,
DTP ⁴	IM	Thigh

IM – intramuscular, **IA** –intraarticular, **IV** – intravenous, **SC** – subcutaneous

DTP – diphtheria/tetanus/pertussis vaccine

peri-neural injections can result in occlusion of peripheral arterial vessels either from vasospasm, vessel damage or embolization of the administered medication. Subcutaneous injections with injury to cutaneous arteries may contribute to the occurrence of this syndrome^{3, 5, 8}. Application of cold compress for

pain relief may accelerate progression of the features and worsen outcome^{6, 7, 18}. Also, the mechanism of injury seems to be related solely to the process of administering the injection as injections of the same agents after an episode of Nicolau Syndrome have not been associated with recurrence of the syndrome^{3, 23}. However, another school of thought proposes that the role of physical and chemical factors such as injection technique and solution pH may not have any bearing on the occurrence of Nicolau syndrome¹³.

Nicolau Syndrome usually presents with sudden onset of excruciating pain at the site of the injection immediately after administration of the injection. Syncope may occur¹⁷. It is then followed by erythema, livedoid patch and hemorrhagic patch of skin at the injection site⁶. This skin reaction is pathognomonic of Nicolau Syndrome^{1, 2}. Ultimately, necrosis of skin, subcutaneous fat and / or muscles occurs over 1 – 2 weeks. Sequelae of this syndrome include: widespread cutaneous necrosis, extensive scarring, ischaemia of ipsilateral limb, organ failure, neurological deficits, superimposed infections, sepsis and compartment syndrome^{1, 2, 5, 7,9,19}. Factors that result in poor prognosis include use of cold compress on the injection site^{6,7,18}, superimposed infection^{5,6,8,9}, sepsis^{18,19} compartment syndrome¹⁹ and prior immunocompromised state of the patient^{5,6,8,9}. These can result in significant morbidity^{9, 19} and mortality^{9,19,24}. It therefore has significant medicolegal implications¹⁴.

No confirmatory diagnostic tests for Nicolau Syndrome exists². Diagnosis is purely clinical with correlation to previous case reports³. Investigations however should be done to rule out other differentials and assess for complications of the syndrome. Tests^{5,18,19,25} to be done include full blood count (FBC) to rule out an infection and anemia from intravascular hemolysis; blood urea, electrolytes and creatinine (BUE and CR) to identify any associated renal impairment; creatinine kinase to detect muscle damage; arterial blood gases (ABG) to assess metabolic derangements; urinalysis to detect myoglobinuria resulting from muscle necrosis; cultures from the ulcers to detect superimposed infection; electrocardiogram (ECG) to rule out any hyperkalemia from renal failure and duplex scan of the vessels of the affected limb to rule out a deep vein thrombosis and arterial occlusion. Computed tomogram (CT) scan or magnetic resonance imaging (MRI) can help to define the extent of the lesion, and also exclude compartment syndrome. Tissue biopsy shows necrosis of dermis and subcutaneous tissue with focal vascular thrombosis and inflammation in the muscles in the acute phase.

There is no specific treatment for Nicolau Syndrome, but patients are managed symptomatically depending on the extent of the lesion and its complications^{1,2}. Tissue damage may however be reversible in the acute phase¹. Treatment involves pain control^{1, 2}, topical^{1, 19} or oral steroids²⁵ for the

inflammation, antibiotics for superimposed infections^{5,6,8}, vasoactive agents e.g. pentoxifylline¹⁸ to counteract the vasospasm, anticoagulants e.g. heparin¹⁸ and hyperbaric oxygen¹⁸ which have been found to be helpful. Wound debridement and dressing are done¹. The wounds usually heal over several months resulting in an atrophic scar^{1, 2, 3}. Skin grafting or flaps if required can be done. Physiotherapy is also employed.

Prevention is essential as no specific treatment exists. Steps to be taken to avoid Nicolau Syndrome include^{5, 7,21,26,27}:

1. Using a long enough needle to reach the muscles when giving IM injections so as to avoid subcutaneous injections of intramuscular preparations. Recommended length of needle for a 90kg patient is 5 -7.5cm and for a 45kg patient 3 -4cm.
2. Injections on the buttock should be given in the upper outer quadrant to avoid injury to the sciatic nerve.
3. Aspiration should always be done before medication is injected into the muscle to avoid intravascular injections.
4. The Z-track method of giving injections should be used where the skin and subcutaneous tissues are retracted from the site of the injection. This de-aligns them from the underlying muscle. The needle is inserted at 90 degrees, the injection is given and the needle withdrawn slowly and smoothly at a 90-degree angle. The finger is then released to trap the medication inside the muscle and minimize subcutaneous injection by blocking the tract.
5. Volumes larger than 5 mls to be injected using the Z-track method should be divided and administered at different sites.
6. Repeated injections at the same site should be avoided
7. Cold compress application after IM injection should be avoided.
8. IM injections should be avoided in obese patients if possible
9. Injections should be stopped if patient complains of excessive unexpected pain during the procedure

Conclusion

Administration of parenteral medication particularly via the IM route can lead to debilitating complications and hence unnecessary injections should be avoided. Awareness of Nicolau Syndrome among health care practitioners should be raised. As no specific treatment exists, the necessary steps to prevent its occurrence should be taken especially when IM injections are administered.

References

1. Senel E. Nicolau syndrome as an avoidable complication. *J Family Community Med.* 2012; 19: 52–53.
2. Engin Senel Nicolau Syndrome: a review of the literature. *Clinical medicine insights: Dermatology* 2010; 3: 1 - 4.
3. Kwang – Kyoun K. Nicolau Syndrome in patient following diclofenac administration: A case report. *Ann Dermatol* 2011; 23: 501 - 503
4. Kienast AK, Mentze D, Hoeger PH. Nicolau's syndrome induced by intramuscular vaccinations in children: report of seven patients and review of the literature. *Clin Exp Dermatol.* 2008; 33:555-558.
5. Lie C, Leung F, Chow SP. Nicolau syndrome following intramuscular diclofenac administration: A case report. *J Orthop Surg (Hong Kong)* 2006; 14:104–107
6. Ezzedine K, Vadoud-Seyedi J, Heenen M. Nicolau syndrome following diclofenac administration. *Br J Dermatol.* 2004; 150:385–387
7. Senel E, Ada S, Gulec AT, et al. Nicolau syndrome aggravated by cold application after i.m. diclofenac. *J Dermatol.* 2008 ;35:18-20
8. McGee AM, Davison PM. Skin necrosis following injection of non-steroidal anti-inflammatory drug. *Br J Anaesthesia* 2002; 88:139–140
9. Segreto F. Nicolau's Syndrome complicated by atypical necrotising fasciitis. *Arch Plast Surg* 2013 ; 40:267 – 268
10. Kim SK, Kim TH, Lee KC Nicolau Syndrome after Intramuscular Injection: 3 Cases *Arch Plast Surg* 2012; 39:249-252
11. Kılıç I, Kaya F, Özdemir A, et al. Nicolau syndrome due to diclofenac sodium (Voltaren®) injection: a case report. *Journal of Medical Case Reports* 2014, 8:404
12. Murthy S C, Siddalingappa K, Suresh T. Nicolau's syndrome following diclofenac administration: A report of two cases. *Indian J Dermatol Venereol Leprol* 2007; 73:429-431
13. Luton K, Garcia C, Poletti E, Koester G. Nicolau Syndrome: three cases and review. *Int J Dermatol* 2006; 45: 1326–1328
14. Lee DP, Bae GY, Lee MW, Choi JH, Moon KC, Koh JK. Nicolau syndrome caused by piroxicam. *Int J Dermatol.* 2005; 44:1069–1070
15. Guarneri C, Bevelacqua V, Polimeni G Embolia cutis medicamentosa (Nicolau Syndrome) *Clinical Picture Q J Med* 2012; 105 : 1127 – 1128
16. Silva a, Ton A, Loureiro T et al Late development of Nicolau syndrome – case report *An Bras Dermatol.* 2011; 86(1):157 -159
17. Cherasse A, Kahn MF, Mistrh R, et al. Nicolau's syndrome after local glucocorticoid injection. *Joint Bone Spine.* 2003; 70:390–392

18. Ocak S, Ekici B, Cam H, et al. Nicolau syndrome after intramuscular benzathine penicillin treatment. *Pediatr Infect Dis J.* 2006;25(8):749
 19. De Sousa R, Dang A, Rataboli P V. Nicolau syndrome following intramuscular benzathine penicillin. *J Postgrad Med* 2008; 54:332-334
 20. Noaparast M, Mirsharifi R, Elyasinia F, et al Nicolau Syndrome after Intramuscular Benzathine Penicillin injection *Iran J Med Sci* 2014;39: 577 - 579
 21. Karimi M, Owlia MB. Nicolau Syndrome following Intramuscular Penicillin Injection. *Journal of the College of Physicians and Surgeons Pakistan* 2012;22:41-42
 22. Humphries D. Embolia cutis medicamentosa after polidochanol injection of neovessels in Achilles tendinosis. *Grand Rounds* 2013; 13: 12 – 16
 23. Martínez-Morán C, Espinosa-Lara P, Nájera L, et al. Embolia Cutis Medicamentosa (Nicolau Syndrome) After Glatiramer Acetate Injection. *Actas Dermosifiliogr.* 2011; 102:742-744.
 24. Madke B, Kar S, Prasad K, et al A fatal case of Nicolau syndrome. *Indian J Paediatr Dermatol* 2014; 15:92-93
 25. Hamilton B, Fowler P, Galloway H, Nebojsa P. Nicolau syndrome in an athlete following intramuscular diclofenac injection. *Acta Orthop. Belg.* 2008; 74: 860-864
 26. Dadaci M, Altuntas Z, Ince B, et al. Nicolau Syndrome after Intramuscular Injection of Non-Steroidal Anti-Inflammatory Drugs (NSAID) *Bosnian Journal of Basic Medical Sciences* <http://dx.doi.org/10.17305/bjbms.2015.1.190>
 27. Pullen RL Jr. Administering medication by the Z-track method. *Nursing.* 2005; 35:24.
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GASTRIC DIVERTICULUM: A RARE CAUSE OF DYSPEPSIA

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Summary

Objective: Diverticular disease of the stomach is uncommon and rarely suspected in all cases of upper gastro-intestinal pathology. The aim of this report is to demonstrate gastric diverticulum as an uncommon cause of upper gastrointestinal symptoms. The literature in Ghana shows no documented record; and we therefore seek to highlight gastric diverticulum as a possible differential diagnosis when evaluating patients with dyspepsia.

Case Report and Intervention: We present a case of a 26 year old man who presented to the outpatient clinic with a 2-year history of recurrent epigastric pain and dyspepsia. This patient had previous upper

gastrointestinal endoscopies which were normal. The recurrence of his symptoms necessitated a referral to Korle-Bu Teaching Hospital, a tertiary hospital, where the diagnosis of gastric diverticulum was confirmed at endoscopy. He was managed with proton pump inhibitors. Treatment is largely conservative, except in complicated cases such as bleeding and perforation where surgery is indicated.

Conclusion: Gastric diverticulum is rare in Ghana. A high index of suspicion is required to make a diagnosis especially in symptomatic patients with apparently normal gastric mucosa at endoscopy.

Key Words: Gastric diverticulum, Epigastric pain, Dyspepsia, Proton pump inhibitors, Endoscopy.

Introduction

Diverticular disease occurs commonly in the large intestine. Less commonly it may be found in other parts of the gastro-intestinal tract. Its presence in the stomach is rare.

The world-wide reported incidence of gastric diverticulum is estimated to be between 0.01 to 2.6% depending on the method of detection¹. Incidence at autopsy is about 0.02% while by gastrointestinal contrast studies it is about 0.04%². Males and females are equally affected and the age at presentation is usually between 20 to 60 years. Gastric diverticulum can however affect children³

There are two types of diverticula namely, congenital or true diverticula and acquired or false diverticula. The congenital type develops during the third to seventh week of embryogenesis where the fusiform stomach is transformed into an adult form. A 90° rotation of the stomach occurs together with the duodenum, pancreas and dorsal mesentery. The dorsal mesentery then fuses with the posterior body wall. A herniation of the posterior wall of the stomach through the dorsal mesentery prior to fusion results in a diverticulum⁴.

Acquired diverticulum is thought to result from increased intraluminal pressure or external traction of an inflamed or diseased portion of the stomach wall⁴.

Most patients with gastric diverticula are asymptomatic. However, few patients present with symptoms. The commonest symptom is vague upper abdominal pain⁵. Other symptoms include dyspepsia, nausea, vomiting, hematemesis and weight loss⁶. Significant numbers of patients with gastric diverticulum also have other gastrointestinal conditions that may explain their symptoms⁵.

Gastric diverticulum is diagnosed by upper gastrointestinal endoscopy or upper gastrointestinal contrast studies. Occasionally abdominal computer tomography scan may be the mode of diagnosis in complicated cases such as perforation with abscess formation^{4, 5}. Many cases are diagnosed while patients are undergoing endoscopy for other reasons.

Currently there is no clearly defined protocol for treatment of asymptomatic cases. However in symptomatic cases, proton pump inhibitors or histamine receptor blockers are administered for 2-4 weeks. In complicated cases such as large diverticulum of more than 4cm in size⁵; bleeding, perforation and malignant change, surgical intervention is employed⁷ with laparoscopy being the preferred approach^{5, 7}. Intra-operative gastroscopy may be required to assist in locating the diverticulum.

CASE REPORT

A 26 year old man was referred for endoscopy from a district hospital on account of suspected peptic ulcer disease. He had a 2-year history of epigastric pain associated with meals. Two previous upper gastrointestinal endoscopies performed on account of his symptoms had been reported as normal.

There was no history of non-steroidal anti-inflammatory drug abuse or significant alcohol use. He had no history of hematemesis, coffee ground vomitus or melaena. His symptoms over the previous 2 years

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Conflict of Interest: None declared

had been relieved by taking proton pump inhibitors for one week. The pain worsened a week prior to presentation and necessitated the referral for further evaluation at the Korle-Bu Teaching Hospital.

On examination, he looked well hydrated, and was not pale or jaundiced. His blood pressure was 120/70mmHg and had a pulse of 84 beats per minute with good volume. His respiratory and cardiovascular systems were normal. The abdomen was soft with mild tenderness at the epigastrium but no guarding or rebound tenderness. He had no melaena on digital rectal examination. His full blood count showed haemoglobin concentration of 12.3g/dl and a normal white cell count. He was prepared for gastro-duodenoscopy the next morning.

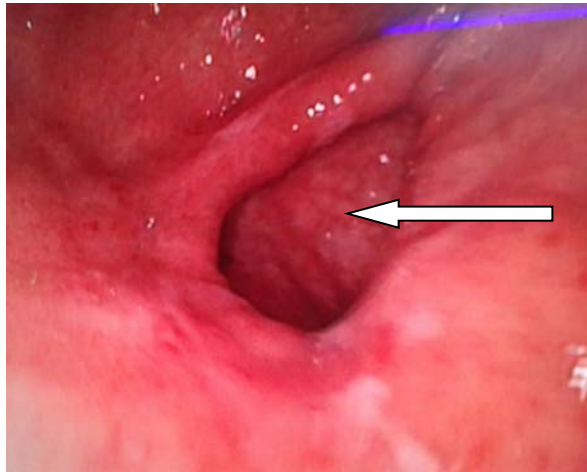


Figure 1: Esophago-Gastro-Duodenoscopy showing moderate proximal gastritis and a 6cm diverticulum (arrow) at the fundus of the stomach.

There were no ulcers, tumors or active bleeding seen. He was subsequently managed on oral proton pump inhibitors for four weeks with good clinical response.

DISCUSSION

Gastric diverticulum is an uncommon condition encountered in clinical practice. It may be congenital or acquired. Congenital forms of gastric diverticulum are usually found near the esophagogastric junction and constitute about 72% of all cases⁸. It is a true diverticulum. The acquired diverticulum is a false diverticulum with only mucosa and submucosa forming its wall⁹. This patient's diverticulum was located at the fundus, the region normally associated with the congenital form of gastric diverticulum. It measured 6cm in diameter, larger than the commonly reported size of 1-3cm in the literature⁷.

Gastric diverticulum is usually asymptomatic with a few patients having symptoms of dyspepsia, epigastric pain, foul smelling belching. Other symptoms may be due to complications such as bleeding and perforation. Our patient had a 2-year history of dyspepsia for which two previous endoscopy findings were normal.

As we experienced in this patient, the diagnosis of gastric diverticulum usually occurs as an incidental finding at endoscopy performed for other reasons¹⁰; in this case, for peptic ulcer disease. This collaborates the trend in the literature regarding the diagnosis being an incidental finding⁸, rather than a suspected diagnosis. The patient's symptoms improved after four weeks' treatment with proton pump inhibitors for proximal gastritis; the diverticulum may probably be a risk factor. It is unclear whether there is any association between gastritis and diverticulum as we found only one report in the literature where both conditions coexist¹¹. He has since not had any symptoms within the last eight months. Our approach towards this patient is conservative even though he has a large diverticulum. The decision for surgical intervention in large symptomatic diverticula lacks adequate evidence in the literature, although it appears to be a risk factor for complications. Some authors recommend surgical resection when the diverticulum is large, symptomatic or complicated by bleeding or perforation¹². At endoscopy, the scope can be used to distend the diverticulum to mimic the patient's symptoms^{4, 13}. This helps in the selection of symptomatic patients who may benefit from surgical resection. We intend to follow him up routinely once a year⁶, in order to diagnose any complication that may occur. Our approach is informed by the belief of some authors that, the need for surgical resection for symptomatic large diverticula depends on the severity of patients symptoms⁴. In our patient, we did not estimate the symptoms to be severe enough to warrant resection at this stage.

CONCLUSION

Gastric diverticulum is a rare clinical entity which might explain a few of the symptoms of the upper gastro-intestinal tract. Treatment is largely conservative except in large symptomatic and complicated cases such as perforation and bleeding.

A high index of suspicion is required in the diagnosis of gastric diverticulum, especially when a patient presents with recurrent epigastric pain and foul smelling belching which subsides on therapy with proton pump inhibitors. In many instances, an unsuspecting endoscopist may miss the diagnosis. Long term follow up is recommended so as to identify possible complications.

REFERENCES

- Schiller AH, Roggendorf B, Delker-Wegener S, Richter K, Kuthe A. [Laparoscopic resection of gastric diverticula: two case reports]. *Zentralblatt Für Chir.* 2007 Jun;132(3):251-5.
- Palmer ED. Gastric diverticula. *Int Abstr Surg.* 1951 May;92(5):417-28.
- Hajini FF, Husain M, Bhat A, Bukhari SI. Gastric diverticulum a rare endoscopic finding. *BMJ Case Rep.* 2014; 2014.

4. Rashid F, Aber A, Iftikhar SY. A review on gastric diverticulum. *World J Emerg Surg WJES*. 2012; 7(1):1.
 5. Marano L, Reda G, Porfidia R, Grassia M, Petrillo M, Esposito G, et al. Large symptomatic gastric diverticula: two case reports and a brief review of literature. *World J Gastroenterol WJG*. 2013 Sep 28;19(36):6114–7.
 6. DuBois B, Powell B, Voeller G. Gastric diverticulum: “a wayside house of ill fame” with a laparoscopic solution. *JSLs J Soc Laparoendosc Surg Soc Laparoendosc Surg*. 2012 Sep; 16(3):473–7.
 7. Muis MO, Leitao K, Havnen J, Glomsaker TB, Søreide JA. Gastric diverticulum and halitosis-A case for surgery? *Int J Surg Case Rep*. 2014;5(7):431–3.
 8. Rodeberg DA, Zaheer S, Moir CR, Ishitani MB. Gastric diverticulum: a series of four pediatric patients. *J Pediatr Gastroenterol Nutr*. 2002 May;34(5):564–7.
 9. Prochotský A, Hlavčák P, Okoličány R, Skultéty J, Sekáč J, Huřan M, et al. [Diverticulum of the greater curvature of the stomach as a cause of anaemia]. *Rozhl V Chir Měsíčník Českoslov Chir Spol*. 2012 Sep; 91(9):481–5.
 10. Eitzen K, Eslick GD, Daneshjoo R. Dyspepsia and gastroesophageal reflux symptoms predominate in gastric diverticulum. *J Dig Dis*. 2012 Jun; 13(6):335–6.
 11. Mahafza WS, Taib AA, Shahait AD, Al Awamleh A. Chronic gastritis in a gastric diverticulum misdiagnosed as a left adrenal mass. *Indian J Surg*. 2015 Apr; 77(Suppl 1):150–2.
 12. Mohan P, Ananthavadivelu M, Venkataraman J. Gastric diverticulum. *CMAJ Can Med Assoc J*. 2010 Mar 23; 182(5):E226.
 13. Anaise D, Brand DL, Smith NL, Soroff HS. Pitfalls in the diagnosis and treatment of a symptomatic gastric diverticulum. *Gastrointest Endosc*. 1984 Feb; 30(1):28–30.
-

RURAL HEALTH: FROM DISEASE ERADICATION TO MEDICAL FIELD UNITS

During the 1930's, reported rates of trypanosomiasis (Sleeping sickness) and yaws increased. Along with malaria, yaws was considered to be one of the biggest contributors to ill health in the country, with the heaviest burden in the north but a significant burden everywhere. In Accra, Cape Coast, Sekondi and Kumasi, 30% of children reporting to infant welfare centres had yaws. In 1937, the government undertook a trypanosomiasis control programme and 1944 a yaws eradication campaign was launched. Both campaigns were enormously successful in reducing the incidence of disease.

Governor Burns advocated for the continuation of medical services to rural areas as the eradication campaigns reached an end. He assigned personnel from the eradication campaigns to medical field units and arranged for them to receive additional training in the identification of several common diseases and pathogens in rural areas (such as malaria, guinea worm, bilharzias, leprosy) as well as training in basic vaccination and laboratory work. Headquarters was at Kintampo, centrally located for rural outreach work. Drs. Waddy and Saunders provided the leadership for the organization and development of the Medical Field Units.

Doctors such as Dr. Akiwumi, M.A. Barnor, and Frank Grant undertook groundbreaking medical research based out of the Medical Field Units, including the gathering of data on bilharzias, onchocerciasis, and guinea worm.

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EXAMPLES

Article

McLendon WW. A historical perspective as a compass for the future of Pathology. Arch Pathol Lab Med 1986; 110: 284-288.

Book

Talbot CH. Medicine in Medieval England. Oldbourne, London. 1926 p 120-136.

Book Chapter

Phillips SJ, Whisnau JP. Hypertension and stroke. In: Laragh JH, Brenner BM, editors, Hypertension: pathophysiology, diagnosis and management. 2nd Ed. New York: Raven Press, 1995, p465-478.

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