COMMENTARY

SPONTANEOUS MASSIVE FETOMATERNAL HEMORRHAGE

Transfer of fetal blood into maternal circulation occurs in most if not every pregnancy. This phenomenon was first proposed by Wiener in 1948 and later confirmed by Chown in 1954[1]. In the absence of antecedent events, the volumes of fetal blood involved are relatively small. Fetomaternal Hemorrhage occurs in 50% of pregnancies and in 1% of cases, the volume will exceed 40 ml[2]. Fetomaternal Hemorrhage generally refers to the entry of fetal blood into maternal circulation before or during delivery. Spontaneous massive fetomaternal hemorrhage is a relatively uncommon phenomenon which is said to have occurred when fetal blood loss into the maternal circulation is more than 150 ml or more than half the fetal blood volume[3]. In spontaneous FMH, there is no preceding history of trauma or clinicopathologic evidence of abruption.

There is no universal consensus on the definition of what constitutes a massive fetomaternal bleed. Massive fetomaternal Hemorrhage is commonly defined as bleed of more than 20% of fetal blood volume or a bleed associated with a Middle cerebral Artery Peak Systolic Velocity (MCA PSV) of more than or equal to 1.5 MoM[4]. These definitions are generally used because a bleed of more than 150mls is associated with severe fetal morbidity and mortality whiles an MCA PSV of the magnitude above is associated with moderate to severe fetal anemia. Some authorities also define it as a bleed of 30 ml to 150 ml of fetal blood[2]. Some experts however believe the interpretation of what constitutes a massive fetal bleed should be made with the fetal size and gestational age in mind. Fetoplacental blood volume is approximately 120 ml/kg estimated fetal weight before 32 weeks and 100 ml/kg after 32 weeks.

Spontaneous Massive FMH can be acute or chronic. In the case of an acute episode, it can result in a rapid fetal hemodynamic collapse and death. In chronic cases on the other hand, it can lead to severe anemia and hydrops.

FMH occurs mostly in the third trimester. Hemorrhage occurs mainly across the terminal villi at the vascular syncytial membranes which consists almost entirely of capillary membranes with little or no intervening stroma. The exact pathogenesis of fetomaternal hemorrhage however remains unclear. In a study that looked at the histology of placentae, retroplacental hemorrhage, intervillous thrombi and infarction within the placental bed increased the likelihood and extent of the hemorrhage[5].

Spontaneous massive FMH can occur at any time during pregnancy. In some cases, fetal death may be the only presenting sign. In a massive nonlethal FMH, the presenting sign may be an abnormal fetal heart rate pattern or a maternal report of a decreased perception of fetal movement. In chronic cases, fetal anemia may lead to hydrops. Further still, in some cases however, the diagnosis is retrospective since there are no signs or symptoms. In a review of cases of severe fetomaternal hemorrhage, 26.8% of patients presented with decreased perception of fetal movements[6]. In women with decreased perception of fetal movements, fetal heart rate patterns such as a sinusoidal pattern, absence of accelerations, recurrent late decelerations and fetal tachycardia should prompt one to think about the possibility of a FMH[7]. In a case series where fetuses had decreased body movement, evaluation of the middle cerebral artery peak systolic velocity was found to be a useful predictor of fetomaternal hemorrhage. In some cases, FMH presented as an unexplained neonatal anemia[8].

In order to detect a massive FMH, one should have a high index of suspicion. The evaluation of a massive FMH includes a detailed history, clinical examination and investigations. Tools used in investigating these cases will include cardiotocography, ultrasonography, and the Kleihauer Betke Assay or flow cytometry. Testing for FMH should be done when women present with decreased perception of fetal movements and also show signs of fetal anemia. These signs may include a sinusoidal FHR pattern, an elevated MCA PSV or fetal hydrops on ultrasound. In some centers, testing for FMH is done in all cases of reduced perception of fetal movement.

Of the various investigations employed, the Kleihauer Betke Test and Flow Cytometry are the definite tests used to quantify the extent to FMH. The Kleihauer Test has been the main test for diagnosis in most laboratories. It involves the separate counting of adult Hemoglobin A cells and fetal hemoglobin F cells. The Kleihauer Test is reported as a percentage. In most cases, maternal blood is assumed to be 5000 mls. Thus, if the test result is 1%, the extent of FMH will be 0.01 x 5000 mls which is 50 mls[9]. Flow cytometry is the other method of assessing FMH. It has been shown to be more accurate, more reproducible and less labour intensive. Many laboratories now prefer it to the Kleihauer Betke Test[10].

There are very few evidence based guidelines on the management of massive spontaneous FMH. This is generally because the condition is not quite common and the available evidence in literature is mostly limited to case series. Guidelines are therefore generally based on expert opinion.

Management depends on fetal status as determined by the various tests, the degree of anemia and the gestational age. Care of these fetuses is multidisciplinary. Care includes the Maternal and Fetal medicine specialist, Blood Transfusion specialist, Hematologist and Neonatologist.

Fetuses are generally divided into those with reassuring as against those with nonreassuring tests. Non reassuring tests will typically include sinusoidal waveforms and others as described above[7]. Viable fetuses with nonreassuring tests will typically require delivery.

For the fetuses with reassuring tests (Biophysical Profile and NST) diagnosed with fetal anemia (MCA PSV ≥ 1.5 MoM) and with massive FMH (> 20%), management will generally depend on gestational age. Fetuses above 32 weeks can be delivered and offered transfusion as neonates.

For the fetuses that are viable but below 32 weeks, serial intrauterine transfusion with weekly monitoring is the available option.

In these cases, when there is evidence of ongoing FMH as evidenced by increasing MCA PSV or evidence on repeat Kleihaeur, delivery will be considered.

In low resource settings where the facilities for intrauterine transfusion are unavailable, preterm delivery and neonatal transfusion may have to be considered in most of these cases[11].

For the fetuses with reassuring tests (Biophysical Profile and NST) diagnosed with fetal anemia (MCA PSV ≥ 1.5 MoM) but without massive FMH (<20%), they can be followed up closely with serial FMH testing and fetal assessment. If FMH worsens, they can be treated as above, otherwise, they are carried to term and delivered.

Most experts recommend Caesarean delivery to prevent any further deterioration of the fetus due to fetomaternal hemorrhage.

In most of these cases, the rhesus negative mother is also at risk of alloimmunization due to exposure to red cell antigens. The appropriate dose of anti-D should be given based on the extent of FMH. The outcome of the fetus often depends on the rapidity of the FMH and the extent of blood loss. Long-term follow however is less well described. Even though recurrence has been reported in case reports, there is no clear recommendation on follow up.

In conclusion, massive fetomaternal hemorrhage can lead to severe perinatal morbidity and mortality, a high index of suspicion is needed to make the diagnosis.

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