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Case Report

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Massive Fetomaternal Hemorrhage: Case Report and Literature Review

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Case Report

This is the case report of our patient, a 43-year-old woman who presented herself with decreased fetal movements at 31 weeks and 6 days of amenorrhea. The patient, G3P1, had a history of vaginal delivery at term in 1998. Her pregnancy results of an oocyte donation. The patient is HIV-positive with a viremia undetectable under antiretroviral therapy. Her blood type is O Rhesus-positive.

On August 22, 2019, she presented herself to an obstetrical ultrasound center at 31 weeks and 6 days of amenorrhea for decreased fetal movements for the last 48 hrs.

The patient had an uncomplicated pregnancy so far, with a negative triple test for trisomy, and normal morphology ultrasounds. The screening test for gestational diabetes was negative.

She had no history of any significant complaints, and reported no recent trauma.

Ultrasound showed a fetus in transverse presentation with an estimated fetal weight of 1755 g (31st percentile). Active fetal movements were markedly decreased on ultrasound, but the amount of amniotic fluid was normal. Umbilical and uterine dopplers were normal. The peak systolic of the middle cerebral artery (MCA) was 54.8 cm/s, which corresponds to 1.24 MoM (Figure 1).

Ultrasound indicated the presence of a small amount of ascites (Figure 2 and Figure 3).

The patient was then sent in emergency to the maternity ward of the Universitary center for a cardiotocogram and serology tests.

Upon arrival, the cardiotocogram (Figure 4) showed a sinusoidal pattern for 45 minutes. An emergency cesarean delivery was performed. Outcome was a baby girl in breech position, hypotonic, with an Apgar score of 1/5/7 and a birth weight of 1855g. The baby appeared very pale. The arterial pH was 7.26, and the venous pH was 7.36. The hemoglobin of the newborn was 28 g/L.

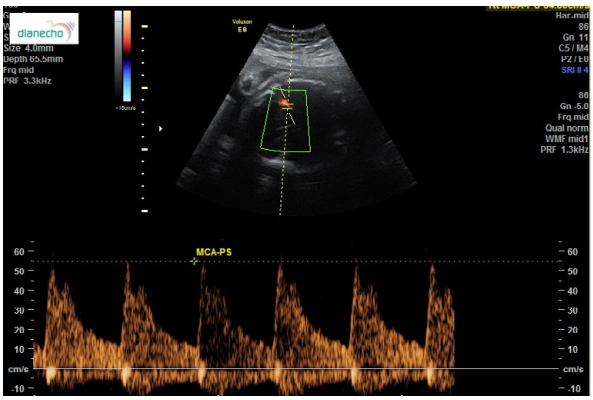

Figure 1: Ultrasound showing an MCA peak systolic velocity of 54.8 cm/s, or 1.24 MoM. It should be noted that the criteria for correct measurements are not fully met (see below).

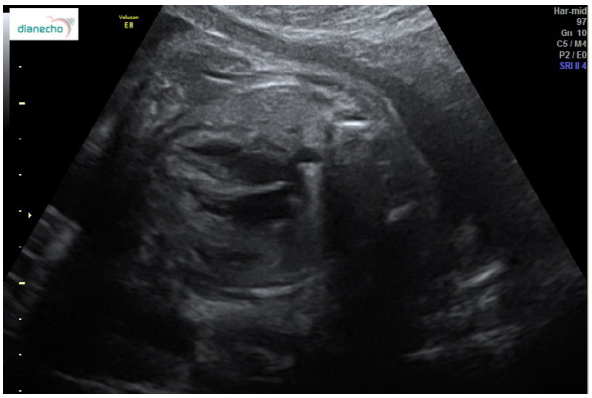

Figure 2: Discrete pericardial effusion.

Notice that the baseline rhythm is 140 bpm, with a classic sinusoid-like pattern, i.e. regular and wave-like, with a frequency of between 2-5 cycles a minute. The oscillations are of 5bpm in this case.

The Kleihauer test performed on the mother's blood was highly

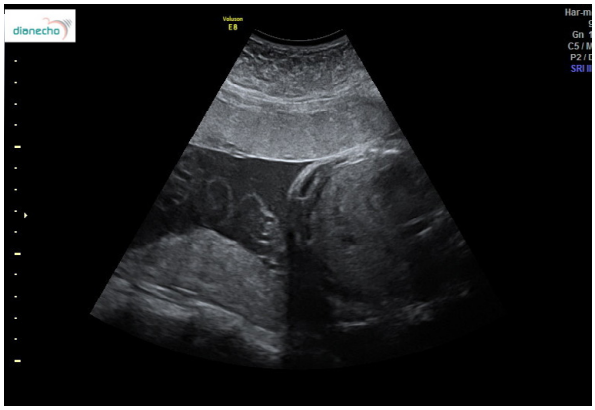

Figure 3: Discrete perihepatic effusion.

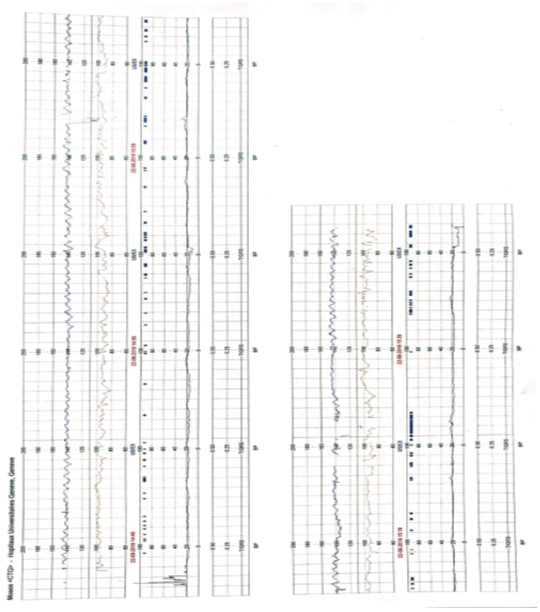

Figure 4: Cardiotocogram performed upon admission to the emergency department (Paper unwinding speed: 2 cm/min).

positive at 34%, which confirmed the diagnosis of fetomaternal hemorrhage.

The newborn showed poor adaptation at birth, with tachycardia and a mean arterial pressure of 28 mmHg. She was transferred to the neonatology unit where she was transfused with 10 cc/kg O Rhesus-negative blood over 30 minutes, and then 10 cc/kg over 2 hrs, allowing the correction of anemia at 134 g/L. She regained color and the hemodynamic parameters improved. At birth the baby presented diffuse and significant edemas. A cardiac and abdominal ultrasound was performed, and revealed pericardial, bilateral pleural and perihepatic effusions attributed to low oncotic pressure, they were addressed by the administration of furosemide and erythrocyte transfusion. The evolution was then favorable and the child was discharged home on day of life 33, or 36 weeks and 4 days corrected gestational age.

Pathological analysis resulted in a normotrophic placenta weighing 353 g, site of fetal erythroblastosis and showing heterogeneous villous maturation with focal villous edema. The umbilical cord showed no abnormality.

Discussion

Physiopathology

Physiologically, there is a bidirectional flow of red blood cells through the placenta during pregnancy and childbirth. Fetomaternal hemorrhage (FMH), however, represents a massive loss of fetal red blood cells in the maternal bloodstream. The mechanism is still poorly understood and some authors put forward the hypothesis of a rupture of the trophoblastic barrier, allowing the passage of fetal red blood cells into the mother's bloodstream [1].

Its exact incidence is not well known but it is estimated to affect about 1/1500 pregnancies, many cases are probably unrecognized, especially during miscarriage or intrauterine death.

Etiologies

Some of the causes of this phenomenon are known, such as the external cephalic version, placental detachment, amniocentesis, abdominal trauma, or some placental tumors [2]. Spontaneous fetomaternal hemorrhage is defined as the transfer of fetal blood into the maternal circulation, with no identified etiology, and accounts for 80% of FMH [1]. In most of those spontaneous FMH only a small volume of blood is transferred, without hemodynamic consequences on the newborn, but it can cause maternal alloimmunization [3].

Severity

In the literature, several criteria are defined to assess the severity of a fetomaternal hemorrhage.

Severe FMH is usually described as the loss into the maternal circulation of more than 30 ml of fetal blood. This threshold is used as it represents the volume for which a 300 µg of Rh(D) Immune Globulin (RhoGAM) is sufficient to prevent maternal alloimmunization. However, it does not reflect the level of severity for the newborn.

Other authors have proposed using thresholds of 80 ml or even 150 ml [4]. It is generally accepted that from 20 ml/kg of transfused blood, severe antenatal and neonatal complications can occur [5].

Rather than using this absolute value, some authors suggest the use of a percentage of fetal blood loss volume, for example a limit of 20% of the fetal blood volume. It is however difficult to estimate the circulating blood volume of a fetus which makes this criterion difficult to use.

Our case reports a massive FMH, with a birth hemoglobin of 28 g/l. It is generally accepted that a positive Kleihauer test of 1%, is equivalent to 5 ml of fetal blood loss in the maternal blood. A positive test of 34 ‰ is therefore equivalent to 170 ml. Assuming that the average fetal circulating blood volume is of 100 ml/kg, our fetus having a birth weight of 1855 g would therefore have lost 170 ml out of 185 ml, i.e. 90 % of its circulating volume!

The severity of this condition also depends on the speed at which the hemorrhage develop itself. It can be acute and cause significant hemodynamic disorders, resulting in rapid in utero death; or chronic causing fetal hydrops or anemia, with fetus adapting to compensate for the blood loss.

In our case, the presence of ascites and a perihepatic effusion suggests a subacute fetal hemorrhage.

Other researchers suggest evaluating the severity of the FMH using the MCA peak systolic, with a threshold at 1.5 MoM. It should be noted

that in our case this value was normal with a value of 1.26 MoM (we will come back to this later).

Warning Signs

The most common warning sign found in the literature is a decrease or absence of fetal movements (up to 54 % of the cases). Other findings include abnormal fetal heart rate tracing like a sinusoidal pattern or a lack of accelerations, recurrent late decelerations or fetal tachycardia. The characteristics of a sinusoidal heart rate pattern are the following: a stable baseline heart rate, with regular and symmetric oscillations above and below the baseline and with a frequency of 2 to 5 cycles per min. It presents no normal or reactive episode and is associated with fetal anemia and/or fetal hypoxia.

Diagnosis

Laboratory Tests

There are two quantitative tests used to establish a diagnosis [6]. The first is the Kleihauer-Betke test, which allows to detect the presence of fetal red cells in the maternal blood. Maternal blood is exposed to an acid solution, which destroys red blood cells containing adult hemoglobin. The sample is then stained with erythrosine B. Erythrocytes containing fetal hemoglobin appear red whereas adult red blood cells are colorless. The number of fetal cells is then counted under a microscope by a technician and the results are expressed as the number of fetal red blood cells per 10,000 maternal red blood cells

False positives can occur in case of maternal hemoglobinopathy, which lowers the adult hemoglobin (e.g. thalassemia). Several studies have demonstrated poor reproducibility of the Kleihauer test.

The second test is flow cytometry. With this method the fetal hemoglobin is tagged by fluorescent antibodies and then quantified in an automated way, allowing more precision and easier reproducibility. However, very few hospitals have the necessary analytical infrastructure available to perform this test.

These laboratory tests are widely used in case of maternal trauma or in case of unexplained in utero death. They should also be performed in event of persistent perception of decreased fetal movement, fetal hydrops, increase of the MCA V_{max} or neonatal anemia.

Ultrasound Evaluation

Ultrasound can reveal several signs of fetal anemia. There are some minor and some major signs.

These are the minor signs: abnormally echogenic intestinal loops, visualization of the intestinal loops wall, hepatomegaly, discrete pericardial effusion, and moderate excess of amniotic fluid.

The major signs are the following: fetoplacental anasarca, or increase in maximum systolic velocity in the middle cerebral artery.

Ultrasound is used to assess the severity of anemia. It makes it possible to evaluate fetal well-being, by "biophysical profile" or Manning's test. Four variables are measured: fetal movements, muscle tone, amniotic fluid volume, and presence of fetal breathing movements. However false-negative can occur, and the diagnosis of FMH should not be excluded in case of biophysical profile score of 8/8 [7].

On the contrary, doppler assessment would be an excellent tool to detect fetal anemia. Anemia leads to a decrease in blood viscosity, an increase in cardiac output, as well as a bloodflow redistribution to the noble organs, and this results in an increase of the MCA peak systolic. According to several studies, there is an excellent correlation between the MCA-PSV and the degree of anemia [8,9]. A threshold value of 1.5 MoM is generally accepted as the sign of severe anemia and the sensitivity would be of 100 % [10]. However, in our case report this measure was normal. It should not be forgotten that this measure is difficult, that it must meet strict criteria, and that there is a lot of inter- and intra-observer variation.

For the measure to be correct according to Moise Jr KJ (2008) [10], the fetal head should be in transverse plane, an angle of insonation of less than 15° should be used, ideally closest to zero. A 2-mm Doppler gate should be placed where the vessel bifurcates from the carotid siphon. A falsely low peak value can be obtained with a more peripheral placement of the gate. The PSV should be measured when there is no fetal movement. Three measures should be taken, and the final value should be the highest one.

The PSV from our case report, as shown in Figure 1, does not perfectly meet all these criteria, even though it was measured in a renowned ultrasound center, and can therefore be falsely reassuring.

Moreover, as Huissoud C, et al. (2008) [8], the clinical and biological history of the patient should be taken into consideration when analyzing the MCA-PSV. In fact, the curves used to interpret an MCA-PSV result are based on studies about anti-D alloimmunization. Large prospective studies would be needed to assess the sensitivity and specificity of this measure in case of FMH which is difficult given the low prevalence of this disease [11].

Moreover, in case of FMH urgent medical intervention is generally needed with fetal extraction, studies published about alloimmunization are therefore non-reproducible in the context of this pathology.

We therefore draw our reader's attention to the fact that a normal value of MCA should not exclude the diagnosis of FMH, especially in the presence of a pathological cardiotocogram.

Treatment options

In case of FMH suspicion, emergency delivery and postnatal transfusion is the treatment of choice if the pregnancy is near-term [12]. Some authors recommend cesarean delivery. Others in utero transfusion in case of prematurity. The risks of this procedure are fetal distress, intrauterine death or premature delivery.

The decision must therefore be discussed with the parents, experienced obstetricians and the neonatology team in order to assess the risks and benefits of such an intervention, which must be carried out in a center with the necessary expertise.

It should be noted that in some reported cases there appear to be a risk of recurrent FMH in subsequent pregnancies [13,14]. There is currently no consensus on which surveillance is required in case of FMH (Doppler ultrasound, repeated Kleihauer tests, etc.) Patients should be informed to be attentive to reduced fetal movements [15-18].

Conclusion

In conclusion, fetomaternal hemorrhages are rare and severe causes of neonatal anemia, which can lead to in utero death. Its diagnosis should be considered in the event of persistent perception of decreased fetal movement. Cardiotocogram and Kleihauer-Betke test should be performed, as well as an evaluation of fetal well-being by ultrasound.

Dopplers should be measured, but normal peak systolic values

alone should not exclude the diagnosis. Cases should be recorded in a database in order to carry out retrospective studies on a larger scale and to enable Doppler curves adapted to this pathology to be developed.

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