



ORIGINAL RESEARCH PAPER

Radiology

FETOMATERNAL HEMORRHAGE

KEY WORDS: fetomaternal hemorrhage, fetal hydrops, Doppler, Middle cerebral artery, fetal anaemia

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ABSTRACT

The placenta acts as natural barrier that prevents significant sensitization of the fetal and maternal red cells. Fetomaternal haemorrhage is a rare complication when this barrier is disrupted. The alloimmunization results in fetal anaemia and hydrops. Poor perinatal outcomes are observed if this condition is not anticipated antenatally. By observing the elevated velocities in the middle cerebral artery in the fetus using doppler ultrasound the fetal anaemia can be graded and diagnosed. Maternal history of reduced fetal movements and elevated velocities in the middle cerebral artery thereby assist in diagnosis and therapeutic decision making.

INTRODUCTION:

Massive fetomaternal hemorrhage is a rare cause of fetal decompensation. The diagnosis is rarely made antenatally.

We diagnosed fetomaternal hemorrhage in a 27 yr old primigravida with 36 weeks gestation complaining of decreased fetal movements.

Fetal hydrops and elevated middle cerebral artery velocities were pertinent observations for diagnosing the condition.

The neonate was salvaged with prompt blood transfusion and made steady recovery

CASE:

A 27 yr old Indian primigravida with 36 weeks gestation came with complaints of decreased fetal movements.

she did not have pain, passage of fluid per vaginum or obvious abdominal tightening for contractions.

The patient did not recollect any history of trauma. There was no history of any invasive procedure or drug use. She was a teetotaler with no history of smoking or tobacco chewing.

On admission the patient's vitals were stable. The fetal heart rate tracing revealed sinusoidal pattern with baseline 140b/m.

On ultrasound the fetus showed sluggish movements. There were no structural malformations seen. The fetus was noted to have ascites, pleural effusion and generalized subcutaneous edema fig.1.

There was mild polyhydramnios observed. AFI 16-17cms. The fetal biometry corresponded with the gestational age calculated by the last menstrual period.

Doppler study showed high velocities in the middle cerebral arteries (100cm/s). There was also presence of tricuspid regurgitation fig.2, fig 3. The umbilical arteries and both the uterine arteries revealed normal low resistance waveform pattern.

The observations of fetal hydrops and notably the findings of elevated middle cerebral artery velocities suggested fetal anaemia as a cause for the fetal hydrops.

The patient underwent caesarean section. There was no findings of retroplacental or uterine wall hematoma. The neonatologists were informed to be prepared for resuscitation of an anaemic fetus. The fetus thus delivered was an anaemic male neonate weighing 2.7

kgs and had Apgar score of 5 and 7 at 1 min and 5 min respectively. Infantogram revealed bilateral pleural effusion and subcutaneous edema. Fig.4. The neonate was intubated.

30 and 20 ml of leukocyte free O negative blood was given immediately after birth and 24 hrs after. The neonate showed progressive improvement with resolution of the

Edema. Fig.5. On discharge the infant had normal vitals and did not suffer any neurological sequelae.

Discussion:

The placenta acts as a natural barrier preventing any significant mixing and sensitization of the fetal and the maternal red blood cells. Small amount of fetal blood enters in maternal circulation in all cases without significant consequences.

Fetomaternal haemorrhage is rare complication when this barrier is disrupted. A large fetomaternal haemorrhage can cause fetal anaemia due to alloimmunization which if severe can result in hydrops with poor outcome (1).

Multiple studies have attempted to define clinically significant or massive fetomaternal hemorrhage by taking into account volume of the blood loss from the fetus. When the fetomaternal hemorrhage measured 150 ml or more significant mortality in neonates was observed by Sebring E S (2). In which 15 Of 41 neonates did not survive.

Few of studies have tried to evaluate the outcomes of the acute or the chronic nature over which the fetomaternal hemorrhage occurs. In animal experiment Brace found that in fetal sheep blood loss of over 30% of the estimated total volume of the fetus was better tolerated if it occurred over hours rather than over minutes (3).

However considering the ambiguity about the volume in various studies it seems logical to consider the rate of blood loss and chronicity as the factors that would affect the fetal outcome. A slow fetal maternal haemorrhage would allow the fetus to compensate thus affecting the outcome (4).

There are no inciting or predisposing factors usually identified for the fetomaternal haemorrhage. The various risk factors attributed for the fetomaternal haemorrhage include external cephalic version, abdominal trauma, placental abruption, monochorionic monoamniotic twins, preeclampsia, placental tumors and amniocentesis (5).

The presentation is usually with reduced fetal movements. In one of the recent largest multi-hospital series that evaluated

fetomaternal hemorrhage in 2,19853 neonates,24 had anemia with laboratory evidence fetomaternal hemorrhage (incidence of 1/1960)Decreased fetal movements was the commonest presentation.The outcomes was poor when the Hb was< 5mg/dl and consisted of death,IVH,Periventricular leukomalacia,bronchopulmonary dysplasia,hyoxic ischemic encephalopathy.(6)

Thus Heightened index of suspicion is needed for fetomaternal hemorrhage in cases presenting with persistent reduced fetal movements.(7)

As the consequences of the fetomaternal hemorrhage depend upon the magnitude but also on the acuity of the blood loss.The anemia can be compensated if it is only minimal .

The pregnancy may continue till the term with the delivery of anemic fetus(8).

Severe fetomaternal hemorrhage leads to hydrops fetalis when the fetal hemoglobin deficit is more than 7 gm/dl.This manifests generalised edema ,fetal ascites and pleural effusions.

The fetal compensatory mechanism consists of increasing the cardiac output.Anemia leads to increased cardiac output, due to the hyperdynamic circulation, and a reduction in blood viscosity, both leading to increased blood flow velocity.(9)

These changes in the velocity can be detected in the fetal vessels using doppler ultrasound. An observation of G Marie way back in 1987 while studying the effects of intravascular transfusion on circulation of the fetus ,that the anemic fetus MCA waveforms following transfusion had lower PSV value dawned the idea of using fetal MCA PSV for predicting fetal anemia.A series of papers later confirmed this observation.10

The peak systolic velocity in the middle cerebral arteries reduces as the fetal hematocrit rises thus there exists an inverse relationship between the peak middle cerebral arterial velocities and the fetal hemoglobin.

The Moderate to severe anemia can be detected noninvasively by observing the increase in the velocities in the fetal middle cerebral artery.In a study which consisted of 32 fetuses of mothers having screen positive for parvovirus B19 infection.The middle cerebral arterial doppler evaluation could predict fetal anemia noninvasively in 17 fetuses in whom the doppler showed elevated velocities>1.5MOM.(11).

The standard method of evaluation of FMH is kleihauer betke test.As fetal hemoglobin is more stable than the adult hemoglobin in acidic solution it can be detected and quantified using Kleihauer Betke test.

This test also helps in quantifying the amount of hemorrhage which helps in predicting the outcome and planning corrective transfusion .

CONCLUSION:

Fetal maternal hemorrhage is a rare complication which can lead to fetal decompensation as a result of anaemia caused by alloimmunization.Maternal history of reduced fetal movements and doppler observations of elevated velocities in the middle cerebral arteries assist in diagnosis and therapeutic decision making.

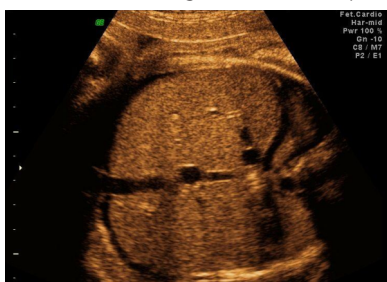


Fig.1a Axial section at the level of the umbilical vein showing hepatomegaly and mild ascites

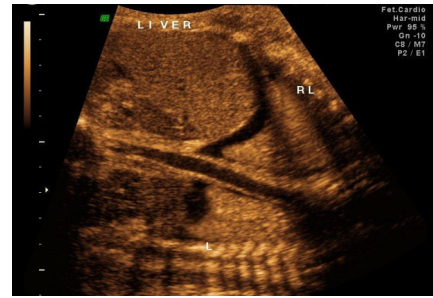


Fig.1b Coronal scan at the level of liver and lung showing pleural effusion.

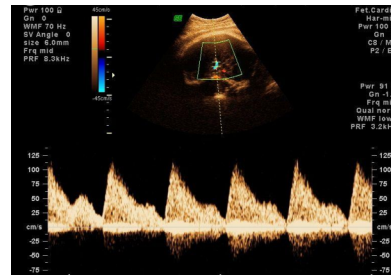


Fig.2 Elevated velocities in the middle cerebral artery



Fig.3 Day 1 neonatal radiograph with anasarca and pleural effusions..

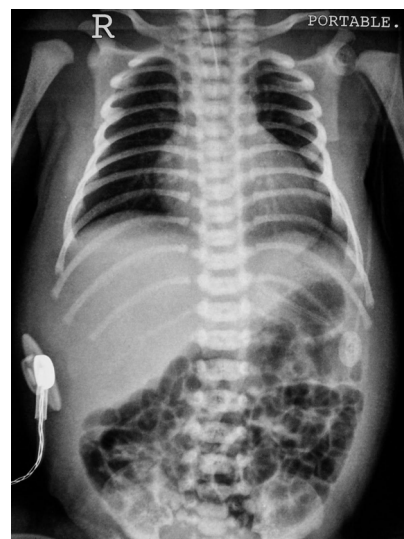


Fig.4 Follow up neonatal radiograph with resolution of edema and pleural effusion.

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