Short Communication

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Fetus in Rh- Isoimmunization

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Abstract

RH isoimmunization is known complication in mother with Rh negative blood group carrying Rh positive fetus. In rh isoimmunization treatment is easier as compare to past because of advancement in ultrasonography and fetal medicine speciality. This article includes pathophysiology in rh isoimmunize fetus ands its managment.

Keywords: RH Negative Mother; Isoimmunization; Hydrops; Anemia; Hypoprotenemia; MCA-PSV.

Rh incompatability is also known as Rh disease, is a condition that occurs when a women with Rh –ve blood type is exposed to Rh +ve fetal blood cells leading to development of Rh antibodies. Rh isoimmunization occurs in Rh –ve mother with Rh +ve fetus. Rh isoimmunization is characterized by obstetric history of repeated pregnancy losses with a progressive worsening output in subsequent pregnancies.

The sensitized mother produce IgG anti D antibody that crosses the placenta and coats D-positive fetal red cell which are then destroyed in the fetal spleen. Hemolysis leads to an increase in indirect bilirubin, fetal anemia and compensatory hematopoiesis in fetal liver and spleen placental edema interfere with placental perfusion and ascites develops, aggravate by reduced albumin production due to further liver damage. Hydrops fetalis is characterized by output cardiac failure, ascites,edema, pericardial effusion and extra medullary hematopoiesis [1]. If untreated 20 to 30 % of the fetuses affected by ethroblastosis die in utero. Kernicterus and jaundice are not component of erythroblastosis fetalis during intrauterine life, because accumulation of the pigment is prevented by its removal through the placental circulation and metabolism in maternal liver however, after birth newborn liver can not effectively handle the large amount of pigment released during the hemolytic process and this leads to rapid increase in serum bilirubin and eventually tissue deposition [2]. Blood production in fetus being at about 3 weeks and Rh antigen has been identified in red cell membrane as early as 38 days after conception [3]. Fetus may get affected as early as from 3 rd week of gestation.

Hepatomegaly, increased placental thickness and polyhydromios often precede the development of hydrops. As liver damage progresses decreased albumin production results in the development of anasarca and effusions [1].

The abnormal collection of fluid in more than one area of the fetal body such as ascites and plural effusion, is termed Hydrops fetalis. It's causes usually are classified as immune and non-immune. In immune hydrops with Rh isoimmunization excessive and prolonge hemolysis causes anemia which in turn stimulate marked marrow erythroid hyperplasia. It also stimulates extramedullary haematopoiesis in the spleen and liver with eventual hepatic dysfunction there may be cardiac enlargement and pulmonary haemorrhage.Fluid collects in the fetal thorax, abdominal cavity, or skin. The placenta is markedly edematous, enlarge and boggy. It contains large, prominent cotyledons and edematous villi. Pleural effusions may be so sever as to restrict lung development, which causes pulmonary compromise after birth. Ascites, hepatomegally, and spleenomegaly may lead to sever labor dystocia. Sever hydropic changes are easily seen with sonography [4].

The precise pathophysiology of hydrops remains

unknown theories includes heart failure from profound anemia and hypoxia, portal hypertention due to hepatic parenchymal disruption caused by extramedullary haemopoisis and decreased colloid oncotic pressure resulting from liver dysfunction and hypoprotenemia. The degree and duration of anemia is the major factor causing and influencing the severity of ascites [5]. Secondary factor include hypoprotenemia caused by liver dysfunction and capillary endothelial leakage resulting from tissue hypoxia both of these lead to protein loss and decreased colloid oncotic pressure wich worsens hydrops [6]. Fetuses with hydrops may die in utero from profound anemia and circulatory failure one sing of severe anemia and impeding death is the sinusoidal fetal heart rate pattern. In addition hydropic placental changes leading placentomegaly can causes preeclampsia ironically, the preclamptic mother may develop sever oedema mimiking that of the fetus this is referred to as the Mirror syndrome. The live born hydropic infant appears pale edematous and limp at birth and usually requires resuscitation.The spleen and liver are enlarged, and there may be widespread ecchymosis or scattered petechiae. dyspnea and circulatory collapse are common.

Once we came across RH immunized baby, investigation for detecting fetal affection are middle cerebral artery peak systolic velocity (MCA-PSV) and the concentration of bilirubin in the amniotic fluid. The MCA PSV is an accurate noninvasive method for diagnosis of fetal anemia. The correlation between the MCA PSV and fetal anemia become stronger as the fetal hemoglobin decreases and also MCA PSV values can be used to predict the fetal hemoglobin concentration. The threshold for the diagnosis of fetal anemia is a value equal to or greater than 1.5 multiples of the median (MoM) for the gestational age abnormally elevated MCA PSV has a sensitivity of 100% and false positive rate of 12% for diagnosis of fetal anemia [7].

Amniotic fluid billirubin analysis is the traditional method for evaluating severity of fetal hemolytic process and for determining the optimal time for intra uterine transfusion (IUT) or for delivery of infant.

Ultrasonography and color Doppler has revolutionized monitoring and decision making in Rh isoimmunized patients. Since all decision making in sensitized patients uses charts based on gestational age, accurate dating of the pregnancy by earliest available parameter, either first trimester crown rump length or second trimester biparietal diameter on ultrasonography is important. Early detection of hydrops is possible by careful serial ultrasonography.

Ultrasonographic features of Hydrops [8]:

- ☞ Ascites
- ☞ Pericardial effusion
- ☞ Hepatomegally
- ☞ Spleenomegaly 3 to 4 times normal
- ☞ Intrahepatic portal vein >5mm
- ☞ Scalp edema
- ☞ Polyhydramnios
- ☞ Placental thickness>4cm

It is very important to diagnose anemia before it becomes severe enough to cause hydrops. Unlike non–hydropic babies, in whom the survival rates are above 90%,in hydropic babies even after intrauterine transfusions, the reversal rate varies between 39-88% indicating a guarded prognosis even after intervention.

Successful management requires a team approach with close coordination among the obstetrician, fetal medicine specialist or ultrasonologist and neonatologist as well excellent laboratory, blood bank and NICU facilities.

Referances

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