CASE REPORT

Hydrops Fetalis–Antenatal Diagnosis

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Abstract

Incidence of immune hydrops fetalis is decreasing with the liberal use of anti-D immunoglobulin, but this condition has not been eradicated. We report here a case of immune hydrops fetalis with meningocele detected on ultrasonography.

Key Words

Hydrops fetalis, Meningocele

Introduction

Immune hydrops fetalis results from hemolysis from isoimmunization. Individuals who lack a specific red cell antigen (like Rh antigen) can potentially produce an antibody when exposed to that antigen. The antibody may prove harmful to the individual in case of a blood transfusion or to a fetus when a mother (Rh negative) conceives. In these cases, the mother could be sensitized if enough erythrocytes from the Rh positive fetus reach her circulation to elicit an immune response. Case report of a pregnant women is presented, where diagnosis of hydrops fetalis with meningocele was established during antenatal period.

Case Report

A 27 year old female, pregnant for second time, reported to the hospital OPD for routine antenatal checkup. Patient was in second trimester of pregnancy. She had history of an abortion in the past. Patient was not given any prophylactic anti-D immunoglobulin therapy after the abortion. The routine blood and urine investigations were normal. Her blood group was B negative. Indirect Coomb's test was positive. Serology for TORCH was negative both for IgG and IgM antibodies. Ultrasonographic examination revealed single live fetus of approximately 17 week of gestational maturity in uterine cavity. Fetal occipital bone revealed a large defect with outpouching of meninges, forming a large meningocele (Fig. 1,2). There was no hydrocephalus. There was bilateral pleural effusion and fetal abdomen showed large amount of ascitic fluid (Fig. 3). Considerable subcutaneous edema of fetal scalp and abdominal wall was also observed (Fig. 4). Placenta

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appeared bulky and amniotic fluid volume was not excessive.

Keeping in view the past history of abortion, Rh negative status of mother and ultrasonographic features described above, a diagnosis of hydrops fetalis with large occipital meningocele was made. As per the desire of the couple, pregnancy was terminated in view of poor prognosis of the fetus. Anti-D immunoglobulin was administered to the patient immediately after termination of pregnancy.

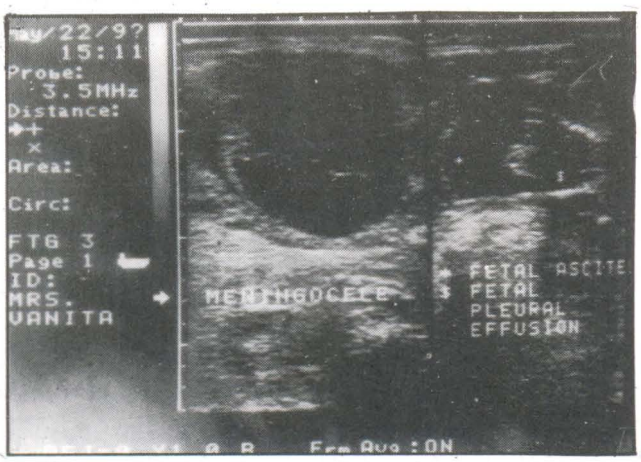

Fig. 1 : Sonogram showing large meningocele, fetal ascitis and pleural effusion.

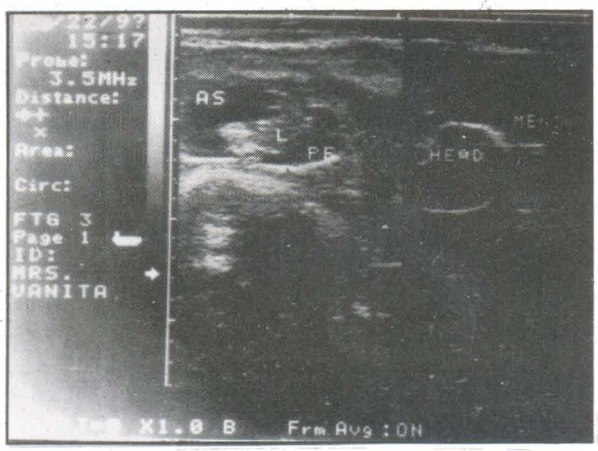

Fig. 2 : Sonogram showing large defect in the fetal occipital bone with outpouching of meninges. It also shows fetal ascitis and pleural effusion.

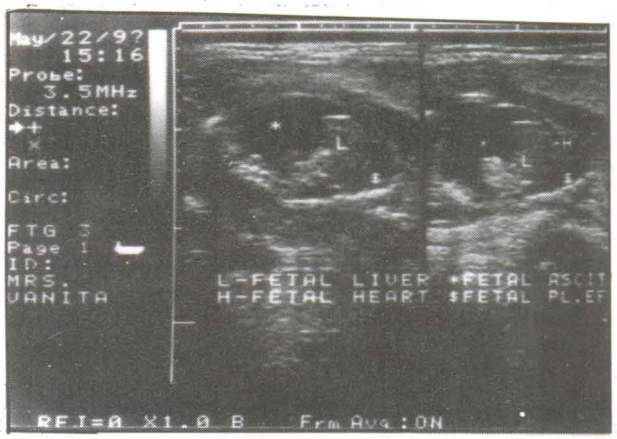

Fig. 3 : Sonogram showing fetal ascitis and pleural effusion.

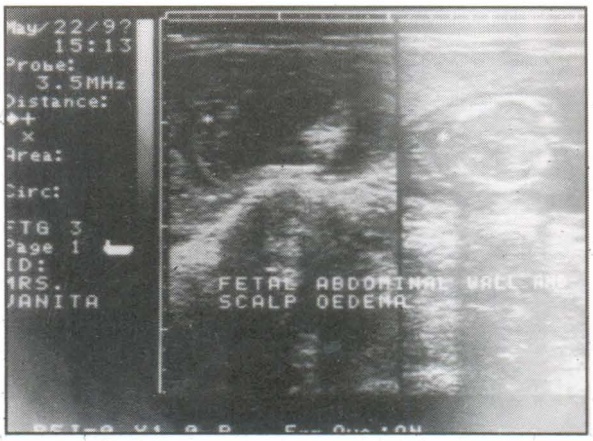

Fig. 4 : Sonogram showing fetal abdominal wall edema and scalp edema.

Discussion

In 1892, Ballantyne established clinicopathological criteria for the diagnosis of hydrops fetalis. Diamond, Blackfan and Baty in 1932, reported that fetal anemia characterized by numerous circulating erythroblasts was associated with this syndrome (1). Levine *et. al.* in 1941, confirmed that erythroblastosis was due to maternal isoimmunization with paternally inherited fetal factors (2). Subsequent development of effective maternal prophylaxis was attributed to Finn and associates of England and Freda and co-worker of United States (3,4).

Pathological changes in the organs of fetus and newborn infant vary with the severity of the process. The severely affected fetus or infant may show considerable subcutaneous edema as well as effusion into the serous cavities - hydrops fetalis. At times, the edema is so severe that the diagnosis can be easily identified using sonography. In these cases, the placenta is also markedly edematous, appreciably enlarged and boggy, with large, prominent cotyledons and edematous villi. Excessive and prolonged hemolysis serves to stimulate marked erythroid hyperplasia of the bone marrow, as well as large areas of extramedullary hematopoiesis, particularly in the spleen and liver leading to hepatosplenomegaly, which may in turn cause hepatic dysfunction (5). Hydrothorax and ascites may be so severe as to compromise respiration after birth or lead to severe dystocia as a consequence of the greatly enlarged abdomen.

Pathophysiology of hydrops remains obscure. Theories of its causation include heart failure from profound anemia, capillary leakage caused by hypoxia from severe anemia, portal and umblical venous hypertension from hepatic parenchymal disruption by extramedullary hematopoiesis and decreased colloid oncotic pressure from hypoproteinemia caused by liver dysfunction. Nicolaides and colleagues concluded that the degree and duration of anemia influence the severity of ascites, and this is made worse by hypoproteinemia. They also hypothesized that severe chronic anemia causes tissue hypoxia with resultant capillary endothelial leakage with protein loss (6).

Fetuses with hydrops may die in utero from profound anemia and circulatory failure. A sign of severe anemia and impending death is a 'Sinusoidal fetal heart rate' pattern. The liveborn hydropic infant appears pale, edematous and limp at birth, often requiring resuscitation. The spleen and liver are enlarged and there may be widespread ecchymoses or scattered petechiae. Dyspnea and circulatory collapse are common.

A single intramuscular dose of 300 µg of D-immunoglobulin is administered routinely to all D-negative, nonimmunized women at 28 to 32 week gestation and again within 72 hours of the birth of a D-positive infant. A similar dose is also given at the time of amniocentesis and whenever there is uterine bleeding, unless the routine dose at 28 to 32 weeks had been given very recently.

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