IMMUNE FETAL HYDROPS - A SEVERE FORM OF RH ISOIMMUNIZATION

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IMMUNE FETAL HYDROPS - A SEVERE FORM OF RH ISOIMMUNIZATION (Abstract). The prevalence of pregnancies with Rh isoimmunization decreased to about 0.6% of all cases due to anti-D immune globulin prophylaxis. Rh incompatibility or Rh isoimmunization pregnancies must be monitored by serial measurements of titers of maternal Rh antibodies and, if possible, by serial Doppler evaluation of peak systolic velocity in the middle cerebral artery. Nowadays, for the detection of fetal blood group and Rh we can use techniques which identify free fetal DNA in maternal plasma. We report the clinical case of a 35 years old woman, admitted to our clinic after not being able to perceive active fetal movement. She proved to be Rhesus negative, with a pathological obstetrical history and through ultrasound examination, the diagnosis of fetal hydrodrops was established. The presence of fetal hydrodrops determines the viability of a small percentage of about 11%, and the long-term prognosis of these children is burdened by the need of repeated transfusions to combat the inhibition of erythropoiesis. Our report highlights the importance of early diagnose and precise antibody monitoring, to provide proper management and prophylaxis. Keywords: IMMUNE FETAL HYDROPS, RH ISOIMMUNISATION, DOPPLER ULTRASOUND.

Fetal hydrops is defined as an abnormal fluid collection in two or more areas of the fetal body, such as ascites, pleural effusion, pericardial effusion and skin edema (1, 2). The etiologies of fetal hydrodrops are classified as immune or non-immune hydrodrops. Immune hydrodrops develops due to fetal hemolysis mediated by circulating maternal antibodies to fetal red blood cell antigens. Recent advances in obstetric and neonatal medicine achieved significant improvements in diagnosis, prevention and management of fetal hydrodrops. Specifically, immune hydrodrops has been decreased by routine screening and prophylaxis of Rhesus iso-immunization (1,3). Non-immune hydrodrops can result from many causes including cardiac abnormalities (structural anomalies, cardiomyopathies and arrhythmias) and non-cardiac anomaly, aneuploidy, congenital infection, twin-to-twin transfusion syndrome, chorioangioma and other conditions. Different types of infections, such as toxoplasmosis, cytomegalovirus, herpes simplex virus, syphilis, and Parvovirus B19 are the most common congenital infections that cause fetal hydrodrops. But, the precise cause of non-immune hy-
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drops remains unknown in 15% to 25% of cases (3, 4), with an unchanged incidence (6) and a high mortality rate (4, 6, 8).

Fetal hydrops is easily recognized by ultrasound examination, during which we should try to figure out the etiology and nature of the hydrops, including the location, number and amount of fluid collections, amniotic fluid index, placental thickness, fetal echocardiography and Doppler velocimetry. However, there is limited information regarding the association between the ultrasound findings and perinatal outcomes of fetal hydrops, specifically neonatal mortality (10, 12, 13). Therefore, the aim of this case report is to show the clinical characteristics of fetal hydrops and to find out whether the antenatal ultrasound findings, especially the location or number of fluid collection sites, are associated with adverse perinatal outcomes.

CASE REPORT

We report the case of a pregnant woman 35 years old from the rural environment that presents to the hospital in emergency for the lack of perceiving of the active fetal movements.

The anamnesis highlights the fact that she didn’t followed the prenatal follow-up, she was Rhesus negative (her partner was Rhesus positive) and she had a titer of serum anti D antibodies of 1/32.

Her obstetric history consists of 2 normal birth of two healthy children of 3000 and 3500g and 2 spontaneous abortion at gestational age of 2 months for which she didn’t followed the anti-D immune globulin prophylaxis.

At admission to the hospital her blood pressure was 160/100 mm Hg, and she complained of epigastric pain that irradiates to her right shoulder (tab. I).

<table>
<thead>
<tr>
<th>TABLE I</th>
<th>Patient biochemical profile</th>
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<tbody>
<tr>
<td>Hb= 9.9g/dl</td>
<td>Glucose = 70mg/dl</td>
</tr>
<tr>
<td>Ht = 29.6%</td>
<td>FIBRINOGEN: 528mg/dl</td>
</tr>
<tr>
<td>PLT = 200000/ mm³</td>
<td>ESR = 12 mm/h</td>
</tr>
<tr>
<td>WBC = 12100/mm³</td>
<td>Creatinine = 0.85mg/dl</td>
</tr>
<tr>
<td>INR = 0.97</td>
<td>Urea = 19mg/dl</td>
</tr>
<tr>
<td>Ind Q = 102%</td>
<td>Uric acid = 7.67mg/dl</td>
</tr>
<tr>
<td>TIMP Q = 12.7/s</td>
<td>TGP = 64 U/L</td>
</tr>
<tr>
<td>MARTOR = 13/s</td>
<td>TGO = 106 U/L</td>
</tr>
<tr>
<td>APTT = 40.6/S</td>
<td>Group Rh: All negative.</td>
</tr>
<tr>
<td>PDF = 1600-3200 ng/ml</td>
<td></td>
</tr>
<tr>
<td>LDH = 658 U/L</td>
<td></td>
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</tbody>
</table>

**Direct urinary exam**: Albumin 100 mg / dl; ketones: traces; bilirubin ++; nitrates present; microbial flora present; leukocyte cylinders present.

Ultrasound reveals the diagnosis of hydrops fetal: a single living fetus of 29 weeks three days with an abdominal circumference (AC) of 39 weeks 3 days; Oligoamnios; Placental thickness 100mm gr I (fig. 1a); Scalp edema 17mm (fig. 1b); IR ACM-0.70; IR AO-0.61; ICP> 1.14; severe fetal ascites (fig. 1c); bilateral pleural effusions (fig. 1d); cardiomegaly.

At approximate 24 h after the admission, we decided to perform caesarean section for the diagnosis: VG IIIP 28 weeks of pregnancy, single living fetus, cranial presentation, intact membranes, Imminent premature birth,
Rh isoimmunization, Immune fetal hydrops, Preeclampsia, Acute Fetal Suffering.

We extracted a newborn who didn’t respond to resuscitation (Apgar score = 0), female, weight 2,040g (fig. 2). The placenta weighed ~ 1,700g.

Fig. 1. Ultrasound at admission
(a. Placental thickness, b. Scalp edema, c. Fetal ascites, d. Pleural effusion)

Fig. 2. Severe fetal ascites – clinical aspects: a. Edema and distention of abdomen due to ascites; b. coarse facial features, hypertelorism, depressed nasal bridge, anteverted nostrils, long philtrum, brachycephaly.

DISCUSSION
The diagnosis and management of fetal hydrops have improved in recent years with advances in prenatal diagnostic and thera-
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peutic interventions, together with the advances in neonatal intensive care. However, fetal hydrops is still associated with a high mortality rate.

In recent years, most newborn infants with this pathology were classified as non-immune fetal hydrops as the proportion of infants with immune fetal hydrops decreased to 10% due to the use of anti-D immunoglobulin prophylaxis, intrauterine transfusions, and close follow-up of the pregnancies with Rh-isoimmunization in developed countries. The etiology of fetal hydrops is known to be significantly associated with prognosis (4, 10).

With the recent advances in prenatal ultrasound and the increase acceptability of the method, fetal hydrops is almost always diagnosed by antenatal ultrasound. Therefore, we presented a case of immune fetal hydrops due to the Rh incompatibility, which was diagnosed incidentally by an antenatal ultrasound, presenting fetal ascites, pleural effusion, scalp edema and placental thickness, with a poor perinatal outcome.

According to various literature sources, the presence of pleural effusion was a poor prognostic factor of fetal hydrops (11). The most important findings in different studies was that the number of fluid collection sites was highly correlated with neonatal outcome including low Apgar score and neonatal death, as it was in our case. A similar result was found in a recent study by Kim et al. who studied 43 women with non-immune fetal hydrops. They developed an ultrasonographic severity scoring of non-immune hydrops (USNIH) defined as a total number of abnormal fluid collections. Perinatal mortality rate, defined as stillbirth or neonatal death ≤28 completed days after birth, was significantly higher in cases with USNIH of ≥3 than in those with USNIH of 2 (13,14).

Usually, in early stages, the doppler velocimetry is one of the most important predictor factors for fetal or neonatal mortality in various fetal compromise conditions, including fetal hydrops (11, 12, 13). The pattern of abnormal Doppler velocimetry indices may depend on different cause of fetal hydrops. For example, abnormal umbilical artery Doppler or MCA pulsatility index reflects fetal hypoxia, abnormal MCA peak systolic velocity may reflects fetal anemia, and abnormal ductus venosus Doppler occurs as a result of fetal cardiac decompensation (15). In our case, Doppler velocimetry wasn’t required, considering the advanced form of the disease, with multiple effusion sites.

CONCLUSIONS

These results from different studies, taken together with our clinical case, may suggest that the number of fluid collection sites is the strongest antenatal ultrasound risk factor of prediction the poor outcome in fetal hydrops, as we illustrated in this presentation.

This case underlines the importance of enforcing anti-D immune globulin prophylaxis when necessary, mainly to avoid the consequences of isoimmunization in Rh-sus negative mothers that can result in unfavorable outcomes for both the mother and the fetus.

REFERENCES


**TREATMENT ALGORITHM FOR THE MANAGEMENT OF COMPLICATIONS CAUSED BY PERMANENT FILLERS IN THE FACE**

Despite recent developments in the usage of resorbable substances such as hyaluronic acid, non-resorbable substances are still used to enhance soft-tissue volumes. Inflammatory reactions are frequent and disabling. Therapeutic options are nonspecific. A study was conducted as a review of 219 consecutive patients referred between 2006 and 2013. Screening was made with an ultrasound soft-tissue examination and the lesions were classified as either cystic or infiltrating, infiltrating patterns were treated intralesionally with 808-nm diode laser laser treatment alone, cystic lesions were also drained through stabwound incisions. In 62 percent the lesions disappeared completely. Partial improvement was obtained in 30 percent, whereas 8 percent discontinued the treatment. A systematic approach is proposed, based on 7 years of experience, with an overall improvement rate of 92 percent (Cassuto D, Pignatti M, Pacchioni L, Boscaini G, Spaggiari A, De Santis G. Management of Complications Caused by Permanent Fillers in the Face: A Treatment Algorithm. Plastic and Reconstructive Surgery 2016; 138(2): 215-227).