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Oligohydramnios with IUGR as a sonographic presentation of postnatally diagnosed hydrops

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Abstract:

Non-Immune hydrops is a rare entity, often fatal illness caused by innumerable causes. Owing to its significant morbidity and mortality, early diagnosis is warranted. We report a case of newborn diagnosed as NIHF postnatally with antenatal oligohydramnios on routine antenatal scans. A preterm baby born at 36+1 weeks of gestation to a pregnancy induced hypertensive mother via LSCS, cried immediately after birth, developed respiratory distress, hypoglycemia, subcutaneous edema and shock at birth. The baby was started on inotropic support, mechanical ventilation, glucose infusion rate and other supportive treatment. Chest Xray revealed cardiomegaly, ultrasound abdomen was suggestive of subacute intestinal obstruction. Other investigations were normal. The baby was diagnosed as NIHF postnatally, who succumbed to death despite best efforts. Oligohydramnios as an antenatal presentation of NIHF is a rare finding, warranting detailed evaluation for early diagnosis of NIHF for better outcomes.

Introduction:

Hydrops Fetalis is defined as the accumulation of extracellular fluid in 2 or more fetal compartments. The fluid collections include skin edema (>5 mm thickness), pleural effusion, pericardial effusion and ascites.[1] Hydrops fetalis are classified into two groups: Immune and Non immune hydrops. Since the advent of prenatal diagnosis and introduction of anti- D in

antenatal period, the incidence of Rh related hydrops has reduced to minimum and the maximum cases (90%) nowadays fall under non immune category. The incidence of NIHF is around 1 per 1700 to 3000 pregnancies [2,3]. There are innumerable causes for non-immune hydrops fetalis, of which a few are cardiovascular (21.4%), idiopathic (18.2%), chromosomal (12.5%), hematological (10%), lymphatic dysplasia (7.5%), infections eg. parvovirus, adenovirus, CMV, enterovirus (6.8%), Thoracic malformations (5%), twin to twin transfusions (5%), syndromic (4.6%), others (8%). Antenatal hydrops is not only a diagnostic challenge comprising difficulty in ascertaining the diagnostic criteria, technological limitations and operator variability but also a complicated entity to manage. A lot of cases are missed antenatally on routine ultrasound scans. NIHF has a very poor prognosis with 50-90% perinatal mortality, hence early diagnosis and management is warranted [4]. Here we present a case of non-immune hydrops with antenatal oligohydramnios and Intrauterine growth restriction (IUGR) with neonatal hypoglycemia.

Case report:

A male baby born to 28 years old primigravida out of spontaneous conception. The mother was diagnosed with gestational hypertension at 6 months of amenorrhoea and was started on restricted salt diet and aspirin. There was mild proteinuria and edema during last trimester. Quickening was felt at 5.5 months. Antenatal scans done at 5 months and 7 months were suggestive of oligohydramnios with IUGR. Level 2 scan was not suggestive of any fetal anomalies. Detailed fetal echocardiography was not done. Last antenatal ultrasound scan done just before delivery was suggestive of Amniotic fluid index (AFI) of 2 and intrauterine growth restriction (IUGR). Mother blood group was B+. There was no history of gestational diabetes, hypothyroidism, intrahepatic cholestasis of pregnancy (IHCP) during pregnancy. There was history of prolonged leaking per vaginum or fever just prior to delivery. There was no history suggestive of TORCH infections, exposure to radiations, UTI or bleeding per vaginum. The viral markers done in antenatal screening; HIV, HbsAg, Anti HCV, VDRL were negative.

The baby was born at 36 weeks +1day gestation, via LSCS in view of oligohydramnios and suspected preeclampsia. Just before the delivery the BP of mother was 160/110 mmHg with proteinuria and hence was treated with labetalol. There was no hepatitis or thrombocytopenia. The baby cried immediately after the birth, with APGAR of 7,9,9 at 1 min, 5 min and 10 min respectively. The birth weight was 1.6 kg, developed respiratory distress within 30 minutes of birth and was referred to our center. At presentation, baby had respiratory rate of 72 breaths/ min with subcostal retractions, Spo2- 67% under room air, pulses were low volume and capillary refill time (CRT) was 3 seconds. Random blood sugar was 36gm/dl and temperature of 36.8^0F. The baby was started on CPAP (Continuous positive airway pressure) support, injection dopamine, Glucose infusion rate (GIR) at 6mg/kg/min and intravenous antibiotics (cefotaxime and amikacin). On examination the baby had generalized edema (anasarca), absent bowel sounds and occasional crepitations in chest. Sensorium was normal, anterior fontanelle (AF) was at level, activity was fair and neonatal reflexes were present. In view of CPAP failure, child was intubated and started

on mechanical ventilation. On investigations, Chest Xray was suggestive of cardiomegaly and subcutaneous thickening with increased bowel gases and absent rectal gas shadow (Fig 1). Blood gas was suggestive of metabolic acidosis (pH-7.32, bicarb- 13.6 mmol/L, base excess of -12.5 mmol/L, Pco2-26.4 mmHg), sepsis screen was negative (TLC- 16700cells/ mm^3, 63% Polymorphs, 22% lymphocytes, 3% monocytes, 2% eosinophils, CRP- 0.5 mg/L). The baby blood group was B+ and Direct Coombs Test (DCT) was negative, there was no evidence of hemolysis on peripheral smear. The child was kept as a case of nonimmune hydrops with hypoglycemia with respiratory distress with shock. The baby continued to have hypoglycemic episodes along with seizures, for which GIR was titrated and anti-epileptic drugs were started. Metabolic screening and genetic studies could not be done in view financial constraints. On ultrasound cranium, baby had mild dilatation of ventricles, but had no intraventricular hemorrhage. Minimal enteral feeds were started on day 2 of life, but could not be tolerated. The baby passed meconium after 50 hours of life when given rectal stimulation. On Ultrasound abdomen, he had subacute intestinal obstruction with poor peristalsis, kidneys were normal in shape and size, there was no malformation noted. On day 3 of life, shock worsened hence inotropes were hiked. Echocardiography done on day 3 revealed normal study. The baby developed pulmonary hemorrhage on day 4 of life with passage of black colored stools and worsening shock. Fresh frozen plasma and packed red blood cell transfusion was given and inotropes hiked. Intravenous immunoglobulin (IVIG) was given. The baby succumbed despite the best efforts.

Discussion:

Non immune hydrops is a rare phenomenon encompassing various etiological factors, either genetic or acquired in utero. It is usually diagnosed antenatally in 2^{nd} trimester by the presence of extracellular fluid in any of the following compartments; subcutaneous tissues, pleura, peritoneal cavity or pericardium. Along with fluid accumulations, polyhydramnios is a very common finding associated with hydrops. Oligohydramnios is rarely associated with hydrops fetalis, but can be seen in fetal anemia, chromosomal anomalies and twin to twin transfusion syndromes. In our case, the hydrops was missed in antenatal ultrasound scans and was diagnosed later postnatally which led to poor prognosis and finally death. Early detection of hydrops in antenatal period helps the clinician and the parents not only in better preparedness for postnatal outcomes but also in the treatment of certain etiologies like fetal anemia and TTS in antenatal period, highlighting the importance of a quality antenatal scan and diagnosis at an early gestation. Umamaheswari, G., et al compared fetal autopsy with antenatal ultrasound findings in the diagnosis of hydrops fetalis. The study revealed that approximately 25% cases had total disagreement from their USG findings, with autopsy revealing much more findings which may help in genetic counseling and prevention of hydrops in subsequent pregnancies.[5]

Rodriguez M et al also highlighted that autopsy in combination with ultrasound scans and placental examination is best combination for etiological diagnosis of stillborn infants.[6]

In our case, the child presented with hypoglycemia and metabolic acidosis reflecting the need for universal metabolic screening which could not be performed due to financial constraints. Other condition called Mirror syndrome, which constitutes a triad of fetal hydrops, placentomegaly and maternal edema in a background of preeclampsia could have been another possible differential for NIHF. But in our case, placentomegaly could not be ascertained as the child was born outside, though no such documentation was noted in birth details. Moreover, polyhydramnios is a prominent feature in Mirror syndrome as opposed to our case which had oligohydramnios. The prognosis of NIHF depends upon the etiology, gestation at diagnosis, delivery timing, extent of edema and intrauterine interventions [7]. With above case, we need to highlight the importance of timely detection of NIHF in early antenatal period, so as to improve antenatal, perinatal and postnatal outcomes. It is to be acknowledged that missing NIHF comes in the light of various limitations in antenatal care in a resource limited country like India. Also, detection of etiologies like metabolic syndrome, chromosomal anomalies and rare congenital infections comes with a challenge of financial and logistic constraints.

Conclusion:

Early detection of NIHF is extremely essential for better perinatal and postnatal outcomes. A rare case of NIHF can present as oligohydramnios in antenatal scan as was in our case. We propose detailed evaluation of every case of oligohydramnios and IUGR with emphasis on etiological diagnosis like chromosomal anomalies, metabolic conditions, fetal anemias and congenital infections.

Keywords: Non immune hydrops, Oligohydramnios, IUGR, Hypoglycemia

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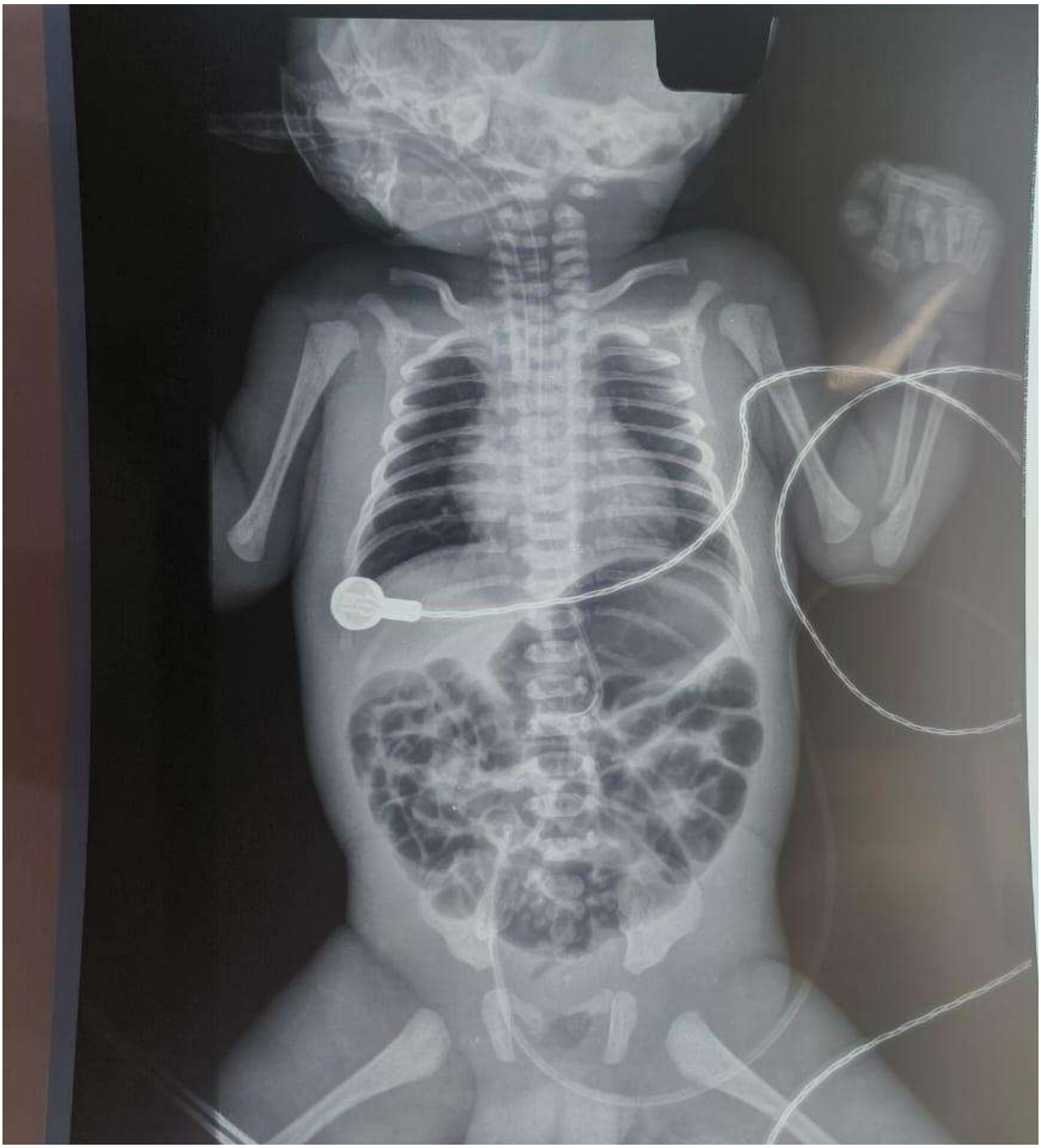

Figure 1: Shows subcutaneous edema with cardiomegaly and absent rectal gas shadow