ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

A Case Report on Hydrops Fetalis.

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ABSTRACT

Hydrops foetalis is defined as a state of excessive fluid accumulation in the extra vascular compartment of the foetus, leading to widespread soft tissue oedema and/or accumulation of fluid in the foetal body cavities. The prognosis of hydrops foetalis is highly dependent on the underlying pathology and early diagnosis is essential to identify treatable cases. The classification of immune and non-immune hydrops foetalis describes the difference between Rhesus haemolytic disease of the newborn and other aetiologies leading to hydrops foetalis. With improved diagnosis and treatment of Rhesus iso-immunisation, non-immune factors have become more frequent. Distinction between anaemic and non-anaemic hydrops foetalis provides a far more useful differentiation between aetiologies. This approach is used to discuss differential diagnosis, work-up and therapeutic options in hydrops foetalis. A structured multidisciplinary
work-up will facilitate early diagnosis and assist in making treatment decisions.
Keywords: Hydrops fetalis,isoimmunisation, anemia, ascites, intrauterine transfusion

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ISSN: 0975-8585

INTRODUCTION

Hydrops fetalis is most serious form of RH hemolytic disease. Excessive destruction of the fetal red cells lead to severe aneamia,tissue anoxmia & metabolic acidosis. These have got side effect on the fetal heart,brain& on the placenta. Hyperplasia of the placental tissue occurs in the effort to increase the transfer of oxygen .But the available fetal red cells are progressively diminished due to hemolysis. As a result fetal anoxemia. There is damage to the liver leading to hypoprotenemia which is responsible for genenaralised oedema-"HYDROPS FETALIS"(Ascitis & hydrothorax). Fetal death occurs sooner or later due to cardiac failure.

A CASE REPORT

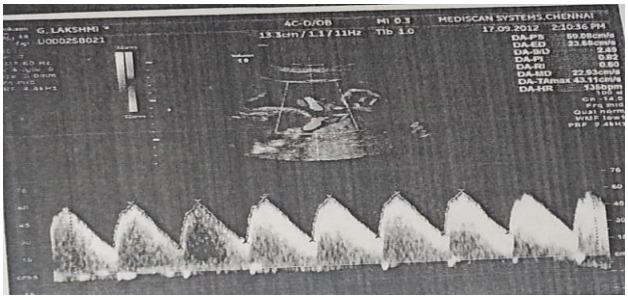
Mrs. Lakshimi ganesh 21yrs G7P6L0 RH negative mother with LMP- 1.01.2012 EDD- 8.10.2012 with GA- 32wks 2days as LMP. Usg was done fetus was normal with single umbilical artery with GA – 24WKS . Indirect coombs test negative. After 4 weeks usg showed SLIUG GA-28wks6days cedd-04.12.2012 with hydramnios, placentomegaly, generalized oedema, spleenomegaly, ascites, pericardial effusion,cardiomegaly,pulmonary hypoplasia with middle cerebral artery peak systolic velocity falls at 2.0 mom suggestive of severe fetal aneamia features of IMMUNE HYDROPS.

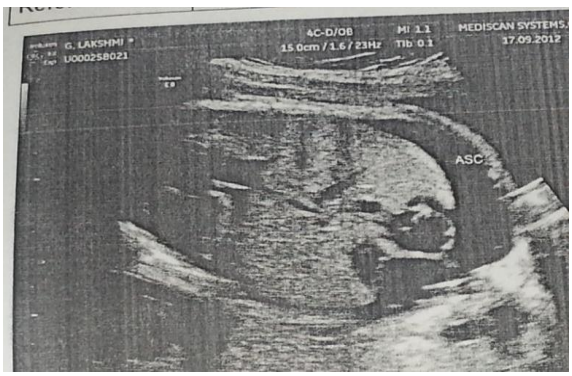
PRENATAL INVASIVE PROCEDURE – INTRAUTERINE TRANSFUSION was done with MCA peak systolic velocity 80ccms/s falls at 2.0 moms for 29-30wks. Fetus heamatocrit 9%,HB-3%. Donor heamatocrit-65.4%,HB-21%. 170ml "O "negative packed cell transfused through portal vein to fetus. POST TRANSFUSION STATUS-FETUS-Heamatocrit-45%, Hb-15.4%,FHR-139Bpm, post IUT MCA peak systolic velocity 13.9cms /sec fall at 0.35moms for 29-30 wks gestation. Usg done after showed no evidence of fetal anemia with MCA peak systolic velocity fall at 1.17moms with GA-31WKS6DAYS with polyhydramnios. Usg after one month showed fetal anemia with MCA peak systolic velocity falls at 1.09moms with GA- 35WKS4DAYS.

Emergency Lscs done on 7.11.12- alive,preterm,male 2.4kg with apgar 6/10,7/10 no obvious anamoly at 12.35pm. placentomegaly was present . Baby was transferred to Nicu baby hb -6gms, billirubin was increased . o positive was transfused and treated with immunoglobulin to stop heamolysis . Anaemia was corrected and hemolysis stopped. Baby had distress which was corrected and discharged after a month.

Images of Hydrops Fetalis:

Features of hydrops fetalis:





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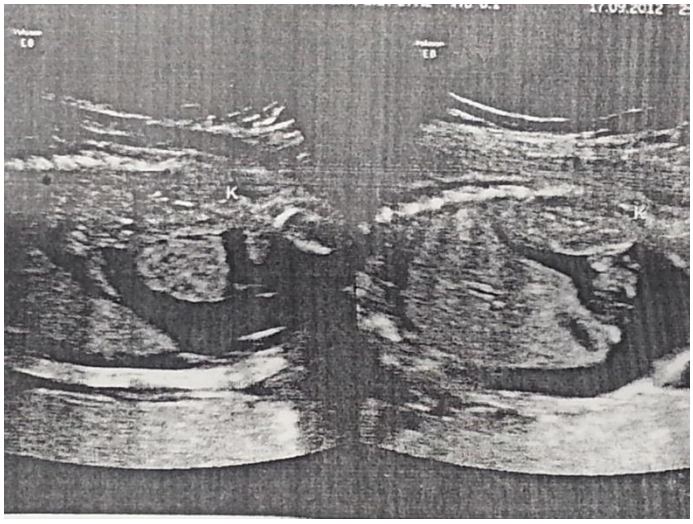
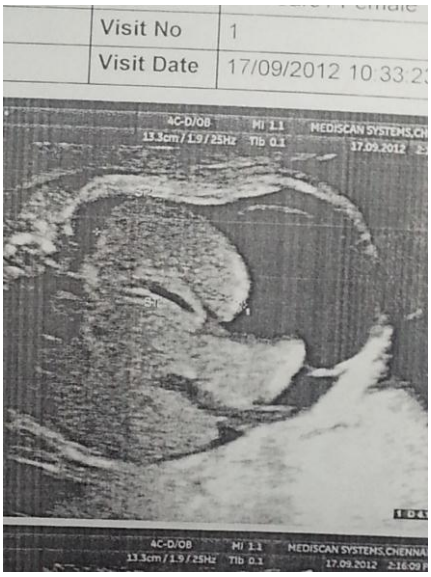
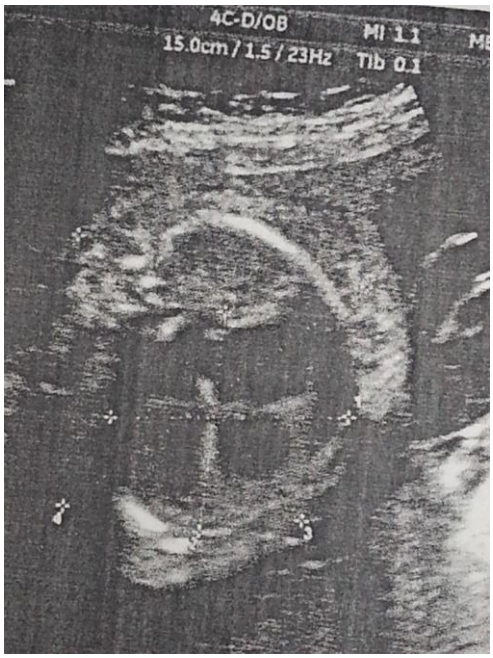
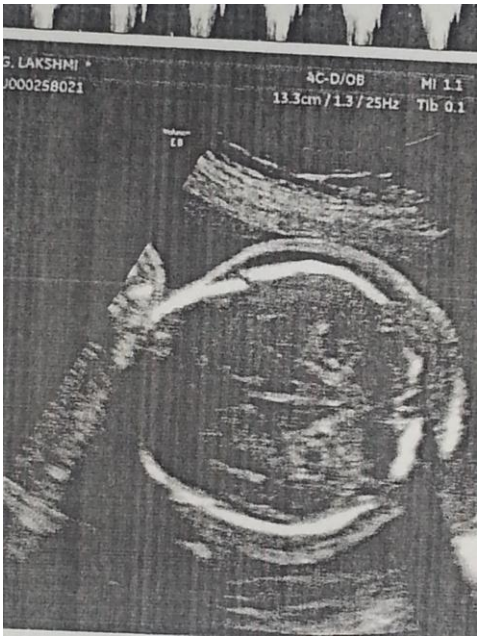


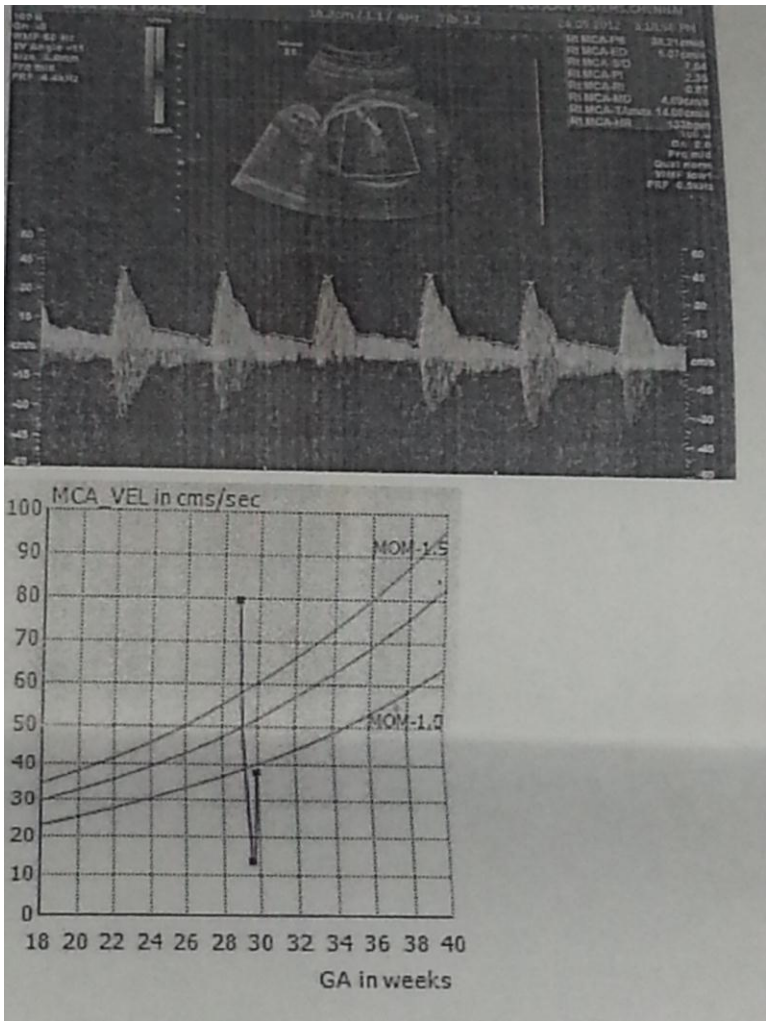




ISSN: 0975-8585

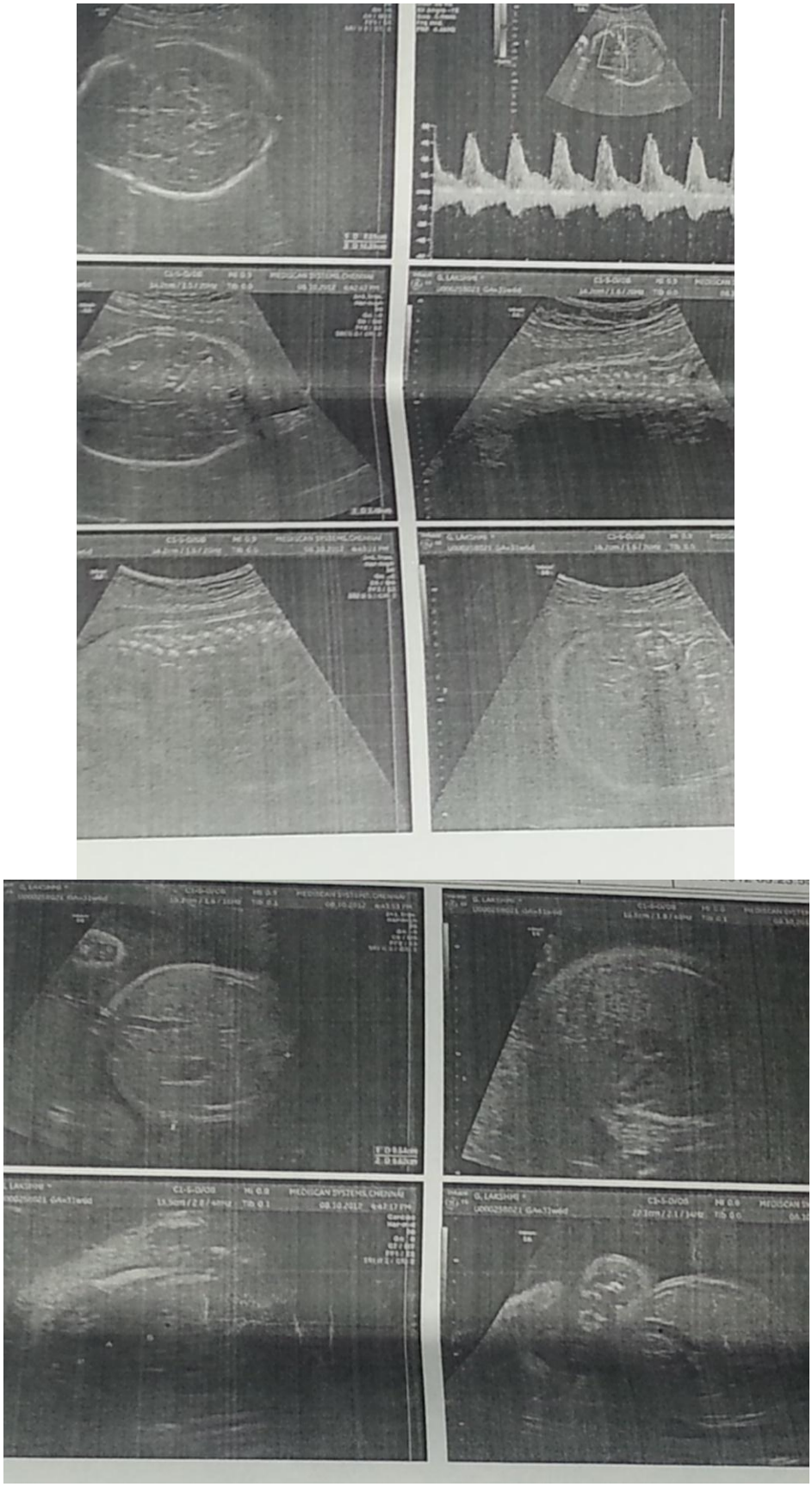


Scalp Edema



During Intrauterine Transfusion

ISSN: 0975-8585



Features Rsolved After Transfusion

ISSN: 0975-8585

DISCUSSION

In 1892, Ballantyne established clinicopathological criteria for the diagnosis of hydrops fetalis .Diamond,Blackfan and Baty in 1932, reported fetal anemia characterized by numerous circulating erythroblast was associated with this syndrome [1]. Levine.et.al. in 1941 confirmed that erythroblastosis was due to maternal isoimmunisation with paternally inherited fetal factors [2]. Development of effective maternal prophylaxis was attributed to Finn and associates of England & Freda & coworkers of united states [3, 4].

The abnormal collection of fluid in more than one area of the fetal body, such as ascites and pleural effusion is termed as hydrops fetalis. Pathological changes in the organs of fetus vary with severity. At times ,the edema is so severe that can be identified by USG as in my case. In this case placenta is edematous and enlarged. Excessive and prolonged hemolysis serves to stimulate marked erythroid hyperplasia of bone marrow as well as large areas of extramedullary hematopoiesis, particularly in the spleen and liver leadingto hepatospleenomegaly,which may in turn cause hepatic dysfunction [5]. Pathophysiology of hydrops remains obscure. Theories of its causation include heart failure from anemia, capillary leakage caused by hypoxia from severe anemia, portal, umbilical venous hypertension from hepatic parenchymal disruption by extramedullary hematopoiesis and decreased colloid osmotic pressure from hypoproteinemia caused by liver dysfunction. Nicolaides and colleague concluded that degree and duration of anemia influence the severity of ascites and 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 .The live born infant appear pale, edematous at birth .Dyspnea and circulatory collapse are common.Diagnosed in utero by middle cerebral artery peak systolic velocity . A threshold of>1.5 MoM correctly identified all fetus with moderate or severe anemia [7]. Intrauterine transfusion can be done for immune hydrops fetus .Nicolides and co workers recommends transfusion when hb% is atleast 2gm/dl below the mean for normal fetus of corresponding age, hematocrit below 30 percent -2sdn below mean at all gestational age(weiner and associates,1991b). A single intramuscular dose of 300mcg of anti-D administered routinely to all D-negative ,non immunized women at 28 to 32 weeks and again within 72 hrs of the birth of a D-positive infant.

REFERENCE

[1] Diamond LK, Blackfan KP, Baty 1M. J Pediatr 1932: 1 : 269.
[2] Levine P. In Karsner HT, Hooker SB (eds) : Year book of Pathology and Immunology. Chicago,Year book Publishers, 1941, p 505.
[3] Finn R, Clarke CA, Donohoe W, McConnell RB, Sheppard PM, Lehane D, Kulke W. Br Med J 1961; I: 1486.
[4] Freda VJ, Gorman JG, Pollack W. Fed Proc 1963 ; 22 : 374.
[5] Nicolini U, Nicolaides P, Tannirandorn Y, Fisk N, Nasrat H, Rodeck CH.Br J Obstet Gynaecol 1991 ; 98 : 287.
[6] Nicolaides KH, Warenski .fC, Rodeck CH. Am J Obstet Gynecol 1985 ; 152 : 341.Vol. I No.
[7] Oepkes and coworker(2006) compared doppler velocimetry to amniotic fluid bilirubinstudies
They reported tat Doppler study showed 88 percent sensitivity and 85% accuracy where as bilirubin studies had 76%sensitivity,77%specificity&76%accracy.