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NON IMMUNE HYDROPS FETALIS

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Abstract:

INTRODUCTION:

Hydrops Fetalis is an accumulation of extracellular fluid in fetal body civilities like pleural, pericardial, Scalp and in body wall [1]. First case of hydrops fetalis was identified by Ballantyne. Edit Potter differentiated between immune hydrops fetalis from nonimmune hydrops fetalis [2]. NIHF is associated with accumulation of extracellular fluid in fetal body Cavities like pleural effusion, pericardial, scalp, body wall with increased skin thickness of more than 5mm. Due to generalized subcutaneous edema and placental enlargement. When first described HF constituted 20% of all cases, but with effective anti D prophylaxis for immune Hydrops, NIHF constitutes 90% cases of fetal Hydrops [3]. NIHF incidence reported as 1 in 3000 pregnancies [4]. Approximately 50% of fetuses with non-immune hydrops fetalis die in utero, and about half of the live born infants survive. Routine Ultrasound has been recommended as the initial diagnosis of NIHF. Placental thickness should be measured in all cases to rule out placental edema. 3D scan helps in determination of Facial dysmorphology which May give clue to inborn errors of metabolism and Cystic hygroma may be in cases of Aneuploidy, fetal Echocardiography, examination of maternal blood for fetal erythrocytes, amniocentesis and sampling of Fetal blood. We report two of NIHF fetuses about 16-18 weeks of gestational age.

OBJECTIVE:

Goal of our study is to find out etiology and outcome of pregnancy with non-immune hydrops fetalis.

RESULTS & CONCLUSION:

A detailed Ultrasound with AFI and Doppler study is most useful in diagnosis of hydrops fetalis and underlying condition responsible for that. Test for fetal infection and antibody in maternal serum is helpful in identifying infective etiology. Amniotic fluid study is costly but most sensitive for karyotyping to identify chromosomal abnormalities. Once condition is identified than a patient referred to higher center for better availability of neonatal care. Outcome of babies is poor in a case who survive till term pregnancy.

KEY WORDS:

Hydrops fetalis, Immune hydrops fetalis, Non immune hydrops fetalis

DEFINATION

Hydrops fetalis (HF) is defined as abnormal fluid accumulation in at least two fetal compartments, which may be pericardial effusion, pleural effusions, ascites and skin edema. This accumulated excess fluid can affect fetal heart and other organ lead to poor fetal outcome.

INTRODUCTION

Hydrops Fetalis is an accumulation of extracellular fluid in fetal body cavities like pleural cavity, pericardial cavity, scalp and in body wall [1].

First case of hydrops fetalis was identified by Ballantyne. Edit Potter differentiated between immune hydrops fetalis from nonimmune hydrops fetalis [2].
NIHF is also associated with increased skin thickness of more than 5mm due to generalized subcutaneous edema and placental enlargement.
When first described HF constituted 20% of all cases, but due to effective anti D prophylaxis for immune Hydrops, NIHF constitutes 90% cases of fetal Hydrops [3].
NIHF incidence reported as 1 in 3000 pregnancies [4]. About 50% of fetus dies in utero and 50% of fetus will survive.

CLASSIFICATION
Hydrops fetalis are classified into 2.

Immune Hydrops fetalis	Non immune Hydrops fetalis
Isoimmunization dure to ABO and RH incompatibility Other antibodies like c,C,e,E,Kell,duffy	1.Chromosomal anomalies: Turner syndrome(45+X0), Down syndrome(Trisomy 21), and Edward syndrome(Trisomy 18)
	2.Infections: TORCHES Parvovirus B19 (fifth disease), cytomegalovirus, and syphilis
	3.Lymphatic causes: Congenital lymphatic dysplasia
	4.Cardiac causes: Paroxysmal supraventricular tachycardia, Hypoplastic left heart syndrome
	5.Maternal Diabetes mellitus and hyperthyroidism
	6.Hematologic causes: Alpha-thalassemia, twin pregnancies, and leukemias
	7.Metabolic cause: Lysosomal storage disorder Niemann-Pick disease type-C (NPC), Gaucher disease type 2, and beta-glucuronidase enzyme deficiency
	8.Disorders of red blood cell (RBC) metabolism: Glucose phosphate isomerase deficiency, pyruvate kinase deficiency, and glucose-6-phosphate dehydrogenase (G6PD) deficiency
	9.Tumor: Sacrococcygeal teratoma

(Williams edition 25, pg no.309)
EPIDEMIOLOGY:

The prevalence of non-immune Hydrops fetalis may vary depending upon the criteria. Prevalence may range from 1:1500 to 1:4000 births. Due to availability of Anti D immunoglobulin prevalence of immune Hydrops fetalis decrease up to 10% of total cases and now non immune Hydrops fetalis is responsible for 90 percent of total cases.[6]

PATHOPHYSIOLOGY:

There is an abnormal fluid movement between plasma(extracellular)and tissues(intracellular)and the imbalance between the interstitial production and lymphatic return [7]

Four theories have been postulated:

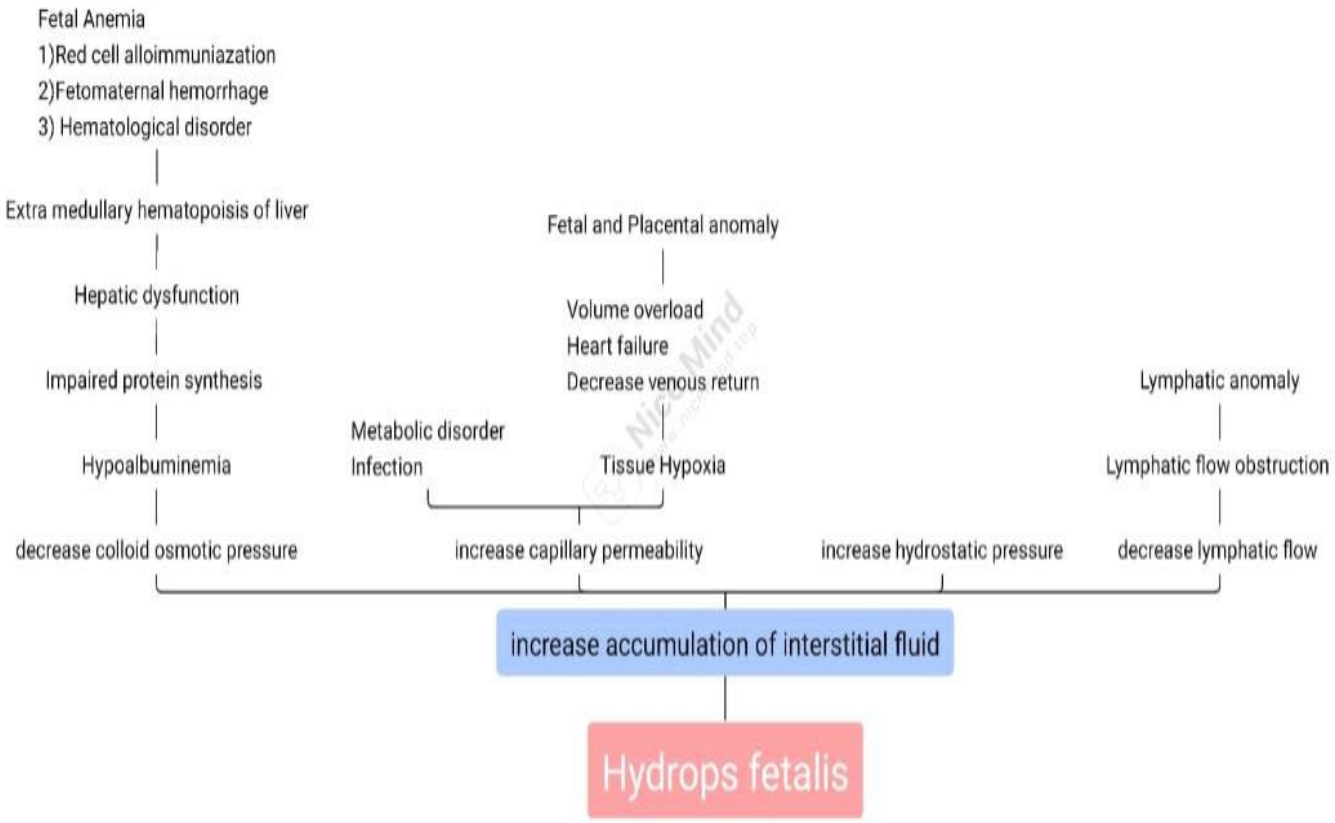
1.Reduced plasma oncotic pressure
2.Increase in hydrostatic capillary pressure
3.Lymphatic flow obstruction
4.Increase capillary permeability

(Flow chart-prepared by self on the basis of Williams edition 25,page no.309)

INVESTIGATION:

Maternal blood group- to rule out immune hydrops fetalis.

Hemoglobin electrophoresis- to rule out thalassemia, both alpha and beta.



TORCHS+P serology (toxoplasma, rubella, cytomegalovirus, herpes, syphilis, and parvovirus).

Anti-Ro and La antibodies

Maternal serum electrolytes and liver function tests, urinary PCR-to rule out maternal mirror syndrome.

Serum beta HCG and Thyroid function test- to rule out triploidy and partial molar pregnancy.

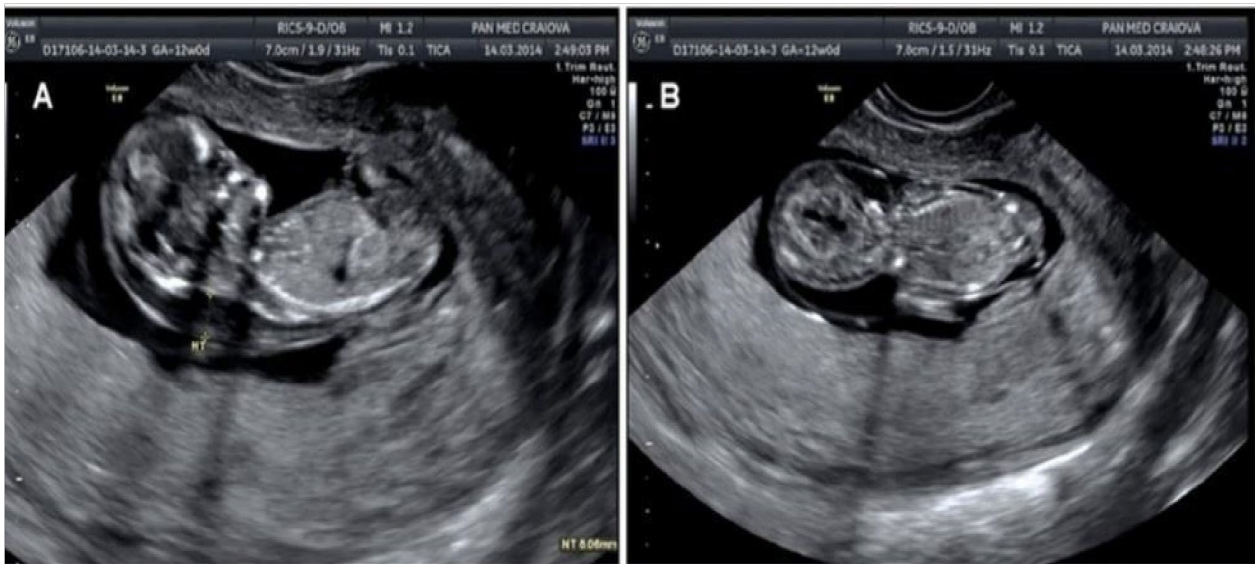
Fetal heart rate- to rule out Tachy and Brady arrhythmias.

Fetal dopplers including umbilical artery, vein, ductus venosus, tricuspid regurgitation, and middle cerebral artery peak systolic velocity (MCA PSV)

MCA Doppler peak systolic velocity- more than 1.5 MOM seen in fetal anemia in which due to fetal anemia produce hypoxia which lead to increase velocity blood flow in fetal brain.
USG guided amniocentesis and amniodrainage- for karyotyping to rule out chromosomal abnormalities.
The amniotic fluid is sent for chromosome tests—fluorescent in situ hybridization (FISH), chromosomal microarray, rasopathy, or hydrops panel including Noonan's syndrome, lysosomal storage disease and in some centers whole exome sequencing.
Amniotic fluid sent for- Polymerase chain reaction (PCR) for fetal infection such as TORCHS+parvovirus' DNA for specific conditions if known.
In pleural effusion- pleural tapping done and sample sent for infection and karyotyping.[9]
Fetal MRI- to see extension of Sacrococcygeal teratoma.

CASE REPORT:

CASE 1: A fetus of 16week with cystic hygroma + hydrops
OBSTETRICS HISTORY: A 24year old G2P1A0L1 female patient came to shardaben general hospital for routine antenatal visit. First pregnancy was full term, normal vaginal delivery,2year old live female. The present case was second pregnancy, spontaneous conception with 16weeks of gestation. At presentation her vital were with in normal limit and her hematocrit and WBC count were normal
USG: A single live intrauterine fetus with 16 weeks' maturity shows
Large Multi loculated cyst with internal septa in neck 4×5×6cm is noted with minimal ascites, pleural effusion and skin edema present.
Liquor- adequate
placentomegaly
IMPRESSION: intrauterine fetus with gestation age of 16 week with pleural effusion and ascitis and placentomegaly. After admission amniocentesis was done and 45 xo was diagnosed. Consent for MTP was taken. she spontaneously delivered live fetus weight 170gm with edematous skin and hydrops changes. placenta and membrane expelled.

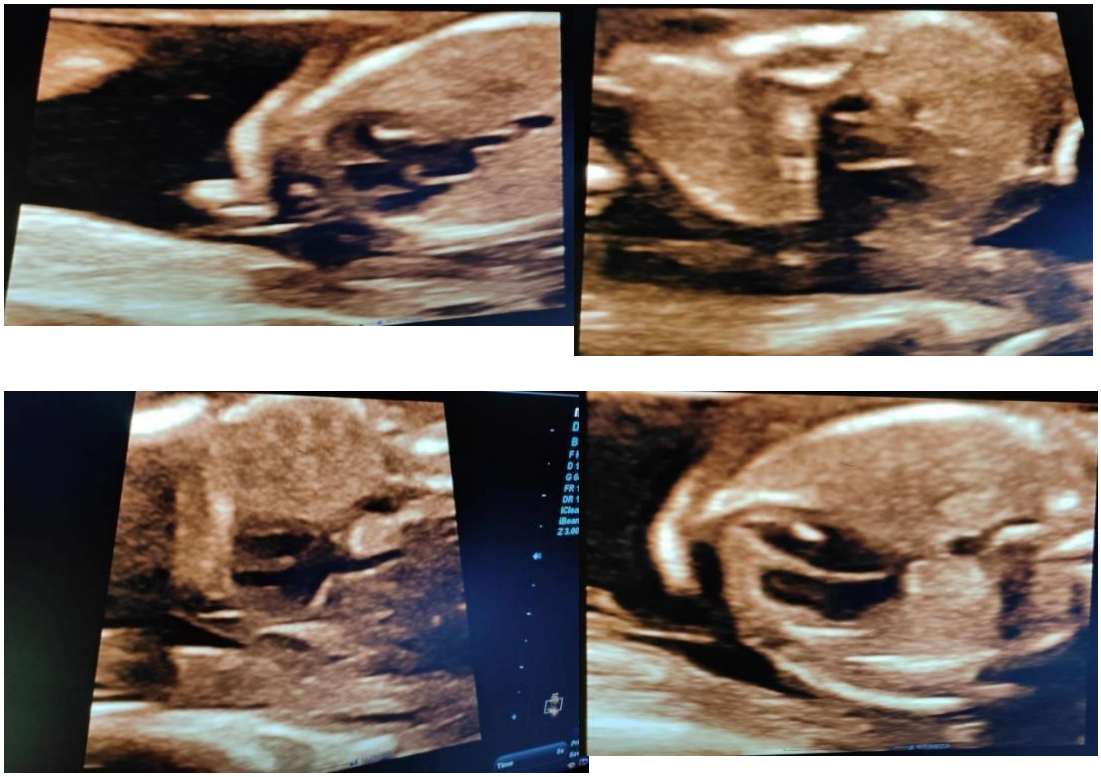


CASE 2 A fetus of 19 week 4 days with congenital heart disease and hydrops fetalis
OBSTETRICS HISTORY:
32-year-old G2P0A1L0 hindu female patient came to shardaben general hospital for routine antenatal visit. First pregnancy was spontaneous abortion at 2 month of amenorrhea 4 year ago followed by D and E. Present pregnancy was spontaneous conception with 19 week 4 days of gestation. On Examination patient is vitally stable.

USG:

Single live intrauterine fetus shows congenital heart disease (tetralogy of fellot)
Pericardial effusion, foci in left ventricle. skin edema present
Placenta fundoposterior
Polyhydramnios
IMPRESSION:
Single live intrauterine fetus with gestational age of 19 week 4 days with and congenital heart disease seen, with pericardial effusion and skin edema. After admission consent for MTP was taken. she underwent medical abortion, expelled fetus and placenta.

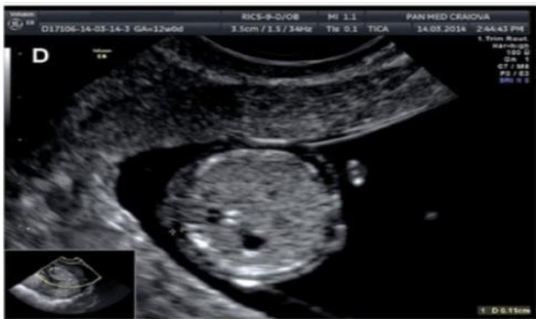
FIGURE:

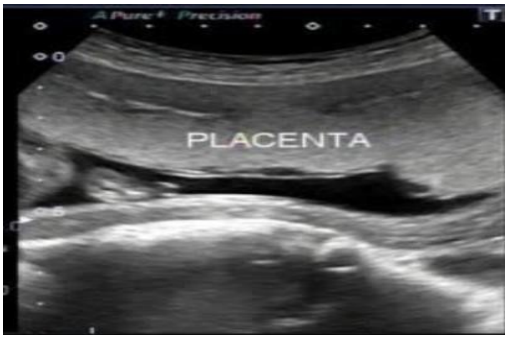


CASE 3:
A fetus of 12 week with maternal parvo B19 virus infection.
OBSTETRICS HISTORY:
A 26-year-old primi gravida patient came to shardaben general hospital with complain of fever and body ach.
On examination:
T – 100.4
P -102/min
Bp -110/72mmhg
All other fever investigation is normal.
Parvo B 19 IG M antibody: **positive**

USG:
Single live intrauterine fetus of 12 weeks 2 days' maturity.
Mild ascites present
Skin edema
Ventriculomegaly
placentomegaly.
IMPRESSION:
A single live intrauterine fetus with gestational age of 12 week 2 days. All causes of fever were ruled out and IG M antibody for parvo B19 virus was positive. patient managed conservatively and patient advice for continuing pregnancy and follow up USG for further evaluation.

FIGURE :





CONCLUSION:

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Conflict of Interest:

Nil

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