Rare case of fetal hydrothorax

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Received: 25th July, 2018 **Accepted:** 21st August, 2018

Abstract
Fetal hydrothorax were diagnosed during ultrasound examination in asymptomatic women in the second trimester of pregnanancy.[1] Fetal hydrothorax is a condition in which fluid accumulates in the fetal chest, in the space between the lungs and the chest wall (known as the pleural space). This fluid may also be referred to as "pleural effusion." Fetal hydrothorax usually develops when there are leaky or obstructed veins or lymphatic channels. occasionally, the fluid leakage may result from a mass (such as a lung mass), infection or chromosome problem, but in many instances no specific cause for the leaky fluid may be found.

Keywords: Preterm labour, Pulmonary hypoplasia, Congestive hert failure, Fetal hydrothorax, Amniocentesis, Thoracentesis, Down syndrome, Idiopathic neonatal pleural effusion, Nonchylous, Respiratory distress idiopathic neonatal pleural effusion, Nonchylous, Respiratory distress, Bronchopulmonary dysplasia, Induction of labour.

Introduction
Fetal pleural effusions are rare congenital anomalies with an estimated incidence of 1:10.000–15.000 pregnancies.[2] Fetal hydrothorax, either unilateral or bilateral is a pleural effusion that may be primary, due to chylous leak or secondary in which the effusions are part of generalized fluids retention associated with immune or non immune hydrops.[3]

The pleural space exists between the parietal pleura of the chest wall and the visceral pleura of the lung. Both pleural surfaces filter fluid into the pleural space and the lymphatics are responsible for most of the fluid reabsorption.[4] Pleural effusion defined as fluid accumulation in the pleural space can occur if the rate of filtration increases or if the rate of lymphatic clearance decreases or if both of these mechanisms are present.[5]

The etiology of pleural effusions can be classified in primary correctly termed hydrothorax antenatally and chylothorax postnatally. Secondary usually associated with immune or non-immune hydrops as infection, congenital lung lesion, bronchopulmonary sequestration or congenital diaphragmatic hernia, congenital heart disease or chromosomal - genetic syndromes.[6] Maternal serology to exclude congenital infections (toxoplasmosis, rubella, cytomegalovirus, parvovirus b19, syphilis and herpes), blood type and antibody screening to rule-out immune hydrops and kleihauer-betke test if there is a concern about maternal-fetal transfusion. Detailed ultrasound and echocardiographic evaluation should exclude major congenital abnormalities. The development of pulmonary hypoplasia or hydrops due to mediastinal shift and vena caval obstruction are the main feared complications. The overall mortality rate is about 22-55% and there is no established consensus for its management, given the high heterogeneity of the disease.[7]

Chylothorax is a rare cause of respiratory distress in the newborn and the most common form of Pleural effusion in the neonatal period.[8]

Case Report
We present a case of a bilateral fetal pleural effusion identified at 22-24 gestational week. A 28-year-old patient gravida 2 para 1 (1 male child normal vaginal delivery four years before) presented to our routine term pregnancy evaluation. Her active married life was six years. (Non consanguineous marriage). Personal and family histories were unremarkable. At ecographic evaluation we identified a moderate anechogenic fluid collection in the both fetal hemithorax, with atelectasis of both lung, displacing the heart and mediastinal structures to the bilateral hemithorax, hydramnios was also identified. A detailed ultrasound examination was performed and no other structural abnormalities were detected, as no signs of hydrops. Fetal biometry was compatible with gestational age. Fetal ecochardiogram detected right ventricular hypoplasia. Doppler evaluation of the peak systolic velocity in the middle cerebral artery was normal.

On Examination: Blood pressure -130/80 mmhg, Pulse rate -90/bpm, Respiratory rate -16/min, SpO_2-98%. Local examination per abdomen: FHS present.

USG findings:
1. Fetal heart rate -144BPM
2. Fetal movement normal
3. Cephalic presentati
4. Placenta posterior grade 1 maturity (No e/o placenta previa

5. Amniotoc fluid index-14.3
6. EFW-1311gm
7. Cervical length -35mm, cervical os closed

Comments: Singal live intrauterine with fetal maturity 23weeks with bilateral moderate pleural effusion. Doppler study: normal flow pattern in both uterine arteries and umbilical artery and middle cerebral artery, normal findings of all 4 cardiac chambers. Investigation. Hb–7.9gm%, WBC-10500cumm, platelets-352000cumm, bgrh–B-positive, HIV- nr, HBsAG- nr,RBS-111.5mg/dl, urea-24 mg/dl, serum creatinine-0.58mg/dl, serum uric acid-2.50mg/dl serum bilirubin: total-0.74, direct-0.42, indirect-0.32, SGPT-10U/L, Bleeding time-3min 0 sec. Clotting time-4min 0sec.prothrombin time-1.11.

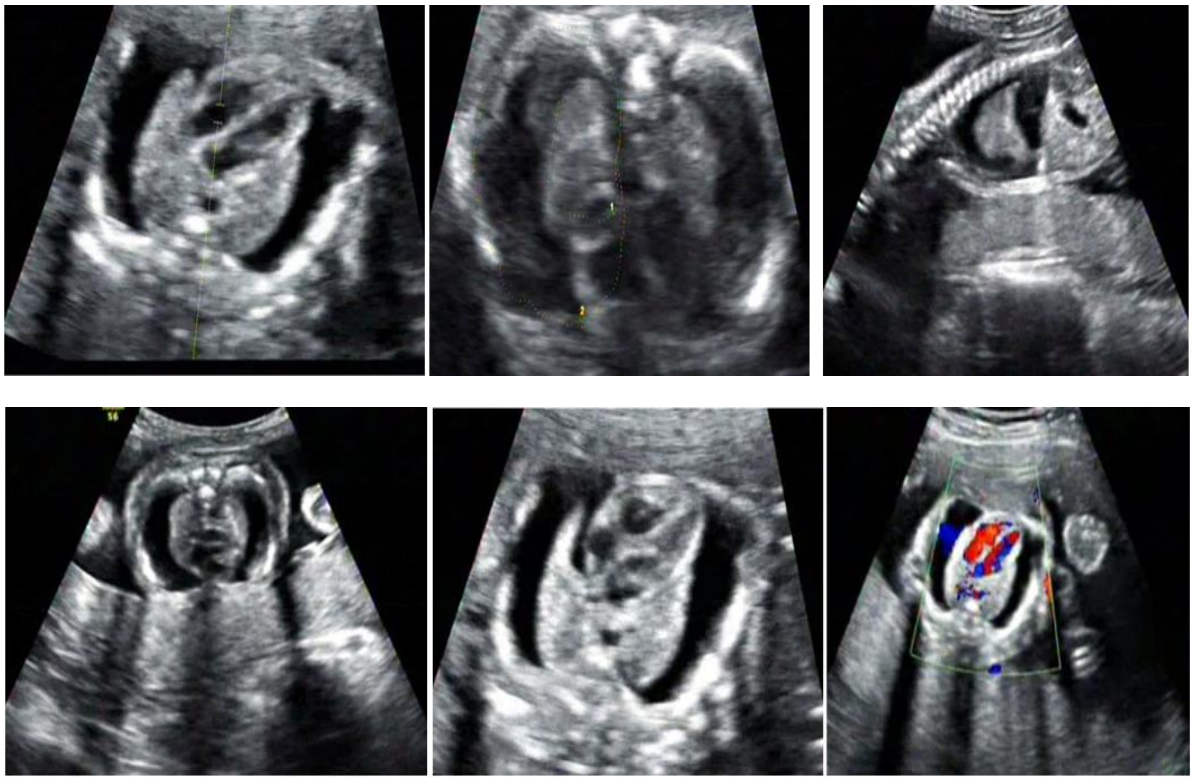


Fig. 1

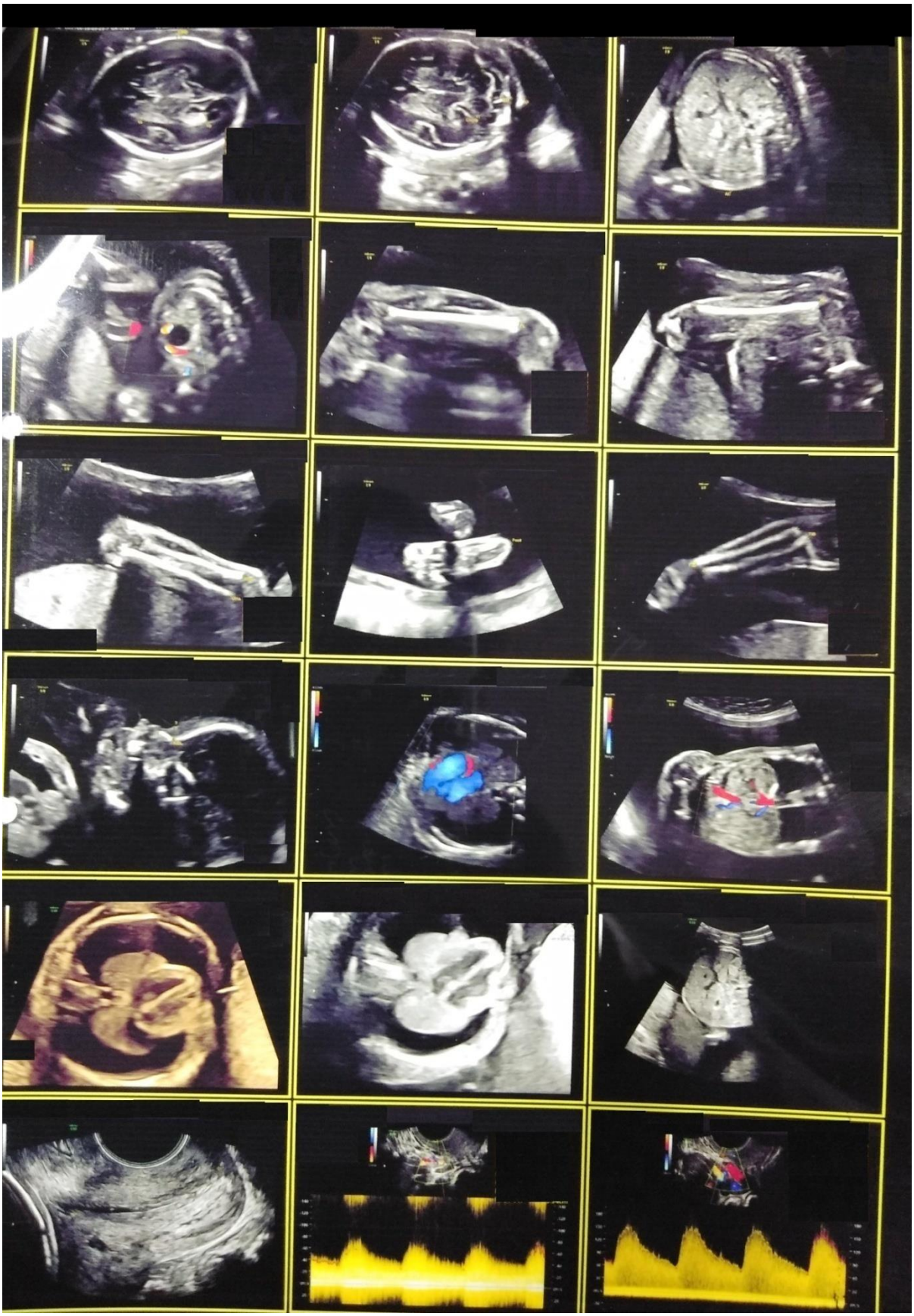
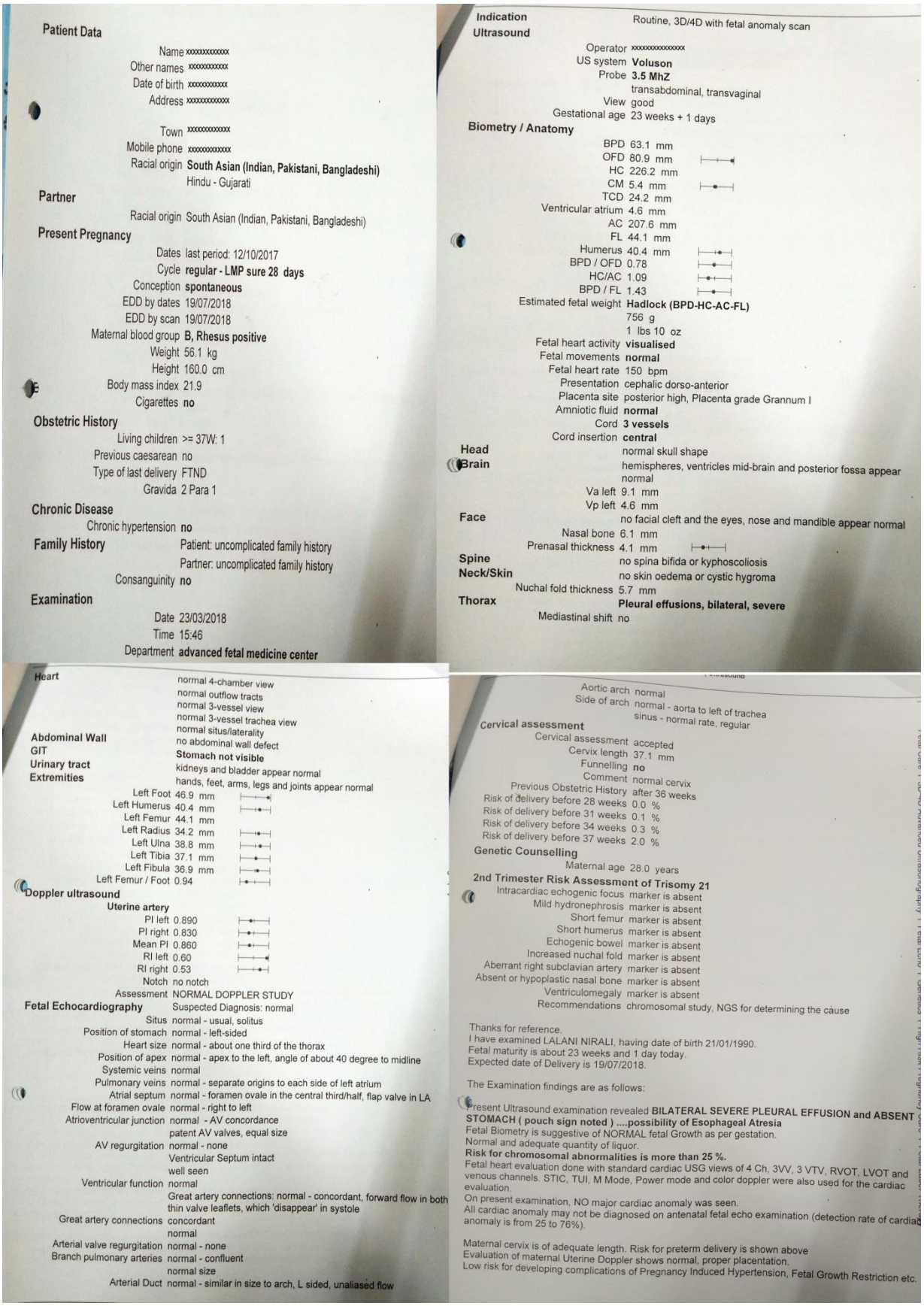


Fig. 2



Patient Data

Name xxxxxxxxxxxx
Other names xxxxxxxxxxx
Date of birth xxxxxxxxxxx
Address xxxxxxxxxxxx
Town xxxxxxxxxxxx
Mobile phone xxxxxxxxxxx
Racial origin **South Asian (Indian, Pakistani, Bangladeshi)**
Hindu - Gujarati

Partner

Racial origin South Asian (Indian, Pakistani, Bangladeshi)

Present Pregnancy

Dates last period: 12/10/2017
Cycle **regular - LMP sure 28 days**
Conception **spontaneous**
EDD by dates 19/07/2018
EDD by scan 19/07/2018
Maternal blood group **B, Rhesus positive**
Weight 56.1 kg
Height 160.0 cm
Body mass index 21.9
Cigarettes **no**

Obstetric History

Living children >= 37W: 1
Previous caesarean no
Type of last delivery FTND
Gravida 2 Para 1

Chronic Disease

Chronic hypertension **no**

Family History Patient: uncomplicated family history
Partner: uncomplicated family history

Consanguinity **no**

Examination

Date 23/03/2018
Time 15:46
Department **advanced fetal medicine center**

Indication Routine, 3D/4D with fetal anomaly scan

Ultrasound

Operator xxxxxxxxxxxxxx
US system **Voluson**
Probe **3.5 MhZ**
transabdominal, transvaginal
View good
Gestational age 23 weeks + 1 days

Biometry / Anatomy

BPD 63.1 mm
OFD 80.9 mm
HC 226.2 mm
CM 5.4 mm
TCD 24.2 mm
Ventricular atrium 4.6 mm
AC 207.6 mm
FL 44.1 mm
Humerus 40.4 mm
BPD / OFD 0.78
HC/AC 1.09
BPD / FL 1.43
Estimated fetal weight **Hadlock (BPD-HC-AC-FL)**
756 g
1 lbs 10 oz
Fetal heart activity **visualised**
Fetal movements **normal**
Fetal heart rate 150 bpm
Presentation cephalic dorso-anterior
Placenta site posterior high, Placenta grade Grannum I
Amniotic fluid **normal**
Cord **3 vessels**
Cord insertion **central**

Head normal skull shape
Brain hemispheres, ventricles mid-brain and posterior fossa appear normal
Va left 9.1 mm
Vp left 4.6 mm
Face no facial cleft and the eyes, nose and mandible appear normal
Nasal bone 6.1 mm
Prenasal thickness 4.1 mm
Spine no spina bifida or kyphoscoliosis
Neck/Skin no skin oedema or cystic hygroma
Nuchal fold thickness 5.7 mm
Thorax **Pleural effusions, bilateral, severe**
Mediastinal shift no

Heart normal 4-chamber view
normal outflow tracts
normal 3-vessel view
normal 3-vessel trachea view
normal situs/laterality
Abdominal Wall no abdominal wall defect
GIT **Stomach not visible**
Urinary tract kidneys and bladder appear normal
Extremities hands, feet, arms, legs and joints appear normal

Left Foot 46.9 mm
Left Humerus 40.4 mm
Left Femur 44.1 mm
Left Radius 34.2 mm
Left Ulna 38.8 mm
Left Tibia 37.1 mm
Left Fibula 36.9 mm
Left Femur / Foot 0.94

Doppler ultrasound

Uterine artery
PI left 0.890
PI right 0.830
Mean PI 0.860
RI left 0.60
RI right 0.53
Notch no notch
Assessment NORMAL DOPPLER STUDY

Fetal Echocardiography Suspected Diagnosis: normal
Situs normal - usual, solitus
Position of stomach normal - left-sided
Heart size normal - about one third of the thorax
Position of apex normal - apex to the left, angle of about 40 degree to midline
Systemic veins normal
Pulmonary veins normal - separate origins to each side of left atrium
Atrial septum normal - foramen ovale in the central third/half, flap valve in LA
Flow at foramen ovale normal - right to left
Atrioventricular junction normal - AV concordance
patent AV valves, equal size
AV regurgitation normal - none
Ventricular Septum intact
well seen
Ventricular function normal
Great artery connections: normal - concordant, forward flow in both thin valve leaflets, which 'disappear' in systole
Great artery connections concordant
normal
Arterial valve regurgitation normal - none
Branch pulmonary arteries normal - confluent
normal size
Arterial Duct normal - similar in size to arch, L sided, unaliased flow

Aortic arch normal
Side of arch normal - aorta to left of trachea
sinus - normal rate, regular

Cervical assessment

Cervical assessment accepted
Cervix length 37.1 mm
Funnelling **no**
Comment normal cervix
Previous Obstetric History after 36 weeks
Risk of delivery before 28 weeks 0.0 %
Risk of delivery before 31 weeks 0.1 %
Risk of delivery before 34 weeks 0.3 %
Risk of delivery before 37 weeks 2.0 %

Genetic Counselling

Maternal age 28.0 years

2nd Trimester Risk Assessment of Trisomy 21

Intracardiac echogenic focus marker is absent
Mild hydronephrosis marker is absent
Short femur marker is absent
Short humerus marker is absent
Echogenic bowel marker is absent
Increased nuchal fold marker is absent
Aberrant right subclavian artery marker is absent
Absent or hypoplastic nasal bone marker is absent
Ventriculomegaly marker is absent
Recommendations chromosomal study, NGS for determining the cause

Thanks for reference.
I have examined LALANI NIRALI, having date of birth 21/01/1990.
Fetal maturity is about 23 weeks and 1 day today.
Expected date of Delivery is 19/07/2018.

The Examination findings are as follows:

Present Ultrasound examination revealed **BILATERAL SEVERE PLEURAL EFFUSION and ABSENT STOMACH (pouch sign noted) ....possibility of Esophageal Atresia**
Fetal Biometry is suggestive of NORMAL fetal Growth as per gestation.
Normal and adequate quantity of liquor.
Risk for chromosomal abnormalities is more than 25 %.
Fetal heart evaluation done with standard cardiac USG views of 4 Ch, 3VV, 3 VTV, RVOT, LVOT and venous channels. STIC, TUI, M Mode, Power mode and color doppler were also used for the cardiac evaluation.
On present examination, NO major cardiac anomaly was seen.
All cardiac anomaly may not be diagnosed on antenatal fetal echo examination (detection rate of cardiac anomaly is from 25 to 76%).

Maternal cervix is of adequate length. Risk for preterm delivery is shown above
Evaluation of maternal Uterine Doppler shows normal, proper placentation.
Low risk for developing complications of Pregnancy Induced Hypertension, Fetal Growth Restriction etc.

Fig. 3

Management

A Lady came to OPD for routine antenatal check up with no complain. On per abdominal examination fundal height around 22-24cm and fetal heart sound present. On per vaginal examination no cervical dilatation and no effacement seen, cervical os closed. Accordingly USG finding we made a diagnosis of bilateral pleural effusion and maturity of 22-24 week of gestation. For second opinion patient sent to other center for USG. Report from other center confirming our diagnosis and said bilateral severe pleural effusin, fetal biometry was suggestive of normal fetal growth as per gestation, normal adequate quantity of liquor, risk for chromosomal abnormalities is more than 25%. So we advised this patient antenataly we can go for thoracocentesis, pleuroamniotic shunting, chromosomal study and delivery should be planned for a tertiary center with staff capable of resuscitating and managing neonates with respiratory compromise. In the delivery room, the neonatal team should anticipate the needs of the most severely affected patient and be prepared to provide respiratory support that includes intubation, positive pressure ventilation, and removal of fluid by needle aspiration.[9] But unfortunately patient never came again for follow up.

Discussion

The overall mortality rate is about 20-35% and the most common cause of neonatal death in a fetus diagnosed with hydrothorax is respiratory insufficiency due to pulmonary hypoplasia.[10] A conservative approach, antenatal thoracocentesis or pleuroamnoiotic shunting are the options for the management and the choice should be based on gestational age, severity of the effusion, evidence of progression, and the presence or absence of concomitant hydrops.[11]

Spontaneous regression has been reported to occur in 10–20%. However, the features predicting a better prognosis remain difficult to define. Unilateral effusion and absence of hydrops or hydramnios at the age of diagnosis seem to be indicators of a better outcome.[12] The presence or progression of hydrops and rapid enlargement of a pleural effusion with mediastinal shift are indicators of a poor prognosis and for urgent fetal intervention.

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How to cite this article: Senta J, Badi I, Hingora MA, Kanziya J, Pandya M. Rare case of fetal hydrothorax. Ind J Obstet Gynecol Res. 2018;5(3):424-428.