Rare case of fetal hydrothorax

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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 bronchopulmonary lesion, 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 case of bilateral а а 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, SpO₂-98%. 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.

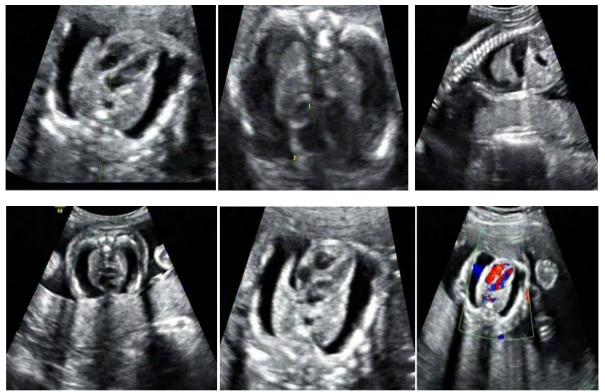


Fig. 1



Fig. 2

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Patient Data		Indication	Routine, 3D/4D with fetal anomaly scan
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	95 X00000000000	PI	robe 3.5 MhZ
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Addres	55 X00000000000 .	Contational	
		Biometry / Anatomy	age 23 weeks + 1 days
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	e xxxxxxxxxxxxx		DFD 63.1 mm DFD 80.9 mm
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	Hindu - Gujarati		CM 5.4 mm
Partner		Т	CD 24.2 mm
Racial origi	n South Asian (Indian, Pakistani, Bangladeshi)	Ventricular atr	
Present Pregnancy	(interaction of an and and a building addesing)		AC 207.6 mm FL 44.1 mm
	s last period: 12/10/2017	Hume	rus 40.4 mm
Date		BPD / C	FD 0.78
Conception	e regular - LMP sure 28 days		AC 1.09
	spontaneous	BPD /	FL 1.43
EDD by date		Estimated fetal wei	ght Hadlock (BPD-HC-AC-FL)
	n 19/07/2018		756 g 1 lbs 10 oz
	B, Rhesus positive	Fetal heart activ	
Weigh	t 56.1 kg	Fetal moveme	nts normal
Heigh	t 160.0 cm	Fetal heart ra	ate 150 bpm
Body mass index	(21.9	Presentati	on cephalic dorso-anterior
Cigarettes		Placenta s	ite posterior high, Placenta grade Grannum I
Obstetric History		Amniotic fit	aid normal
	>= 2710/-1	Cord insertion	ord 3 vessels
Living children		Head	normal skull shape
Previous caesarean		(Brain	hemispheres, ventricles mid-brain and posterior fossa appea
Type of last delivery			normai
Gravida	2 Para 1		eft 9.1 mm
Chronic Disease			eft 4.6 mm
Chronic hypertension	no	Face	no facial cleft and the eyes, nose and mandible appear norm
Family History			ie 6.1 mm
Tanny motory	Patient: uncomplicated family history	Prenasal thicknes	
	Partner: uncomplicated family history	Neck/Skin	no spina bifida or kyphoscoliosis no skin oedema or cystic hygroma
Consanguinity	no		no skil oedema or cystic hygroma
- total		Nuchal fold thicknes	5.7 mm
Examination		Nuchal fold thicknes	
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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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