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## Case Report

# Case report of serial pleural effusion managed with good fetal outcome

Shreya I. Patel, Priyadatt Patel\*, Mayank Choudhary, Meena Jhala, Harmi Thakkar

Department of Obstetrics and Gynecology, Balaji Horizon Women's Hospital, Ahmedabad, Gujarat, India

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### \*Correspondence:

Dr. Priyadatt Patel,

E-mail: priyadatt112@gmail.com

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### ABSTRACT

Fetal pleural effusion is a rare congenital anomaly that occurs when some amount of fluid surrounds the fetal lung without concomitant hydrops. It may remain the same or progress more. We present here a case of a 32-year-old lady with primary infertility who conceived naturally. She presented to us for her routine term pregnancy evaluation. At 17 weeks her targeted imaging for fetal anomalies (TIFFA) scan revealed left side severe fetal hydrothorax. Again at 29 weeks left pleural effusion was noted. A C-section was performed at 38<sup>th</sup> week of pregnancy and a male child weighing 2.995 grams was born, with no need for ventilator support.

**Keywords:** Fetal thoracocentesis, Hydrothorax, Fetal pleural effusion, Amniocentesis

### INTRODUCTION

The incidence of fetal pleural effusion estimates to be 1:10000 to 1:15000 pregnancies, they can be classified as primary or secondary.<sup>1</sup> The causes of primary effusions can be lymphatic leakage due to raised intrathoracic pressure, they can either be unilateral or bilateral that may progress to hydrops. However, secondary effusions are usually a part of generalized fluid retention in immune hydrops, chylothorax, infections and genetic disease.<sup>2,3</sup> In case of smaller effusions, they can resolve spontaneously or remain stable, whereas larger effusions may lead to fetal hydrops, pulmonary hypoplasia, esophageal compression with resultant polyhydramnios and preterm birth. The proper antenatal management is controversial, it is observed that some fetuses are not compromised, meanwhile others either die in - utero or at birth from pulmonary hypoplasia and/or prematurity.<sup>4-8</sup> Hereby, we report our experience with intra partum thoracocentesis in fetus with bilateral pleural effusion.

### CASE REPORT

Patient with primary infertility aged 32 years was managed with laparoscopy + bilateral tubal testing + adhesiolysis

hysteroscopy + fundal synechiolysis. She conceived naturally in the same period cycle post procedure. Her Nuchal translucency scan at 12 weeks 2 days was normal with enhanced 1<sup>st</sup> trimester screening eFTS showing low risk for trisomy 13, 18, 21. It showed an increased risk for pre-term pre-eclampsia with test probability of 1:71. Apart from routine prenatals and iron and calcium tablets, she was started on ecosprin 150 mg once daily. Her tetanus and flu vaccinations were completed.

Her TIFFA scan at 17 weeks 1 day revealed normal growth and normal left side severe fetal hydrothorax 45×15×29 mm. Left side lung was collapsed and right lung not collapsed. Heart was pushed to right side. After counseling patient underwent bilateral thoracocentesis and amniocentesis 20 ml was drained in 1<sup>st</sup> procedure at 19 weeks: amniotic fluid rapid sure prenatal microarray analysis was normal; fetal pleural fluid for cytological examine – mild chronic non-specific inflammation, CMV RTPCR – negative, PB19 negative; and fluid cytology normal.

At 27 weeks 2 days' growth scan was normal + Doppler scan was normal; AFI – normal. 73×49×34 mm<sup>2</sup> left pleural effusion noted with collapsed left lung. At 29

weeks and 2 days bilateral thoracocentesis + amniocenteses were done for the second time. At 31 weeks 2 days' growth scan + Doppler – normal, AFI – 17cm, 61×17×55 mm left hemithorax. At 35 weeks 2 days' growth + Doppler – normal, AFI – 10cm. After Both Tapping fluid accumulated again after 7 days. Magnetic resonance imaging (MRI) at 37 weeks 2 days showed: pleural effusion in left hemithorax with passive ipsilateral lung compression. Trachea + bilateral bronchi are normal + patent, tracheal displacement is 8.2 index. Both lungs show abnormal heterogeneity. Our signal with fluid filled channels extending to pleural surface in right lung. Lung to liver signal intensity ratio of <2/gr III. Primary pulmonary lymphangiectasia patient was delivered via caesarian section – male child weighing 2.995 kg was born. Baby did not require ventilatory support and was on spontaneous respiration.

### **Postnatal management**

#### *Respiratory system*

On admission he was maintaining saturation on room air without distress. Chest X-ray was suggestive of left pleural effusion. 20-gauge long aspiration needle was inserted on 2nd DOL and pleural fluid was drained. Pleural fluid cytology was suggestive of chol 19, TG-20, TC-580, POLY -20, lympho - 80, RGB>1000, ALB-2.0, glucose-81, protein fluid-2.2.

USG was done on 3rd DOL which showed weak left diaphragmatic movement, no pleural effusion on side. Pigtail catheter was clamped after that. After 1 day of clamping the catheter, USG was repeated which showed no pleural effusion on left side. Pigtail catheter was removed on 6th DOL.

#### *Cardiovascular system*

Hemodynamically he remained stable. No inotropes required.

#### *Sepsis*

No antibiotics were given. Septic screen was negative.

#### *Feeding/diet*

On admission he was kept NBM. RTF (simyl MCT) was started on 2nd DOL and graded up and he reached full RTF on 3rd DOL. Spoon feeds by mother was tried on 6th DOL which he took well. He was shifted in room with mother on 7th DOL and discharged next day with advise of spoon feeds (Simyl MCT formula) as required. Medium chain triglycerides are very important for digestion and absorption of nutrients, such as disturbed bile secretion, classic coeliac disease, short bowel syndrome, inflammatory diseases of the intestines, disturbed outflow of lymph, some metabolic disease, and severe food

allergies, as well as in prematurely born neonates. He passed urine and meconium within 24 hours.

#### **USG head**

USG of the head was normal.

X-ray post-delivery recorded bilateral lung expansion. It seems that, due to cardiovascular circulatory changes post-delivery, pressure gradient of lymphatic system might have been altered in a favorable way along with the pressure from the expanded lung. All these factors cumulatively might have worked as a good prognostic factor.

The child in Figure 1 is the child 8 months' post-delivery.



**Figure 1: Child 8 months' post-delivery.**

### **DISCUSSION**

When abnormal amounts of fluid forms within the chest of a fetus it is called fetal hydrothorax. This fluid may be in the space between the lungs and the chest wall (pleural space) or within the core of the lung or chest masses. Fetal hydrothorax may also be referred to as a pleural effusion. Because the chest is an enclosed space, the presence of fluid can compress the lungs and even displace the heart.

Compression of the lungs can interfere with their normal development in the womb. When this occurs, the lungs may not allow oxygen intake at the normal level (pulmonary hypoplasia). If fetal hydrothorax causes the heart to move it is called a mediastinal shift. When this happens a fetus has trouble receiving and pumping blood and may develop heart failure. When fetal hydrothorax worsens to cause fluid accumulation in other parts of the fetal body, it is called fetal hydrops.

Thoracentesis is a procedure to remove fluid or air from around the lungs. A needle is put through the chest wall into the pleural space. The pleural space is the thin gap between the pleura of the lung and of the inner chest wall.

Fetal hydrothorax may be detected during a routine ultrasound. A maternal-fetal medicine specialist will also perform a test known as fetal echocardiogram. This test will allow him/her to determine the severity of the illness and the condition of the heart.

If a case of fetal hydrothorax is linked to fetal lung failure or fetal cardiac dysfunction, doctors may recommend one of the following prenatal intervention procedures:

#### **Fetal thoracocentesis**

In this procedure, doctors, under the guidance of ultrasound, insert a small needle into the chest of the fetus and drain the fluid. During thoracocentesis, doctors are also able to obtain amniotic fluid, which can be tested along with the chest fluid, for underlying conditions that may have led to fetal hydrothorax. In up to 10 percent of patients, this procedure completely resolves fetal hydrothorax. However, many patients experience a recurrence of fluid buildup. For these patients, repeat thoracocentesis is not a viable alternative, as it will not be able to prevent underdeveloped lungs.

#### **Thoracoamniotic shunting**

For this procedure, doctors insert a small plastic tube (pigtail catheter) into the fetal chest. This allows the fluid to drain into the amniotic cavity in the uterus. This treatment provides relief of hydrothorax, continuous decompression of the fetal chest and offers the best chance to prevent underdeveloped lungs.

Studies have shown that pleural effusion can disappear spontaneously after thoracocentesis.<sup>9</sup> Thoracocentesis may be considered to be repeated when pleural effusion occurs again. Depending on the repeated thoracocentesis hypoproteinemia can develop and this may cause the development of hydrops.<sup>10</sup> Therefore, caution should be taken. Thoracocentesis done before the birth, lessened the burden of respiratory of fetus at neonatal period.<sup>11</sup>

In determination of the correct treatment modality, gestational age is also important. Aubard et al propose thoracoamniotic shunt placement before 32nd gestational weeks and the thoracocentesis after 32nd gestational week.<sup>12</sup>

#### **CONCLUSION**

Cases of fetal hydrothorax are creating new challenges, protocol based timely intervention and investigation leads to favorable fetal outcomes. Pregnancy itself is complicated if there is abortion, preterm labor, polyhydramnios, fetal death in the womb or any congenital abnormality. There are three methods available for managing fetuses with fetal hydrothorax: serial thoracocentesis, thoracoamniotic shunting and

thoracomaternal cutaneous drainage after due investigation testing are done. We conclude that early diagnosis and intervention with justified sequential thoracocentesis which is less invasive and reduces the requirement of shunting/drainage (more invasive) leads to good prognosis in selected case after counseling.

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