

SINGLE THORACENTESIS IN UNILATERAL FETAL HYDROTHORAX: A CASE REPORT

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ABSTRACT

A case with a pleural effusion and hydrops fetalis has been presented. Good outcome had been achieved by a single thoracentesis and a healthy baby was delivered at the 39th week of pregnancy. In a fetus with a pleural effusion and hydrops fetalis, normal like echogenic view of the affected side lung and lung expansion during aspiration without any signs of fetal distress are good prognostic signs. We believe that in such conditions performing a single thoracentesis could be the first choice of treatment, which is a less complicated and traumatic method than thoraco-amniotic shunting.

Key Words: Single thoracentesis, Fetal hydrothorax

INTRODUCTION

Primary fetal hydrothorax (PFHT) is fetal intrathoracic effusion that may occur uni-or bilaterally. The incidence of this uncommon disorder is about 1 in 10,000 – 15,000 pregnancies. It can develop and be associated with a variety of etiologies. Fetal pleural effusion

may occur as a part of fetal hydrops and more rarely, as an isolated lesion. Isolated pleural effusion may progress to generalize hydrops; thus, fetal pleural effusion may represent an early stage in the development of nonimmune hydrops and may be more common than appreciated. On the other hand, such an isolated hydrothorax may progress to nonimmune hydrops fetalis (1).

The outcome of PFHT is variable and unclear; the evolution can result in spontaneous regression, progress to hydrops and no change with an unpredictable clinical course. Thus, the reported perinatal mortality varies between 34,8% to 100%. The clinical therapeutic approach is debated and variable. Thoracentesis and pleuroamniotic shunting are the choices of treatment (2,3).

Recently, pleuroamniotic shunting seems to be the first choice of treatment as quick reaccumulation takes place after a single drainage. Some authors are in favor of thoracoamniotic shunting, and they find single drainage unsatisfactory to prevent pulmonary hypoplasia, and to reverse hydrops and polyhydramnios (4,5). By this case report we tried to discuss the conditions where single

thoracentesis could be helpful in the management of PFHT.

CASE REPORT

A 19-year-old woman, G:2/P:1; applied to Obstetric and Gynecology Department, Cerrahpaşa Medical School, University of Istanbul, for a targeted ultrasound scan at 28 weeks of gestation for a possible pleural effusion. Ultrasound examination revealed massive unilateral pleural effusion, abdominal ascites, scalp and subcutaneous edema and polyhydramnios. Massive left pleural effusion had displaced the heart to the right, and the left lung was compressed and displaced to midline (Figs.-1a, b). There was no major cardiac defect and pericardial effusion on fetal echocardiography. Diagnostic fetal blood sampling was performed for chromosomal analysis 46 XX, hemoglobin 15g/dl, platelet $170000/\text{mm}^3$ and TORCH infections were negative.

Treatment options and the associated risks and benefits were explained to the parents. The fetus was observed without any intervention till the 32nd week of gestation. During the follow up the subcutaneous edema and abdominal ascites regressed spontaneously and at the 32nd week of gestation, the only sonographic findings left were pleural effusion with polyhydromnios (Fig.-2a). With this favorable follow-up we waited for another 2 weeks, but there was an increase in the amount of amniotic fluid. The pleural effusion remained unchanged and the left lung collapsed. In order to relieve the compression off the left lung, single thoracentesis was performed at 34 weeks of gestation. With the use of a 20-gauge needle under ultrasound guidance 70 cc of clear golden fluid was removed from the left fetal chest. After the procedure, shifted heart returned to the normal position and the left lung expanded (Fig.-2b). The fluid composition was consistent with chylothorax (2.5gr/lit albumins, 80% of the cells were mononuclear cells) and negative bacteria culture.

Weekly ultrasound examinations were performed after thoracentesis. There was no fluid reaccumulation and the amount of amniotic fluid

decreased to normal levels within one week (Fig.-3). Caesarian section was performed because of fetal bradycardia during the labor at 39 weeks of gestation. A 2750 gr female infant with Apgar scores of 8/9 was delivered. The infant had a normal neonatal course. The infant's chest X-ray revealed normal left and right lung. Mother and infant were discharged home on day 4 and a following examination at 12 months revealed a normally developing infant.

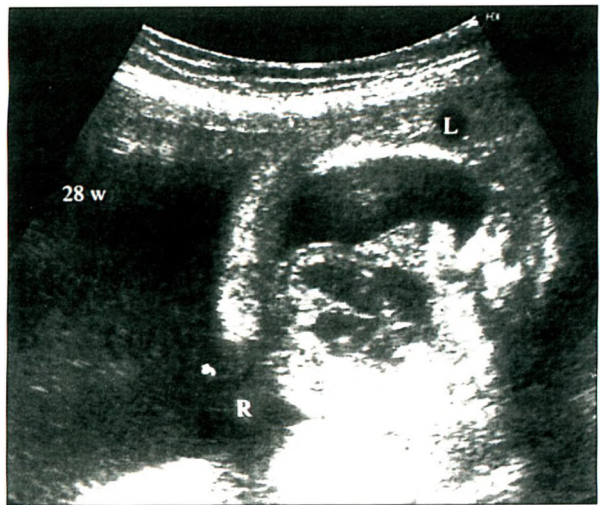


Fig. 1 a : Pleural effusion is demonstrated. heart is shifted to right mediastinum and left lung is of normal ultrasonic echogenicity and collapsed.

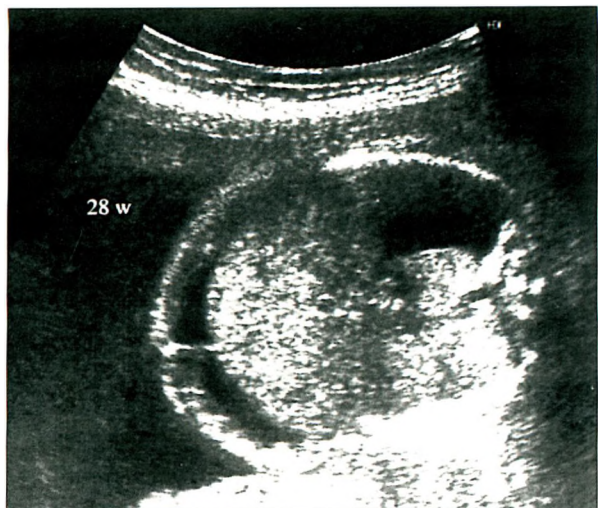


Fig. 1 b : Fetal abdominal ascites. at 28 weeks of gestation.

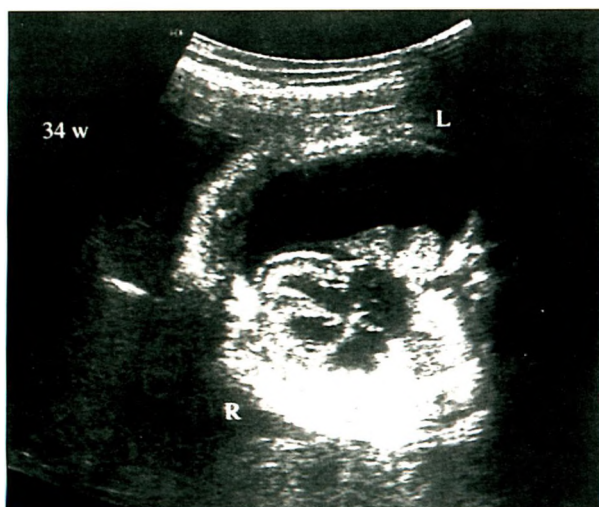


Fig.2a : Before thoracentesis. pleural effusion and fetal chest.

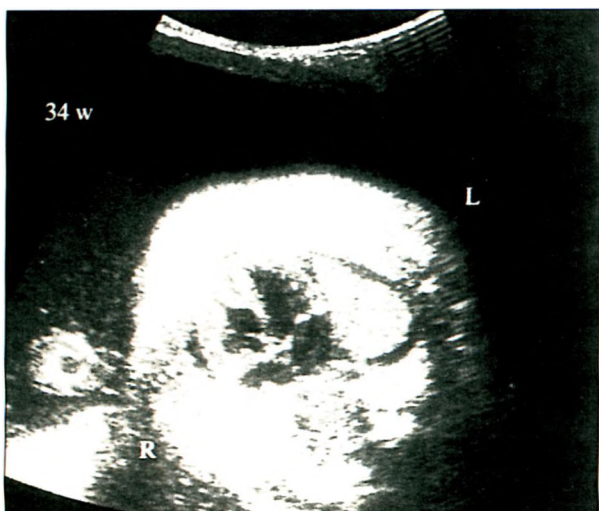


Fig.2b : The view of fetal thorax immediately after thoracentesis: the lung has expanded and the heart has returned to normal position at 34 weeks of gestation.

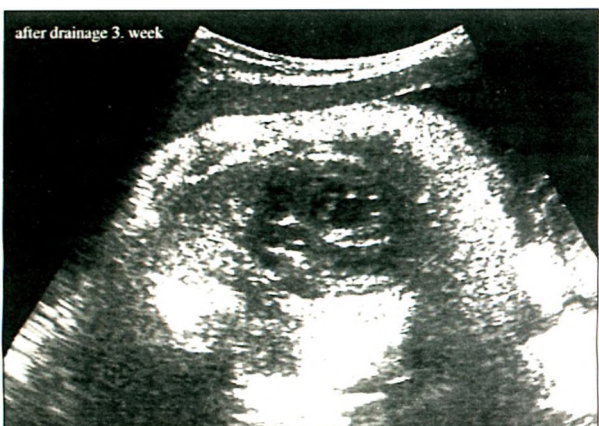


Fig.3 : The fetal chest. 3 weeks after drainage: there was no reaccumulation of pleural effusion.

DISCUSSION

Fetal morbidity and mortality rates increase in the presence of pleural effusion as a result of the developing pulmonary hypoplasia. The end stage of lung development is the alveolar stage, which starts at 22-24 weeks of gestation and is completed in postnatal 10 years (6). The gestational week, at which pleural effusion occurs, affects lung development. It was observed that the less the gestational week, the worst the pulmonary hypoplasia and so, in such fetuses polyhydramnios is seen to occur earlier in time.

Harrison et al. (7) showed that lung development of thoracally-decompressed animals is normal and that on the contrary; the compressed lung would be hypoplastic. On the other hand, it was shown that patients with the pleuroamniotic shunt applied beyond the 22nd gestational week had a low fetal mortality and absent pulmonary hypoplasia. Thoracentesis or pleuroamniotic shunt applied to fetuses with pleural effusion observed before 24-26 gestational weeks showed an increase in mortality rate (8).

Prognosis is even worse in those with pleural effusion together with non-immune hydrops. About 30% of these fetuses die in the antenatal period. Generally, non-immune hydrops fetalis due to hydrops, prematurity and pulmonary hypoplasia has a 36% perinatal mortality rate (3). These findings and studies show that it is a must to apply thoracic decompression in either hydrops associated or isolated type pleural effusions to decrease the fetal mortality rate. Thoracentesis was accepted to be the first choice for decompression. Nevertheless, reaccumulation of pleural effusion was seen later in these patients. Therefore, thoracentesis was replaced by pleuroamniotic shunt that is a more complicated and expensive procedure.

Reported in literature are few cases of fetuses with hydrops associated pleural effusion where more than one thoracentesis was applied as a way for successful treatment. Petres et al. (8) applied antepartum and postpartum thoracentesis for a bilateral chylothorax patient and a postnatal thoracic tube as treatment. Schmidt et al. (9) reported a case, where postnatal fluid reaccumulation occurred in a

thoracentesis applied patient. Longaker et al. (1) in a 5 patient series, were obliged to perform pleuroamniotic shunt in 2 fetuses without hydrops after more than one thoracentesis had been applied. Arguirre et al.(10) treated a baby with hydrops by applying antepartum thoracentesis twice. Aubard et al.(3) reported after a wide analysis that only 3 patients, where only one thoracentesis was applied, gave an appropriate fetal outcome . On the other hand, Rodeck et al. (4) and Nicolaidis (5) suggested that it would be more appropriate to perform immediately pleuroamniotic shunting in hydrops associated or isolated type pleural effusions. All these reports show that thoracentesis is not enough and does not lead to a good outcome in hydrothorax or pleural effusion. Besides, there is no idea, about which patients will respond well to thoracentesis.

In our case, the isolated hydrothorax fetus was treated on the 34th gestational week by only one thoracentesis. Here, the hydrops fetalis diagnosed on the 28th week of gestation was seen to regress at 32. weeks of gestation. According to this regression, it was thought that pleural effusion would also regress so we waited till 34 weeks of gestation. There was no sign of pleural effusion regression. On the other hand, polyhydroamnios was seen to take place. In the same week, the left lung lobes had the same ultrasonic echojenicity with those of the right lung and it was seen to be collapsed.70cc of fluid was taken on one trial thoracentesis at 34 weeks of gestation. While taking the fluid, the heart was recognized to change place back to the left and the left lung expanded. Meanwhile, no fetal distress was recorded (such as bradychardia). One week after aspiration of pleural effusion, polyhydroamniosis was seen to regress and no reaccumulation signs were noticed. We think that the good outcome, we got after performing a single thoracentesis on our patient is relative to the following reasons:

1-Pleural effusion that started together with the hydrops took an isolated form after regression of the hydrops. Thus, the intraabdominal pressure over the fetal circulation was released which positively affected the fetal circulation. During follow-up no Doppler signs of fetal asphyxia were seen.

2-The left lung lobes had the same ultrasonic echojenicity of those of the right lung which was considered as a good prognostic sign. Although the left lung collapsed, the normal echojenicity signs show us that pleural effusion occurred after the end stage (alveolar) development of the lung had taken place and that the pulmonary circulation was near normal.

3-After thoracentesis, the heart changed place rapidly back to the left mediastinum and the left lung expanded at the time, which we thought to be ideal for no reaccumulation of fluid to occur. As a matter of fact, regression of polyhydroamnios and no reaccumulation occurrence after the aspiration support our physiopathologically based thoughts.

Although thoracic shunting is preferred as a first line treatment method, our case was treated successfully with single thoracentesis. We consider, regression of fetal hydrops during follow-up, seeing normal or normal like echojenicity of the collapsed lung ultrasonographically and expansion of the lung during pleural aspiration without any signs of fetal distress, as criterias for success in the treatment of pleural effusion with single thoracentesis. Therefore, thoracentesis as a simpler mode of treatment could be applied before a more complicated and expensive procedure like pleuroamniotic shunting.

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