Cardiotocographic and Doppler velocimetric patterns, pre- and post-thoracentesis, in a case of fetal hydrothorax

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Summary

Fetal hydrothorax is associated with elevated perinatal mortality. Management of this condition is controversial given that in utero spontaneous resolution has been described. A case of fetal hydrothorax associated with an extralobar lung sequestration that showed pathologic cardiotocographic patterns and abnormal Doppler velocimetry indices in several fetal vascular beds is reported. All pathologic patterns improved after fetal thoracentesis. It can be concluded that monitoring fetal well-being by means of cardiotocography and Doppler velocimetry may help in timing thoracentesis in cases of fetal hydrothorax.

Key words: Cardiotocography; Doppler; Fetal hydrothorax; Thoracentesis.

Introduction

Primary fetal hydrothorax may be observed as an isolated finding or in association with structural and caryotypical abnormalities. It occurs in 1:15,000 pregnancies and, if untreated, carries a perinatal mortality of 50%, which approaches 100% in cases with associated hydrops [1]. In spite of the high fetal mortality, few data are available in the literature regarding circulatory changes in these cases. Unilateral pleural effusion can shift the mediastinum, impair venous return and lead to congestive heart failure and hydrops.

The beneficial use of cardiotocography and Doppler velocimetry in monitoring fetal well-being in a case of fetal hydrothorax associated with an extralobar lung sequestration is described.

Case Report

A 37-year-old gravida 2, para 1 at 32 weeks' gestation was referred to the Chieti University obstetrical service because of polyhydramnios and fetal hydrothorax. Upon arrival the cervix was 1 cm dilated and 40% effaced, the fetal heart rare (FHR) tracing was reactive and regular uterine contractions were noted every three minutes. An ultrasound examination showed an amniotic fluid index of 46 cm and left fetal hydrothorax with right mediastinal shift (Figure 1). Fetal growth and morphology appeared otherwise normal. Intravenous beta mimetics for tocolysis and corticosteroids for lung maturity were started.

After three days of therapy, during which the FHR tracing was always reactive, the pleural fluid increased, ascites appeared and the FHR tracing showed reduced variability and variable decelerations (Figure 2). Doppler velocimetry of fetal cerebral vessels suggested decreased resistance. Peak velocities in the ascending aorta and pulmonary artery were reduced and an increased percentage of reverse flow in the inferior vena cava was also observed (Table 1). By means of a 20 gauge needle, under sonographic guidance, 80 cc of fluid was drained from the left hydrothorax, with normalization of the mediastinal position. With the same needle 1000 cc of amniotic fluid was slowly removed. Four hours later the FHR tracing became reactive (Figure 3) and Doppler velocimetry of fetal vessels returned to be within normal limits (Table 1). Bacterial cultures of the fluid obtained at prenatal thoracentesis were negative. The white blood cell count was 100 per mm^3, with 53% lymphocytes. In the following two weeks fluid partially reaccumulated in the left thorax, without signs of fetal distress. At 34 weeks plus 5 days, after establishment of fetal lung maturity, a 2280 gram male infant was delivered by repeat cesarean section. The caryotype was normal and the neonate was diagnosed with an extralobar lung sequestration, that was surgically removed without complications. Parvovirus B19 infection was ruled out by specific antibodies and viral genome polymerase chain reaction. The elevated percentage of lymphocytes in the pleural fluid, together with the presence of numerous dilated lymphatic vessels in the resected lung sequestration, were suggestive of a diagnosis of chylothorax.

Discussion

Fetal hydrothorax is associated with elevated perinatal mortality: in a review of 11 prenatally diagnosed cases, Romero *et al.* reported five perinatal deaths [2]. In the preterm fetus, drainage of the pleural fluid may prevent the development of generalized hydrops and pulmonary hypoplasia [3], however the management of fetal hydrothorax is controversial given the fact that in utero spontaneous resolution has been described [4]. It has been suggested that the management might have to depend on the degree of mediastinal shift: if the pleural effusion is small and no mediastinal shift is demonstrated intervention might not be justified, on the other hand if significant mediastinal shift or hydrops develop, intervention is warranted [2].

In spite of the elevated fetal mortality we found only few data regarding fetal well-being monitoring in such cases. Cardiotocographic signs of fetal distress in the pre-

Table 1. — *Doppler velocimetry of fetal vessels immediately before and 4 hours after thoracentesis*

	Prior to thoracentesis	After thoracentesis
Umbilical artery (PI)	0.81	0.87
Middle cerebral artery (PI)	1.12	1.61
Ascending aorta (peak velocity)	59.7	71.0
Pulmonary artery (peak velocity)	58.1	65.4
Transmitral E/A	0.60	0.69
Transtricuspid E/A	0.61	0.74
Inferior vena cava (percentage reverse flow)	13.9%	9.2%
Ductus venosus (PV/A)	3.1	2.0

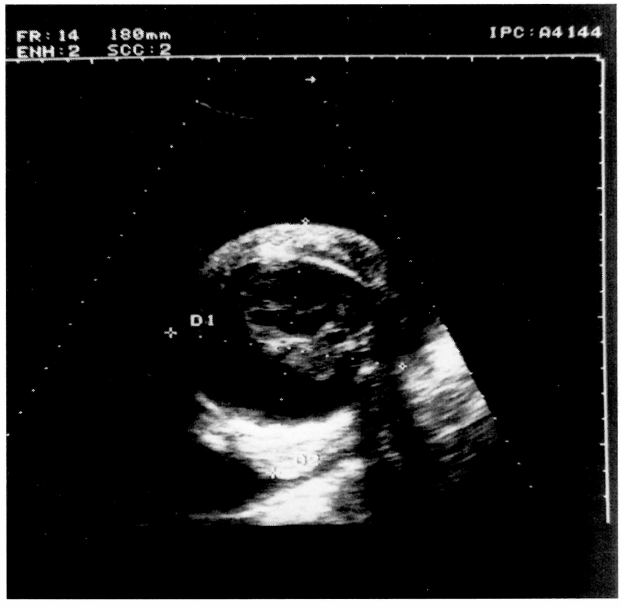

Figure 1. — Left fetal hydrothorax with right mediastinal shift.

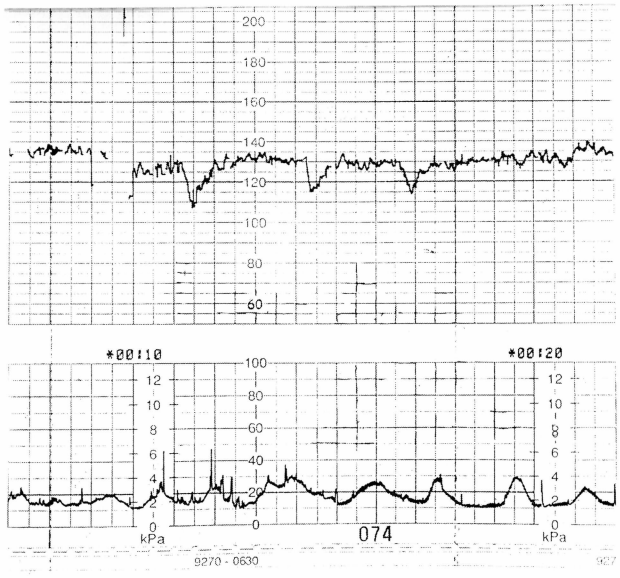

Figure 2. — Fetal heart rate trace before thoracentesis, showing reduced variability and variable decelerations.

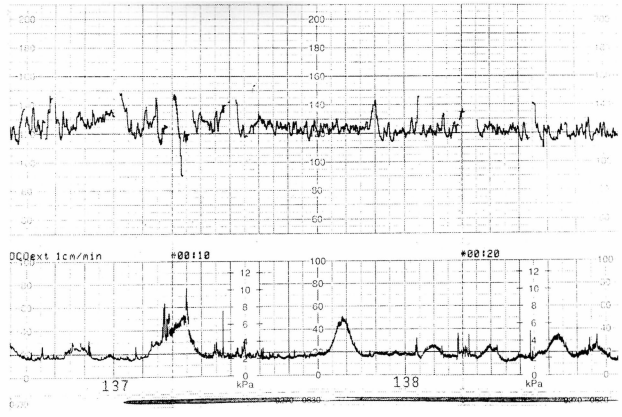

Figure 3. — Fetal heart rate trace after thoracentesis, showing normal reactivity.

sence of fetal hydrothorax with significant mediastinal shift have been described by Mandelbrot *et al.* Similar to our case, when pleural effusion became significant, sudden deterioration of fetal well-being became evident by the onset of reduced FHR variability, late deceleration, and the presence of ascites [5].

In addition to cardiotocographic parameters, we also looked at Doppler velocimetry of various fetal vascular beds, before and after thoracenthesis. The reduced transmitral and trans-tricuspid E/A ratios, together with the increase in both the percentage of reverse flow in the inferior vena cava and the PV/A ratio in the ductus venosus, observed in our study, likely reflect decreased ventricular compliance, because of compression on the heart walls, and hampered venous return because of the heart latero-deviation. The diminished peak velocities observed in the ascending aorta and pulmonary artery are likely consequent upon reduced diastolic filling and ventricular compliance. In this situation peripheral oxygen delivery is impaired, leading to decreased cerebral vascular resistance and abnormal cardiotocographic patterns. The observation of prompt normalization of the altered Doppler and cardiotocographic parameters a few hours after thoracentesis, in our case, make it likely that heart compression and latero-deviation were the causes of the observed signs of fetal distress. No changes were observed in umbilical artery Doppler velocimetry, since the villous arterial bed is not involved in this clinical situation.

Polyhydramnios has been described in association with fetal hydrothorax [5] and might occur secondary to compression of the esophagus, while fetal ascites is probably related to the decreased cardiac efficiency. The association of fetal hydrothorax and extralobar pulmonary sequestration has already been described [6-7], its etiology being attributed to vascular overciculation [8], abnormal lymphatic drainage [9] and torsion of the sequestration with occlusion of the efferent venous and lymphatic channels [10].

We conclude that fetal hydrothorax with significant mediastinal shift may impair cardiac systemic venous

return, adequate cardiac diastolic filling and, consequently, adequate cardiac systolic output, leading to acute fetal distress. Cardiotocography and Doppler velocimetry may show those cardiocirculatory changes. Emergency prenatal thoracentesis may reverse fetal distress in fetuses with hydrothorax not associated with other anomalies, allowing time for corticosteroid therapy and delivery.

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