

An unexpected temporary fetal acid reason: rupture of fetal ovarian cyst

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Abstract

Objective: To discuss the relationship between isolated fetal acid cases and fetal ovarian cysts.

Case: We presented an isolated fetal acid case developing the rupture of fetal ovarian cyst.

Conclusion: Fetal hydrops may occur depending on the systemic or local pathological processes. With this case, we concluded that the rupture of fetal ovarian cyst may be the reason of isolated fetal acid.

Keywords: Fetal acid, fetal ovarian cyst, rupture of fetal ovarian cyst.

Özet: Beklenmedik bir geçici fetal asit nedeni: Fetal over kisti rüptürü

Amaç: Bir olgu nedeniyle izole fetal asit olguları ile fetal over kistlerinin ilişkisine değinmektir.

Olgu: Fetal over kisti rüptürüne bağlı olarak gelişen bir izole fetal asit olgusunu sunduk.

Sonuç: Fetal hidrops sistemik veya lokal patolojik süreçlere bağlı ortaya çıkabilmektedir. Biz bu olgu ile fetal over kisti rüptürünün izole fetal asit nedeni olabileceğini sunduk.

Anahtar sözcükler: Fetal asit, fetal over kisti, fetal over kisti rüptürü.

Introduction

Fetal acid can be accepted as the indication of fetal hydrops and it may develop as a response to various etiological factors. With the wide use of Rh immunoglobulin recently, non-immune hydrops cases are seen more frequently than immune hydrops cases. Due to many different fetal reasons, fetal acid may developed as an isolated way independent from other serosal cavities and subcutaneous tissue. The reasons of isolated primary fetal acid are not clear. Although some of them regress spontaneously, the condition progresses towards fetal hydrops in many cases. Ovarian cysts are the abdominal cystic masses observed frequently in female newborns. They are generally not symptomatic and disappear spontaneously. Together with the wide use of ultrasonography, ovarian cysts are diagnosed

more often both in fetuses and newborns. These formations are followed up and monitored since they are usually asymptomatic and have no clinical significance. In this report, we have presented a case with the rupture of fetal ovarian cyst which is a rare and unexpected condition for fetal acid.

Case Report

Thirty-four-year-old patient (gravida 2, parity 0) with a pregnancy loss at early period referred to our clinic at 32 weeks of gestation due to fetal intraabdominal cystic formation. Her pregnancy progressed normal until this week and no pathology was found in the follow-up. There was no concomitant apparent disease in her anamnesis. During the pregnancy, she attended all pre-

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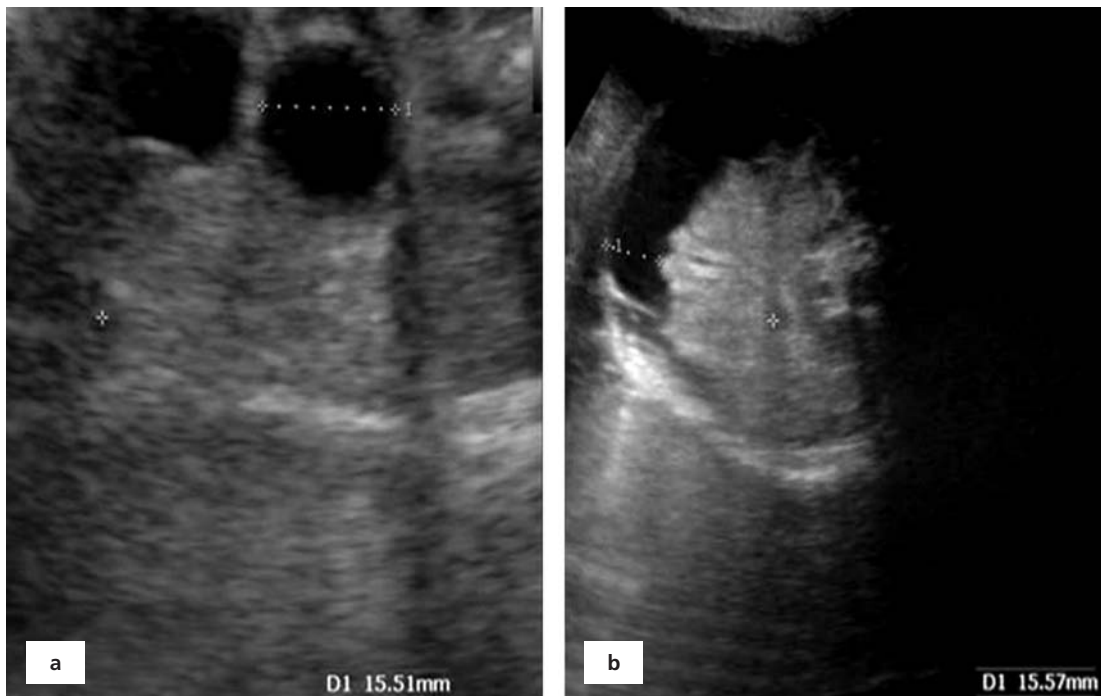


Fig. 1. (a, b) 15 mm cystic formation in left fetal ovary.

natal visits and no abnormal finding was established. In her ultrasonography at 32 weeks of gestation, the fetal biometry was consistent with gestational age, and amniotic fluid volume was normal. Her blood pressure and fetal movements were normal. In the ultrasonography, 15 mm round cystic formation was found in the left inferior fetal abdomen (**Fig. 1**). Cystic formation was independent from fetal kidneys and bladder. Pericystic or intracystic vascularity was not observed in the Doppler ultrasonography. Bilateral fetal kidneys and intestines were in normal structures. Since the fetus was female, had a round anechoic structure and not associated with urinary and gastrointestinal structures, ovarian origin was suspected first. Therefore, re-evaluation was made for maternal diabetes screening and thyroid functions, and the results were normal. The patient was asked to come for a check 2 weeks later. In the ultrasonography carried out at 34 weeks of gestation, fetal biometric measurements were consistent with gestational age and amniotic fluid was within normal ranges. Only abdominal circumference was measured as bigger than gestational age, and it was found as consistent with 9th percentile. There was diffusive free fluid in the fetal abdomen (**Fig. 2**). It was seen that the cystic formation

in the left ovary regressed and its diameter was measured as 14 mm (**Fig. 3**). It seemed that the intestines were floating in the free fluid. No effusion was observed in the thorax and there was no edema in the subcutaneous tissue; therefore, it was defined as isolated primary fetal acid and further evaluation was performed for hydrops fetalis. Full blood count, HbA1c, VDRL and TORCH screenings, Parvovirus B-19 screening, indirect coombs test and anti-cardiolipin IgM-IgG screenings were carried out. Peak systolic velocity of middle cerebral artery was found 55 cm/sec (1.12 MOM) and it was not predictive for fetal anemia. As it may be the indication for fetal hydrops, fetal karyotyping by cordocentesis was recommended; however, the patient did not accept karyotyping. Fetal cardiac examination was evaluated as normal, and all screening procedures were resulted normal. The patient was asked to come for a check 2 weeks later. Ultrasonography made at 36 weeks of gestation showed normal results. The free fluid in the fetal abdomen was completely regressed and ovarian cystic formation disappeared. At 40 weeks of gestation, the patient delivered a healthy 3400 g baby. No abnormal formation was found in the postnatal ultrasonography of the baby.

Discussion

Hydrops fetalis is defined as pathological effusion in soft tissues and at least two serosal cavities due to immune or non-immune reasons. While placental edema and subcutaneous edema accompany the condition, pericardial, pleural fluid collection or acid-like pathological fluid collection accompanies in serosal cavities.^[1] They are classified as immune or non-immune hydrops fetalis. Isolated fetal hydrothorax or isolated acid is pathological fluid collection as isolated in these cavities without any subcutaneous edema depending on various etiologies.^[2] Fetal acid frequently accompanies subcutaneous edema which is a component of fetal non-immune hydrops and/or fluid collection in other serosal cavities. Multiple factors consisting of chromosomal disorders, intrauterine infections, fetal cardiac failure and structural disorders of various organs are among the etiology of non-immune hydrops fetalis. The cases where fetal acid develops independent from other serosal cavities or organs are called as isolated fetal acid. Isolated fetal acid can be the indication of hydrops fetalis and may progress towards hydrops in time. Therefore, we recommended fetal karyotyping to our case. When observed as isolated, it may occur frequently as a result of the rupture of an abdominal mass or after rupture in cases such as intestinal obstruction, posterior urethral valve and cloacal persistence. In many studies made on non-immune fetal acid, it was seen that isolated fetal acid has a better prognosis compared to the cases with concomitant hydrops.^[3] Isolated fetal acid may result with polyhydramnios and hydrops with pressure to inferior vena cava and other abdominal organs. In our case, fetal acid regressed within 2 weeks and did not cause any complication.

Hormonal stimulation is considered to be responsible in the etiology for the development fetal ovarian cysts (fetal gonadotropins, maternal estrogen and placental human chorionic gonadotropin). The incidence of ovarian cysts is considered above 30% (this rate is calculated according to the autopsies of babies which born dead and died within 28 days after delivery).^[4] They are usually isolated and seen more frequently depending on the placental hcG production increased possibly in cases such as maternal diabetes, hypothyroidism, toxemia, or Rh isoimmunization.^[5] Mesenteric cyst, urachal cyst, ectopic hydronephretic kidney, intestinal duplication anomalies,



Fig. 2. Diffusive free fluid in fetal abdomen.

cystic teratoma and intestinal obstruction should also be kept in mind in the differential diagnosis.^[6] We found no additional anomaly in our case with a detailed ultrasonographic screening. Since the fetus was female and the cystic structure was of pelvic origin, we first focused on ovaries. The shape and anechoic structure of the cyst and lack of bleeding confirmed our diagnosis. Ovarian cysts are diagnosed as a result of pelvic cystic formations found mostly during routine obstetric examination. When it is suspected of a possible fetal ovarian cyst, structural changes (dimension, appearance) of cystic formation or complications (hydramnios, acid, torsion) should be



Fig. 3. Regressed cystic formation in fetal ovary.

checked by serial ultrasonography. Simple ovarian cysts have thin walls and do not include internal echogenities.^[7] They are usually unilateral. Anechoic simple cysts may become a complex condition including internal echogenities and fluid levels. Detection of these ultrasonographic changes frequently indicates torsion. The incidence of torsion, which is the most common complication of fetal ovarian cysts, is 40%. Although torsion generally develops in intense and bigger cysts, it may also develop in cysts with 2 cm diameter.^[8] We followed up our case due to the appearance and dimension of the cystic structure. Fifty percent of them disappear spontaneously after birth.^[9] Other rare complications of fetal ovarian cysts reported in the literature are gastrointestinal obstruction, polyhydramnios and ovarian autoamputation.^[10] In many different studies, the criteria for intrauterine decompression of ovarian cysts were reported as being bigger than 4 cm and rapid development.^[11] Although it is expected that the cysts smaller than 2 cm regressed highly and do not cause complication, we observed in our case a temporary acid condition developed due to the rupture of a simple cyst with 15 mm diameter. It was an unexpected condition to develop due to a cyst with such dimension; however, changes in the cystic structure and dimension together with acid, being temporary and complete recovery within 2 weeks made us to focus on ovarian cyst in the etiology.

As in adults, when ovarian cyst ruptures, this ovary may develop hemorrhage. When rupture develops, cystic formation undergoes change in structure and dimension, and hemorrhage to the abdominal cavity causes ultrasonographic acid appearance. In our case, we observed structural and dimensional change of cystic formation in the examination performed two weeks later. With this observation, we associated free intraabdominal fluid with ruptured ovarian cyst. We found that fetal acid regressed two weeks later.

Conclusion

The rupture of fetal ovarian cyst should be evaluated as an etiological factor in isolated fetal acid cases. It is

hard to evaluate fetal ovaries by ultrasonography, and it is generally not possible to distinguish in other pelvic structures. However, when a cystic formation is found especially in female fetuses, fetal ovaries also should be kept in mind.

Conflicts of Interest: No conflicts declared.

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