



An Unusual Cause of Transient Fetal Ascites: Fetal Ovarian Cyst Rupture

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Abstract

Background: Ovarian cysts can be diagnosed prenatally and in case of persistence are found in newborns. Most resolve spontaneously, although in a small proportion of cases they can persist and be symptomatic. Fetal ascites may be due to immune or non immune hydrops fetalis or to a series of locoregional problems (bowel perforation, ovarian cyst rupture, uroperitoneum, etc...) However, it can be an isolated finding rarely.

Case: In this case we reported an isolated fetal ascites case that was caused by fetal ovarian cyst rupture. Conclusion: Fetal hydrops may be expression of a systemic or abdominal locoregional pathological process. We mentioned that fetal ovarian cyst rupture could be a cause of isolated fetal ascites with report of a case.

Keywords

Fetal ascites, Fetal ovarian Cyst, Fetal ovarian cysts rupture.

Introduction

Fetal ascites can be recognized as a symptom of fetal hydrops and can develop in response to numerous etiologic factors. Recently, due to frequent usage of Rh immunglobulin, non-immune hydrops is more frequent than immune hydrops fetalis in contrast to past. Fetal ascites can also occur isolated, independent from other serousal cavities and subcutaneous tissue due to various congenital abnormalities. Etiology of primary isolated fetal ascites is unclear. Most of them progresses to hydrops fetalis and some of them resolves spontaneously. In female newborns, ovarian cysts are frequently detected abdominal masses. Usually they are not symptomatic and resolve spontaneously. According to the common use of antenatal ultrasonography, we are now diagnosing ovarian cysts more common both in utero and in the female newborn. However, they are rarely symptomatic and they have no clinical significance. In this presentation is wereported an unexpected etiologic factor of fetal ascites; fetal ovarian cyst rupture.

Case Report

A 34-year-old woman, gravity 2, parity 0, abortion 1 is referred to our clinic at 32 weeks of gestation because of fetal intraabdominal cystic formation. She had an unremarkable prenatal course until this week. In history, she had no clinical significant coincident disease.

We learned that she attended all prenatal visits regularly and he had no pathological finding in all previous visits. In the 32 weeks ultrasonography fetal biometric measurements were compatible with gestational age, amniotic fluid volume was in normal limits. Maternal blood pressure was 110/70 mmHg and fetal movements were well. In ultrasonography, we detected a 15mm spheric cystic formation on the left lower side of fetal abdomen (Figure 1). The cystic formation was not related with renal structures and bladder. In Doppler sonography, we did not determine intracystic blood supply. Bilateral renal structures were evaluated normal, fetal intestines were observed normal. Because the fetus is female, cystic formation was anechoic and fetus had no accompanying genitourinary or gastrointestinal abnormalities we primarily suggested on an ovarian cyst. Due to existing fetal ovarian cyst, patient was evaluated about maternal diabetes and hypothyroidism again and resulted both in normal limits. Patient was called for a new visit two weeks later. In the 34 th week, ultrasonography fetal growth was compatible with 34 weeks gestation and amniotic fluid was in normal limits again. Only abdominal circumference was measured as 99% for

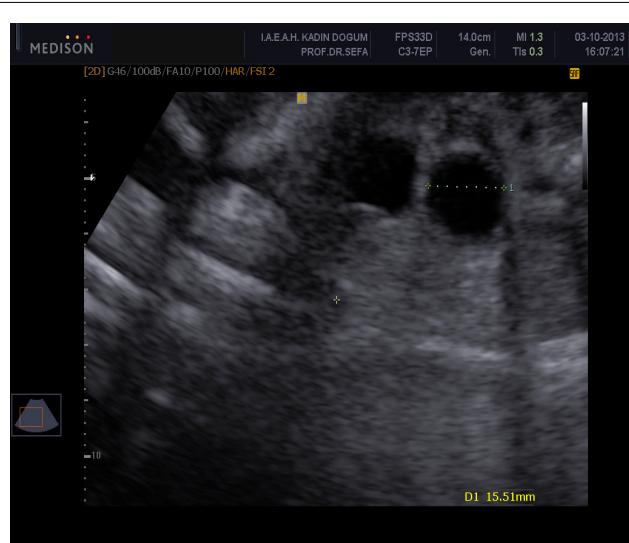


Figure 1: 15mm spheric cystic formation on the left fetal ovary.

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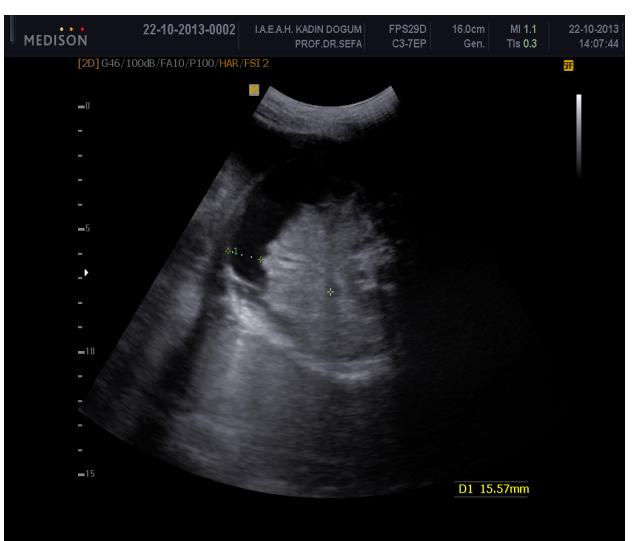


Figure 2: Widespread fluid accumulation in fetal abdomen.



Figure 3: 14mm regressed ovarian cyst on the left fetal ovary.

this week because of fetal intraabdominal fluid. In fetal abdomen, a widespread fluid accumulation was observed (Figure 2). Ovarian cyst on the left fetal ovary was regressed and was measured 14 mm with an ellipsoid shape (Figure 3). Intestines were floating in this fluid. Fetal thorax was normal and hydrothorax was not determined. We did not observe edema in subcutaneous tissue and we evaluated as primary fetal ascites even so we performed an assessment about hydrops fetalis. Hemogram, HbA1c, VDRL, TORCH screening, Parvovirus B-19 screening, indirect coombs test and anticardiolipin IgM and IgG studies were performed. Middle cerebral artery peak systolic velocity was 55cm/sec (1,12 MOM) and was not signifying fetal anemia. We recommended fetal karyotyping to patient because it could be an onset of fetal hydrops. However, patient did not accept karyotyping. All screenings were evaluated normal. Fetal cardiac examination was normal. Patient was called for a visit two weeks later again. At the next examination in the 36th weeks of pregnancy, fetal ultrasonography was completely normal. We have not observed any fetal ovarian cyst and fluid in fetal abdomen was completely regressed. Patient had a 3400 gr healthy baby on the 40th weeks of her pregnancy. No abnormal finding determined in postnatal abdominal ultrasonography.

Discussion

Hydrops Fetalis is classically defined as pathologic fluid accumulation in soft tissues and in at least two fetal serous

cavities including skin and placental edema, pericardial and pleural effusion and ascites with or without evidence of isoimmunization [1]. It is classified as immune and non-immune hydrops fetalis. Isolated fetal hydrothorax or ascites is a condition characterized by accumulation of pleural effusion or ascitic fluid, without generalized skin edema, due to various etiologies [2]. Fetal ascites is generally a part of the symptoms in cases with non-immune fetal hydrops, which refers to fluid collection in at least two body cavities or fluid collection in one cavity plus diffuse subcutaneous edema. The etiology of non-immune fetal hydrops includes numerous causes such as chromosomal anomalies, intrauterine infections, cardiac failure, and structural anomalies of various organs. Fetal ascites sometimes occur independently without fluid accumulation in any other serous cavities or subcutaneous tissue. It has been defined as primary isolated ascites. Primary isolated ascites can be a sign of fetal hydrops at early stage and may progress to hydrops consecutively. We suggested karyotyping to the patient because ascites could be an onset for hydrops. It may be isolated and usually as a result of abnormalities of abdominal structures like posterior urethral valve or cloacal persistence, bowel obstruction if they rupture or rupture of any abdominal mass. In a large series study focusing on non-immune fetal ascites, it was reported that, the fetal ascites accompanied by fetal hydrops had more unfavorable prognosis in comparison with isolated fetal ascites without an association of hydrops [3]. Isolated hydrops can progress and cause hydrops and polyhydramnios by compression to inferior vena cava and other abdominal organs. In our case ascites resolved spontaneously in two weeks and did not cause any complication.

Hormonal stimulation is generally considered responsible for the etiology of fetal ovarian cysts (fetal gonadotropins, maternal estrogen and placental human chorionic gonadotropin). The incidence of ovarian cysts has been estimated at more than 30 % (this estimate is based on an investigation of still born or infants who died within 28 days of birth) [4]. They are often isolated and associated with pregnancies complicated by maternal diabetes, toxemia or Rh isoimmunization, probably because of the increased placental production of hCG in these conditions [5]. Especially mesenteric cyst, urachus cyst, ectopic hydronephrotic kidney, intestinal duplication anomalies, cystic teratoma and intestinal obstruction should be kept in mind in differential diagnosis [6]. After a complete ultrasound examination, we did not detect any other anomaly in other structures. Because fetus was female and cyst was pelvic localized, we condensed on ovaries. Formation of the cystic mass was anechoic and was not vascularized, these were supporting our diagnosis. Ovarian cysts are usually diagnosed in a routine obstetrical ultrasonography with detection of a fetal cystic pelvic mass. Once the prenatal diagnosis of a probable ovarian cyst has been made, it is most important to perform serial ultrasound examinations to detect any structural change (size, appearance) in the mass or complications (hydramnios, ascites, torsion). A simple ovarian cyst has smooth borders and does not have internal echogenic structures [7]. Most of them are unilateral. Anechoic simple cysts can become complex masses, featuring echogenicity, fluid levels, and internal echoes. The presence of these ultrasonographic elements is usually considered a symptom of torsion. Prenatal torsion, the most frequent complication of prenatal ovarian cysts, has an about 40% incidence. The torsion occurs more frequently in voluminous cysts, but it also occurs in cysts sized 2cm [8]. Some cysts remain anechoic inside the uterus. In our case due to size and ultrasonographic appearance we decided to follow patient. A relevant number of them, about 50%, resolve spontaneously after birth [9].

Like adults when an ovarian cystic formation ruptures, hemorrhage can result from that ovary. When rupture is actualized, ruptured cystic formation changes in size and shape, abdominal bleeding causes ultrasonographic ascites image.

In our case two weeks later we detected the cystic formation changed in size and shape. With this information we related free fluid with ruptured ovarian cyst. In accordance with this, two weeks later fetal ascites regressed spontaneously.

In conclusion, fetal ovarian cyst rupture should be evaluated as an etiological factor in isolated fetal ascites cases. Evaluation of fetal ovaries is difficult and generally ovaries can not be differentiated from other pelvic structures. However, when an abdominal detected cystic formation is presented, fetal ovaries should be kept in mind.

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