

Spontaneous Resolution of Fetal and Neonatal Ascites after Birth

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الشفاء التلقائي من الاستسقاء في الأجنة و المواليد بعد الولادة

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الملخص: إن استسقاء الجنين هو مرض غير مألوف ينتج عن عدة أسباب منها غير المناعية. حيث أن عدد وفيات الأجنة والمواليد مرتفعة، لاسيما عندما يتطور الاستسقاء قبل الأسبوع 24 من الحمل. إن التناقص من شدة استسقاء الجنين دون تدخل علاجي عند المواليد أمر غير معروف. نعرض هنا حالة استسقاء الجنين المعزولة والتي اكتشفت في الأسبوع العشرين للحمل. كانت جميع الفحوصات التي أجريت طبيعياً ولكن أظهرت الفحوصات المتتالية بالموجات الصوتية عن وجود استسقاء الجنين عند الأسبوع 20 من الحمل. كما أن متابعة الفحوصات بالموجات فوق الصوتية للطفل عند عمر 6 أشهر بعد الولادة أظهرت شفاء كامل من الاستسقاء. إن الشفاء التلقائي من استسقاء الجنين ذي التنبؤ الجيد يمكن أن يحدث في الحالات مجهولة السبب.

مفتاح الكلمات: استسقاء الجنين، الشفاء التلقائي، تنبؤ، تقرير حالة، عمان.

ABSTRACT: Fetal ascites is an uncommon abnormality usually reported in relation to non-immunological causes. The prospect for fetal and neonatal mortality is high, particularly when the ascites develops before 24 weeks of gestation. The diminution of severe fetal ascites without intrauterine management, especially with an uncomplicated neonatal outcome, is unusual. We report a case of isolated fetal ascites detected at 20 weeks' gestation. All investigations carried out were normal. Consecutive ultrasound examination showed ascites at 20 weeks' gestation. A follow-up ultrasound examination at 6 months of age revealed complete recovery from the ascites. Spontaneous resolution of fetal ascites, with a good prognosis, can occur in cases with an idiopathic aetiology.

Keywords: Fetal ascites; Spontaneous resolution; Prognosis; Case report; Oman.

FETAL ASCITES COMMONLY OCCURS linked to fetal hydrops. After the recognition of ascites in antenatal ultrasound, it is essential to establish whether this is an isolated fetal ascites or associated with hydrops.¹ Isolated fetal ascites is defined as "ascites not associated with fetal hydrops".² It is an uncommon condition and mainly occurs as an early manifestation of *hydrops fetalis*. Isolated ascites is commonly caused by intra-abdominal disorders due to urinary tract obstruction. Around 20% of cases occur as a result of gastrointestinal tract disorders.^{1,3-5} Intestinal obstruction resulting in meconium peritonitis is considered to be one of the commonest gastrointestinal disorders associated with isolated ascites.^{3,6}

Case Report

A 37-year-old Omani (gravida 3, para 0) woman presented with isolated fetal ascites diagnosed at 20 weeks' gestation. Fetal parameters and amniotic fluid volume were normal according to ultrasound examination. There was no evidence of *hydrops fetalis* or any other abnormality, particularly in the urinary and gastrointestinal systems. Ultrasonographic examination revealed no cardiomegaly or placental enlargement to indicate fetal anaemia. The findings upon medical examination included the discovery that the mother was blood group O and Rh positive with a normal complete blood picture, a negative venereal disease research laboratory (VDRL) test, and a negative finding for parvovirus antibodies, cytomegalovirus, and toxoplasmosis. Amniocentesis showed a normal female fetal karyotype.

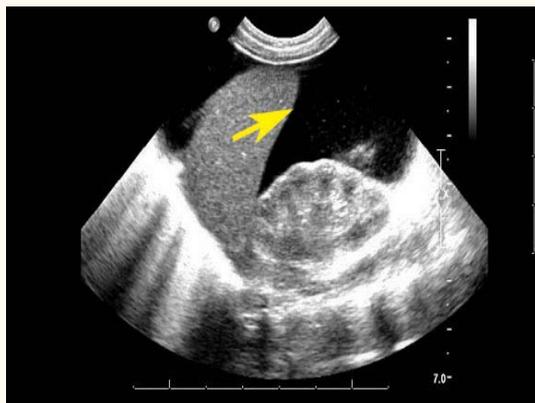


Figure 1: Moderate ascites.

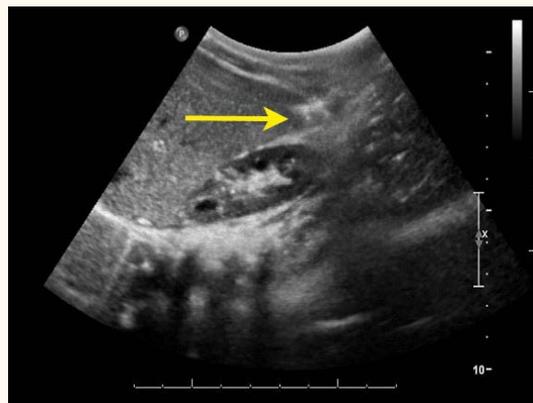


Figure 2: Complete resolution of ascites after 6 months.

Spontaneous vaginal delivery occurred at 38 weeks' gestation. A female infant weighing 3,390 grams was delivered with Apgar scores of 9 and 9 at 1 and 5 minutes, respectively. There was no abdominal dystocia during delivery. Systemic examination was normal with no evidence of dysmorphic features. The infant did not require respiratory support and oxygen saturation was 100% at room air. Ultrasonography after birth revealed moderate ascites [Figure 1]. Other radiological investigations included an antero-posterior plain X-ray view of the abdomen and a barium enema. The follow-through of the gastrointestinal tract was normal. Following abdominal paracentesis, 150 ml of clear, yellow, sterile fluid was obtained. Ascitic fluid showed white blood cells ($50 \times 10^6/L$), red blood cells ($10 \times 10^6/L$), albumin (27 g/L), and glucose (6 mmol/L). The initial serum albumin was 33 g/L. Serum-ascites albumin gradient (SAAG) is frequently used to find out the cause of ascites and to discriminate between transudate and exudate. In this case it was 6 g/L, indicating portal hypertension *versus* non-portal hypertension aetiology for the ascites in this patient. Blood count, serum electrolytes, liver function tests, and serum triglycerides and lactate dehydrogenase were within normal limits. The ascites progressively resolved over a two-week period. Oral feeding with normal infant formula was instituted and tolerated. The patient was discharged home in good condition. A follow-up after 6 months revealed normal growth and development. No recurrent ascites could be detected by abdominal sonography [Figure 2].

Discussion

The aetiology of isolated fetal ascites can be idiopathic or may occur as a result of many conditions, including fetomaternal haemorrhage, glucose-6-phosphate dehydrogenase deficiency, and thalassaemia affecting the mother. In the fetus, chromosomal abnormalities tend to occur mostly due to congenital heart disease, congenital infections, hepatic and metabolic storage disorders, and lymphatic disorders of the peritoneum.^{1,2,9} It is essential to differentiate hydrops from fetal ascites, as fetal hydrops is more commonly caused by systemic diseases, whereas the latter occurs more frequently due to local intra-abdominal causes. Despite the fact that hydrops is usually considered a serious condition, fetal ascites is not necessarily considered thus.⁷⁻⁹

Isolated fetal ascites presents antenatally with fluid around the "spleen, liver, bowel, bladder, extrahepatic portion of the umbilical vein, falciform ligament, and/or greater omentum", usually discovered by ultrasonography.¹ Other features of hydrops, including skin oedema, and pleural and pericardial effusion, are not present.

After the diagnosis of fetal ascites, a follow-up ultrasound after one week is required to establish if there has been progression to fetal hydrops. The development of hydrops is not likely to occur if the ascites remains localised to the abdominal cavity.^{1,7}

Possible fatal complications of isolated fetal ascites include the development of lung hypoplasia and hydrops.^{9,10} Pulmonary hypoplasia leading to respiratory distress following birth can develop as a result of the ascites moving the diaphragm upwards, thereby compressing the lungs.¹¹ Seeds *et al.* were

the first ones to report *in utero* abdomino-amniotic shunting as useful in managing fetal ascites. Nevertheless, the procedure is not a prerequisite in cases of simple isolated ascites, as such an intervention might predispose a fetus to preterm delivery.¹² Abdominal paracentesis performed prenatally has been recommended as helpful in improving the outcome of pulmonary function and preventing abdominal dystocia if done prior to a vaginal delivery.^{13,14} On the other hand, the ascitic fluid generally reaccumulates quickly following the procedure. Seeds and Fung *et al.* recommended abdominoperitoneal shunting to avoid recurrent paracentesis.^{15,16}

Occasionally, polyhydramnios and fetal ascites can occur together. The mechanism of the development of polyhydramnios in such cases is still not clear. In our case, there was no evidence of polyhydramnios in the mother, and the baby did not require any respiratory support after birth.

The outcome and prognosis of isolated fetal ascites is determined by the primary cause, given that a good prognosis has been documented in affected newborns with idiopathic fetal ascites.^{4,5}

Earlier reports have analysed the wide range of diseases that can present as isolated fetal ascites.^{8,13} As reported by El Bishry, in a series of 12 patients with isolated fetal ascites, 10 survived after delivery out of whom 9 had no other anomalies detected on antenatal or postnatal ultrasound. Only one of the 10 cases had ileal atresia detected postnatally which was surgically corrected. Two cases diagnosed before 20 weeks' gestation died. One of them was found to have laryngeal atresia, which was life-threatening.⁸ In another report by Favre *et al.*, a 100% survival rate was reported in 8 patients with idiopathic isolated ascites and no abnormalities were detected.¹³ Furthermore, a patient's prognosis depends on an antenatal diagnosis of dystocia. Fetal demise has been documented in cases that were not predicted during antenatal follow up.^{11–13}

Satoko *et al.* reported that gestational age is inversely correlated with the severity of the ascites at diagnosis and carries a major risk factor for prognosis.¹⁷ Nevertheless, the outcome of fetal ascites in this case report was favourable in spite of an early antenatal diagnosis.

The work-up to determine the aetiology of the fetal ascites in this patient was negative; however, the serum ascites albumin gradient (SAAG) was less

than 11 g/L, indicating a non-portal hypertension aetiology. There was no evidence of infectious, malignant, or inflammatory peritoneal disease. Although we were not able to identify a cause for the ascites, in a large proportion of cases the cause was never determined, even with wide-ranging investigations.⁸

Conclusion

This patient was diagnosed with isolated fetal and neonatal ascites without other related abnormalities, which is an entity separate from *hydrops fetalis*. The patient had a favourable perinatal outcome.

References

1. Ulreich S, Gruslin A, Nodell CG, Pretorius HD. Fetal hydrops and ascites. In: Nyberg DA, McGahan JP, Pretorius DH, Pulu G, Eds. *Diagnostic Imaging of Fetal Anomalies*. New York: Lippincott Williams & Wilkins, 2003. Pp. 713–45.
2. Winn HN, Stiller R, Grannum PA, Crane JC, Coster B, Romero R. Isolated fetal ascites: Prenatal diagnosis and management. *Am J Perinatol* 1990; 7:370–3.
3. Agrawala G, Predanic M, Perni SC, Chasen ST. Isolated fetal ascites caused by bowel perforation due to colonic atresia. *J Matern Fetal Neonatal Med* 2005; 17:291–4.
4. Ohno Y, Koyama N, Tsuda M, Ariei Y. Antenatal ultrasound appearance of cloacal anomaly. *Obstet Gynecol* 2000; 95:1013–15.
5. Persutte WH, Lenke RL, Kropp KA. Atypical presentation of fetal obstructive uropathy. *J Diagn Med Sonogr* 1989; 1:12–15.
6. Chen FY, Chen M, Shih JC, Tsao PN, Lee CN, Hsieh FJ. Meconium peritonitis presenting as a massive fetal ascites. *Prenat Diagn* 2004; 24:930–1.
7. Arikan Ilker, Barut A, Harma M, Harma M, Dogan S. Isolated fetal ascites. A case report. *J Med Case Rep* 2012; 3:110–12.
8. El Bishry G. The outcome of isolated fetal ascites. *Eur J Obstet Gynecol Reprod Biol* 2008; 137:43–6.
9. Stocker JT. Congenital cytomegalovirus infection presenting as massive ascites with secondary pulmonary hypoplasia. *Hum Pathol* 1985; 16:1173–5.
10. Bernaschek G, Deutinger J, Hansmann M, Bald R, Holzgreve W, Bollmann R. Feto-amniotic shunting—report of the experience of four European centres. *Prenatal Diagn* 1994; 14:821–33.
11. Ng HT, Chang SP, Ho SC. Dystocia due to fetal ascites. *Mod Med Asia* 1976; 12:10.

12. Cederqvist LL, Williams LR, Symchych RS, Sarry ZI. Prenatal diagnosis of fetal ascites by ultrasound. *Am J Obstet Gynecol* 1977; 15:229–30.
13. Favre R, Dreux S, Dommergues M, Dumez Y, Luton D, Oury JF, et al. Nonimmune fetal ascites: A series of 79 cases. *Am J Obstet Gynecol* 2004; 190:407–12.
14. de Crespigny LC, Robinson HP, McBain JC. Fetal abdominal paracentesis in the management of gross fetal ascites. *Aust NZ J Obstet Gynaecol* 1980; 20:228–30.
15. Seeds JW, Herbert WN, Bowes WA Jr, Cefalo RC. Recurrent idiopathic fetal hydrops: Results of prenatal therapy. *Obstet Gynecol* 1984; 64:S30–33.
16. Fung HY, Lau TK, Chang AM. Abdomino-amniotic shunting in isolated fetal ascites with polyhydramnios. *Acta Obstet Gynecol Scand* 1997; 76:706–7.
17. Nose S, Usui N, Soh H, Kamiyama M, Tani G, Kanagawa T, et al. The prognostic factors and the outcome of primary isolated fetal ascites. *Pediatr Surg Int* 2011; 27:799–804.