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. Isolated fetal ascites is defined as “ascites not associated with fetal hydrops.” 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. Intestinal obstruction resulting in meconium peritonitis is considered to be one of the commonest gastrointestinal disorders associated with isolated ascites.
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 anterio-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 x 10^6/L), red blood cells (10 x 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. 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.

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. 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.

Possible fatal complications of isolated fetal ascites include the development of lung hypoplasia and hydrops. Pulmonary hypoplasia leading to respiratory distress following birth can develop as a result of the ascites moving the diaphragm upwards, thereby compressing the lungs. 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. 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. 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.

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.

Earlier reports have analysed the wide range of diseases that can present as isolated fetal ascites. 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. 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. 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.

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. 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.

**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


