Neonatal Haemorrhagic Ascites: A Rare Case Report

Dr. Snehal Jagtap¹, Dr. Abhijeet Shinde², Dr. Suresh Waydande³, Prof. Dr. Sunil Natha Mhaske⁴, Dr. Shushrut Kumar²

¹Junior Resident, ²Assistant Professor ³Professor & Head, Department of Paediatrics, DVVPFs Medical College & Hospital, Ahmednagar-414111, Maharashtra, India
⁴Dean & Professor Paediatrics, DVVPFs Medical College & Hospital, Ahmednagar-414111, Maharashtra, India

Abstract:
Haemorrhagic ascites in the newborn is rare. Ascites is the pathological accumulation of fluid within the peritoneal cavity. Various causes of ascites have been described in different age groups. Abdominal paracentesis does provide symptomatic relief and can be used as a diagnostic tool to find the cause of ascites. Here we report one such case of haemorrhagic neonatal ascites in a 37-week neonate, where therapeutic paracentesis was done for the gross ascites. Close follow-up is recommended for signs of frozen abdomen and intestinal obstruction.

Key words: Haemorrhagic Ascites, Puddle sign, Abdominal Paracentesis.

Introduction:
Haemorrhagic ascites in a newborn is a rare condition. Accumulated ascites might be transudate, exudate, chylous, biliary, pancreatic, haemorrhagic, associated with specific infections[1] or extravasated urine.[2] Ascites results from incompatibility of blood groups of mother and foetus, cardiac, hepatic, renal, pancreatic, biliary, ovarian, intestinal disease, trauma, malignancy and ruptured mesenchymal hamartoma.[1,3] The diagnosis is challenging and management is directed at the cause.[4] Invasive procedures and surgery is advised in cases with ruptured viscus. Haemorrhagic ascites in the newborn is rare and complicates trauma during birth, e.g. ruptured spleen or liver, ruptured omental venous malformations,[5] ruptured hemangioma, or mesenchymal cyst, midgut volvulus and bleeding disorders.[6] Serious complications of haemorrhagic ascites includes peritoneal adhesions and frozen abdomen.[7]

Ascites is the pathological accumulation of fluid within the peritoneal cavity. Various causes of ascites have been described in different age groups. In children, hepatic and renal diseases are the most common causes, but ascites can also be caused by cardiac disease, trauma, infection or neoplasia.[7] The clinical hallmark of ascites is abdominal distention. Early satiety and dyspnoea can occur with a moderate amount of ascites. A considerable amount of intraperitoneal fluid should accumulate before ascites is detectable by the classic physical signs like bulging of flanks, dull percussion note, shifting dullness, a fluid thrill, and puddle sign (percussion of a supine person's abdomen over the umbilicus becomes dull as the patient is moved to prone position and ascitic fluid puddles in dependant regions). Umbilical herniation can also be associated with tense ascites.[7]

Abdominal paracentesis does provide symptomatic relief and can be used as a diagnostic tool to find the cause of ascites. Determining the serum-ascites albumin gradient can help determine the cause of the ascites. Ascitic gradient greater than 1.1g/dl (high ascitic gradient) concludes ascites caused by portal hypertension, whereas ascitic gradient less than1.1g/dl (low gradient ascites) means ascites of non-portal hypertensive etiology.[7] For most patients, treatment consists of dietary sodium restriction and diuretic therapy with spironolactone, with the addition of furosemide in most severe cases. Supplemental albumin can also aid in ascitic fluid mobilization. Refractory cases may require large volume paracentesis or trans-jugular intrahepatic portosystemic shunting. Patients with any type of ascites are at increased risk for spontaneous bacterial peritonitis.[7]
Case report:
A late preterm baby born to a G2P1L1 mother via Lower Segment Cesarean Section in view of previous LSCS and pre-eclampsia in current pregnancy with birth weight of 1.74 kgs. The baby had subcostal retractions and intercostal retraction with nasal flaring soon after birth, with Silvermann Anderson score of 2/10 and so the baby was kept on O2 by nasal prongs at 2L/min. The patient was then started on trophic feeds but was then discontinued due to brownish aspirates in the oro-gastric tube and distension of abdomen. Baby was kept NBM and cold saline wash was given twice a day in view of brownish aspirates. X-ray abdomen in erect posture and Ultrasound abdomen was suggestive of necrotising enterocolitis like changes. The baby was started on ionotrope supports due to poor heart rate. In view of thrombocytopenia, positive C - reactive protein and blood culture suggestive of fungal growth, patient started on intravenous caspofungin along with appropriate parenteral antibiotics. Patient also received two blood transfusions i/v/o low haemoglobin level.
As abdomen distension increased (Fig. 1) hence repeat x-ray abdomen in erect posture (Fig 2) and abdominal ultrasound was done, findings on both suggestive of gross ascites. Contrast Enhanced Computed Tomography (CECT) abdomen was also done which shows mild hepatomegaly with oedematous gall bladder wall and significant ascites with diffuse mesenteric congestion. Therapeutic ultrasound guided ascitic tapping was done after bleeding tendencies were ruled out. The ascitic fluid was non-clotted dark chocolate and 110ml of fluid was drained after a single trial. (Fig 3) Neonate was taken off O2 after 5 days and enteral feeding was started after stabilization and reached to full oral intake with no increase in abdominal girth and no increase in abdominal peritoneal fluid collection on repeat ultrasound. The neonate was discharged after 40 days in good general condition on breastfeeding with instruction about any signs of intestinal obstruction. On regular follow up visit's patient were assessed for physical growth, abdominal circumference, haemoglobin and abdominal ultrasonography. At 5 months of age her blood investigations were within normal parameters and abdominal ultrasound showed no fluid in the abdomen.

Discussion:
Clinical examination, laboratory investigations, imaging including ultrasound, CECT and MRI, diagnostic as well as therapeutic tapping, are the initial steps for diagnosis of the underlying cause of ascites in neonates. At times the diagnosis will be straightforward like in chylous ascites, as it is associated with congenital lymphatic malformation, thoracic duct rupture as well as lymphatic obstruction. It has characteristic milky appearance with triglyceride level more than 200mg% and protein more than 2g%. In our case neonate had haemorrhagic ascites which was completely resolved after conservative management.
Here in this case all possible causes were ruled out and cause was taken to be as sepsis, hence after the sepsis was under control, no repeated ascites was observed in the baby. After exclusion of ongoing bleeding, any surgical emergencies and abdominal masses with laboratory investigations and imaging, surgical exploration to identify an underlying cause was ruled out. Complications such as peritoneal infections and intestinal adhesions were not present.

All the investigations which were done did not detect the cause of haemorrhagic ascites, e.g. a large cystic mass with multiple septa, ruptured cystic mesenchymal hamartoma, or necrotizing enterocolitis with complications, any iatrogenic injury during umbilical catheterization or very rarely birth trauma, wherein the previously mentioned causes are acquired after birth.[8] Ascitic tapping was done as and 110 ml of haemorrhagic ascitic fluid was removed. The respiratory distress which the baby had due to abdominal distension was relieved.

There are various theories that have been proposed for the formation of ascites[10], which include the following:

A. Under filling theory: Portal hypertension causes decreased sequestration of fluid in the splanchnic circulation that causes hypotension and activates the renin angiotensinogen aldosterone and sympathetic systems resulting in renal sodium and water retention.

B. Overflow theory: There are renal sodium and water retention in the absence of volume depletion.

C. Peripheral arterial vasodilation: Due to portal hypertension underfilling there is renal sodium and water retention. Overfilling theory has a role in the pathogenesis of cirrhosis.

Presenting features include abdominal distension, increasing weight, respiratory distress, associated pedal oedema. Ascites that is associated with malignancies is usually painful while cirrhotic patients have painless ascites.[11] Ascites should always be differentiated from other causes of abdominal distension like gaseous distension, any bowel obstruction, obesity, or abdominal mass. The examination done should also focus on signs of any chronic liver disease as well as portal hypertension. Flank dullness is present in about 90% of the patients and is one the most sensitive physical sign of ascites.[11]

Clinical grading of ascites:[12]

<table>
<thead>
<tr>
<th>MILD</th>
<th>MODERATE</th>
<th>SEVERE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Puddle sign +</td>
<td>Shifting Dullness + No Fluid Thrill</td>
<td>Fluid Thrill +</td>
</tr>
</tbody>
</table>

As per the modified criterion of the European Association for the Liver Study, ascites grades are as follows:[12]

Grade 1 | ascites is detected by only radiological tests such as abdominal ultrasound
Grade 2 | there is moderate abdominal distension which is detected on physical examination and/or by radiologic tests.
Grade 3 | there is gross ascites with marked distension of abdomen.

Conclusion:

Haemorrhagic ascites is a rare condition in newborns as it is life threatening that requires immediate management. Etiology is not known in some of the cases, but in this case all other possible causes were ruled out and sepsis was considered to be the cause of haemorrhagic ascites in this newborn. The baby was managed conservatively in this case. Regular follow-up is advised to look for anemia, intestinal obstruction, and repeated ascites.

References:


