

CASE REPORTS

NEONATAL ASCITES - A CASE SERIES

Saugat Ghosh, Kheya Ghosh Uttam, Priti Khemka, Rafikul Islam, Shabana Kabir.

Institute of Child Health, Kolkata, India.

ABSTRACT

Neonatal ascites is a rare condition characterized by the accumulation of fluid in the peritoneal cavity of a newborn. It is caused by a number of etiologies, the urogenital anomalies are most common cause of neonatal ascites. GIT causes and TORCH infections are the next common causes. The management and prognosis depends on the etiology. There is scarcity of literature in this subject. About 5 cases of neonatal ascites, admitted in last 3 years in our institute, was retrospectively studied. The 5 cases include 2 chylous ascites, 1 urinary ascites, 1 ascites due to liver failure and galactosemia and 1 meconium peritonitis.

Introduction

Neonatal ascites is a rare condition characterized by the accumulation of fluid in the peritoneal cavity of a newborn. It is caused by a number of etiologies which include diseases of genitourinary system, gastrointestinal system, cardiac disease, hepatic disease, TORCH or parvovirus infection, chylous, ovarian cause, inborn error of metabolism and idiopathic.¹ The urogenital anomalies are most common cause of neonatal ascites. GIT causes and TORCH infection are the next common causes.² Neonatal ascites is a rare problem and there is scarcity of literature in this subject,⁵ cases of neonatal ascites, admitted in last 3 years in our institute, was retrospectively studied and is described below:

Case 1:

A term newborn, on exclusive breastfed had 1 episode of hypoglycaemic seizure on day 7 of life. He was treated in a local hospital and cause of hypoglycaemia was suspected due to poor feed intake. The baby presented later on day 28 of life with significant weight loss, conjugated jaundice and ascites. Diagnostic and therapeutic ascitic tap was done, cell count-25 cells/cu mm with protein 1.8 gm/dl. LFT and PT, APTT was severely deranged with INR>2 persistently. Urine reducing substance was positive so soya milk formula was started but no significant improvement. There was proteinuria and passage of urobilinogen and bilirubin in urine. After 5 days of admission baby developed sudden anuria so peritoneal dialysis was started but there was no significant improvement. USG KUB was suggestive of raised echogenicity of bilateral kidney. Blood culture was negative but there was *Acinetobacter* growth in BAL. Urine succinylacetone, CMV PCR, blood IEM screening, was negative and serum ferritin was mildly raised. Due to fulminant sepsis, uncorrected coagulopathy with multiorgan dysfunction, the baby expired after 1 month of hospital stay. Liver biopsy done posthumously because of deranged coagulation profile, suggestive of cholestasis with bridging fibrosis. Genetic work up was sent which revealed Galactosemia, variant c.562C>T, homozygous, location-exon 6.

Address for Correspondance: Saugat Ghosh,
Institute of Child Health, Biresh Guha Street, Park
Circus, Kolkata, 700017, India.

Email: ghoshsaugat6@gmail.com

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Figure 1. Galactosemia with ascitis, peritoneal dialysis catheter in situ.



Case 2:

A 9 days old late preterm, newborn admitted with abdomen distension since birth. USG abdomen was suggestive of ascitis. Ascitic tap revealed yellowish white fluid with cell count 3200/cu mm, 92% lymphocyte, triglyceride level was 397 mg/dl which was suggestive of chylous ascitis. Started on TPN followed by MCT powder feeds and ocreotide infusion. Blood culture revealed growth of *Klebsiella*. Abdomen distension progressively increased, so ascitic fluid drain was done repeatedly with albumin cover which lead to peritonitis. Due to uncontrolled sepsis baby also received immunoglobulin. Sepsis gradually got settled with appropriate antibiotics and general condition improved. Lymphoscintigraphy was planned but, could not be done. MRI abdomen done which revealed ascitis with no other significant abnormality. Exploratory laparotomy was planned to look for any lymphatic vessel abnormality but was cancelled due to dense adhesions. Baby was discharged after 1.5 months of NICU stay on MCT powder feeds in a haemodynamically stable condition.

Case 3:

A term male baby presented with abdomen distension since birth. Ultrasound was suggestive of ascites.

Ascitic fluid tap was done, appearance was milky with cell count 5500/cu mm and triglyceride was 1188 mg/dl which was suggestive of chylous ascites. Aggressive TPN support followed by ocreotide infusion and MCT powder feed was given. Baby was not having any symptomatic improvement. He was having persistent hypoalbuminemia inspite of albumin transfusion so received multiple albumin transfusion and IVIg also due to uncontrolled sepsis. Upper GI endoscopy was done which was suggestive of intestinal lymphangectasia. Gradually baby also developed chylous pleural effusion. The baby expired after 3 months of hospital stay.

Figure 2. Ascitic tap done in a chylous ascitis patient.



Case 4:

A 10 days old baby was admitted with abdomen distension with decreased urine output and dribbling of urine noticed since day 3 of life. USG abdomen was suggestive of bilateral gross hydronephrosis. Serum electrolytes and urea, creatinine was normal. MCUG was done which was suggestive of posterior urethral valve. PUV fulguration was done and overnight, baby developed abdomen distension and decreased urine output. USG whole abdomen was suggestive of ascites. In view of suspected urinary ascites, ascitic fluid creatinine was sent and it was raised 1.6 mg/dl while serum creatinine was 0.5 mg/dl. Peritoneal drainage and bilateral uterostomy was done on the next day. Post operatively baby developed kleibsella sepsis which was treated and discharged in a haemodynamically stable condition and advised for follow up.

Case 5:

A 33 weeks female newborn was admitted on day 1 with respiratory distress after birth with abdomen distension. Chest and abdomen X-ray was done, suggestive of lower end esophageal obstruction, ascites and 3 irregular opaque shadows in abdomen. 2D echo was done, there was no pericardial effusion. USG abdomen suggestive of meconium pseudocyst with calcification and ascites. Ascitic tap was done which was suggestive of peritonitis. Ascitic fluid cell count was 1320/cu mm with neutrophilic predominance with protein 3 gm/dl. Laparotomy was done which revealed ileal perforation 4 cm proximal to the ileocaecal junction

and dense adhesion, so ileostomy was done. Ascitic fluid culture sensitivity was negative but blood culture had grown MDR Kleibsella. Baby ultimately expired due to septicaemia.

Figure 3. Meconium peritonitisto.



Discussion

Though urinary ascites is the most commonly reported cause of ascites in newborn but out of above 5 cases, 2 of them had chylous ascites. Similar result was noted in a study done by Chaudhury et al³ which showed chylous ascites is the most common cause. About 50% of cases are caused due to malformation of lymphatic vessels and remaining may be caused due to external compression of the lymphatics, trauma, child abuse. Most of them has no etiology and is due to delay in maturation of the lacteals, called "leaky lymphatics".⁴ It is diagnosed by lymphocyte-rich, milky peritoneal fluid with triglycerides >110 mgr/dl. The imaging study of choice is lymphoscintigraphy but it is mostly avoided because of technical difficulties in a sick neonate. This condition is often refractory to treatment and causes severe EUGR, immunodeficiency and sepsis because of the loss of proteins and lymphocytes. IVIg can be given due to loss of immunoglobulins and sepsis.⁵ Treatment is mostly conservative, requires TPN, ocreotide infusion and MCT feeds.^{6,7} Mouravas et al reported that if any definite lesion like a chylous cyst is visible in ultrasound and MRI, then exploratory laparotomy can be done.⁸

Galactosemia, though can have progressive hepatic failure but ascites is rare. Lone KS et al reported 16.7% of early infantile liver failure due to galactosemia.⁹ Patients with galactosemia present with hypoglycaemia, jaundice, failure to thrive, lethargy, feeding difficulty, vomiting, diarrhea, sepsis, cataract and also hepatomegaly, liver failure and renal tubular dysfunction in severe cases. Patients who cannot be properly managed die due to liver failure, kidney failure or sepsis.¹⁰ Grama A et al reported a case of galactosemia with group B streptococcus infection and acute liver failure.¹¹ Ascites in galactosemia is rarely reported in past and develops 2-5 weeks after birth with continued galactose.¹² Renal failure in early infancy is a rare manifestation of galactosemia. Nesrin C et al reported a case of galactosemia which required peritoneal dialysis due to metabolic acidosis.¹³ Hepatorenal syndrome is defined as AKI in liver failure in absence of shock, nephrotoxic drugs, no proteinuria, microhematuria and normal kidney ultrasound.¹⁴ In our patient with

galactosemia, bilateral kidneys are hyperechoic and also he had significant proteinuria along with excretion of bile salts, urobilogen and bilirubin in urine which might have caused cholemic nephropathy which is diagnosed by kidney biopsy and has poor outcome.¹⁵

Meconium peritonitis is sterile chemical peritonitis due to intrauterine bowel perforation and meconium excretion into the peritoneal cavity seen as classic eggshell calcifications on X-ray. Incidence is 1 in 35 000 and has high mortality.¹⁶ A. Lamrissi studied that clinical features of MP are non-specific and may be asymptomatic also. Antenatal diagnosis by obstetrical ultrasound can be done. On examination there is abdominal distension and a mass, commonly in the right lower quadrant. Meconium peritonitis after birth results in bacterial sepsis with results in poor prognosis. Meconium peritonitis may be due to meconium ileus, intestinal stenosis or atresia etc.¹⁷ Ruptured ileum is the most common pathology.¹⁸ Cystic fibrosis is associated with 8-40% cases.¹⁹ Chiba T et al described two cases of ileal atresia with perforation and fetal ascites due to meconium peritonitis.²⁰ IV antibiotics, laparotomy, drainage, temporal enterostomy with delayed reconstruction of intestinal continuity is the management of choice.²¹

Neonatal urinary ascites is also a rare condition. It can be spontaneous or iatrogenic. It is a life-threatening condition as the peritoneal membrane "autodialyzes" the urine, leading to deranged electrolytes and kidney function test. Posterior urethral valve is the most common cause of spontaneous urinary ascites which occurs due to rupture of calyceal fornices or urinary bladder perforation.²² Yue He reported Neonatal Urinary Ascites due to Anterior Urethral Valve and Diverticulum in Preterm Newborn.²³ USG is the 1st line of investigation in urinary ascites. MCUG is used to detect the site of leak in the urinary.²⁴ Ascites Cr: serum Cr ratio of over 1 is diagnostic of urinary ascites.²⁵ Management consists of catheter drainage or surgery to divert the urine from the peritoneal cavity.²⁶

Compliance with Ethical Standards

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