lumen to prevent stent migration during peristalsis.

There is a concern in these patients regarding bile gastritis and meal induced cholangitis. In the absence of pancreatic secretion bile should not damage the gastric mucosa and the symptoms usually settle with proton pump inhibitors. Ramesh et al[15] reported surgical hepaticogastrostomy without bile gastritis or gastric acid induced cholangitis. The other potential complications are bile leak, bleeding, hemobilia and stent migration which did not occur in our case. To the best of our knowledge, this is the first report of percutaneous hepaticogastrostomy in a patient with portal biliopathy in whom surgical attempts at biliary decompression had failed and portosystemic shunting was not possible.

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References

1. Chandra R, Kapoor D, Tharakan A, Chaudhary A, Sarin SK. Portal biliopathy. *J Gastroenterol Hepatol*. 2001;**16**:1086–92.
2. Dhiman RK, Behera A, Chawla YK, Dilawari JB, Suri S. Portal hypertensive biliopathy. *Gut*. 2007;**56**:1001–8.
3. Mitra SK,Kumar V, Dutta DV, Rao PN, Sandhu K, Singh GK, Sodhi JS, Pathak IC. Extrahepatic portal vein hypertension: a review of 70 cases. *J Pediatr Surg*. 1978;**13**:51–7.
4. Dilawari JB, Chawla YK. Pseudosclerosing cholangitis in extrahepatic portal venous obstruction. *Gut*. 1992;**33**:272–6.
5. Sarin SK, Bhatia V, Makwane U. Portal biliopathy in extrahepatic portal vein obstruction. *Indian J Gastroenterol.* 1992;**11**:82.
6. Khuroo MS, Yatoo GN, Zargar SA, Javid G, Dar MY, Khan BA, et al. Biliary abnormalities associated with extrahepatic portal venous obstruction. *Hepatology*. 1993;**17**:807–13.
7. Chandra R, Tharakan A, Kapoor D, Sarin SK. Comparative study of portal biliopathy in patients with portal hypertension due to different etiologies. *Indian J Gastroenterol*. 1997;**15**:A59.
8. Condat B, Vilgrain V, Asselah T, O'Toole D, Rufat P, Zappa M, et al. Portal cavernoma-associated cholangiopathy: a clinical and MR cholangiography coupled with MR portography imaging study. *Hepatology*. 2003;**37**:1302–8.
9. Malkan GH, Bhatia SJ, Bashir K, Khemani R, Abraham P, Gandhi MS,et al,Cholangiopathy associated with portal hypertension: diagnostic evaluation and clinical implications. *Gastrointest Endosc*. 1999;**49**:344–8.
10. Agarwal AK, Sharma D, Singh S, Agarwal S, Girish SP. Portal biliopathy: a study of 39 surgically treated patients. *HPB(Oxford).* 2011;**13**:33–9.
11. Chaudhary A, Dhar P, Sarin SK, Sachdev A, Agarwal AK, Vij JC, et al. Bile duct obstruction due to portal biliopathy in extrahepatic portal hypertension: surgical management. *Br J Surg*. 1998;**85**:326–9.
12. Soulez G, Gagner M, Thérasse E, Deslandres E, Pomp A, Leduc R, et al. Malignant biliary obstruction: preliminary results of palliative treatment with hepaticogastrostomy under fluoroscopic, endoscopic, and laparoscopic guidance. *Radiology*. 1994;**192:**241–6.
13. Tipaldi L. A simplified percutaneous hepatogastric drainage technique for malignant biliary obstruction. *Cardiovasc Intervent Radiol*. 1995;**18**:333–6.
14. Park do H, Song TJ, Eum J, Moon SH, Lee SS, Seo DW, et al. EUS-guided hepaticogastrostomy with a fully covered metal stent as the biliary diversion technique for an occluded biliary metal stent after a failed ERCP (with videos). *Gastrointest Endosc*. 2010;**71**:413–9.
15. Ramesh H. Intrahepatic cholangiogastrostomy for malignant biliary obstruction at the hilum. *Br J Surg*. 1992;**79**:1349–50.

Segmental ileal dilatation: an unsuspected cause of neonatal intestinal obstruction

Introduction

Segmental dilatation of small intestine although described in literature, is among the rare causes of intestinal obstruction in neonates. The etiology of this condition remains unknown.[1] Clinical presentation in these patients is like any other intestinal obstruction but investigations often reveal a confusing picture, thereby arousing clinical suspicion of this condition. Once the diagnosis is confirmed resection of the affected segment is the treatment of choice. We present here a case of neonatal intestinal obstruction due to segmental ileal dilatation and discuss the pertinent clinical aspects of this condition with a relevant review of literature.

Case report

We present here a female child born to a primigravida mother at 37 weeks of gestation with birth weight of 2.2 kg. The baby

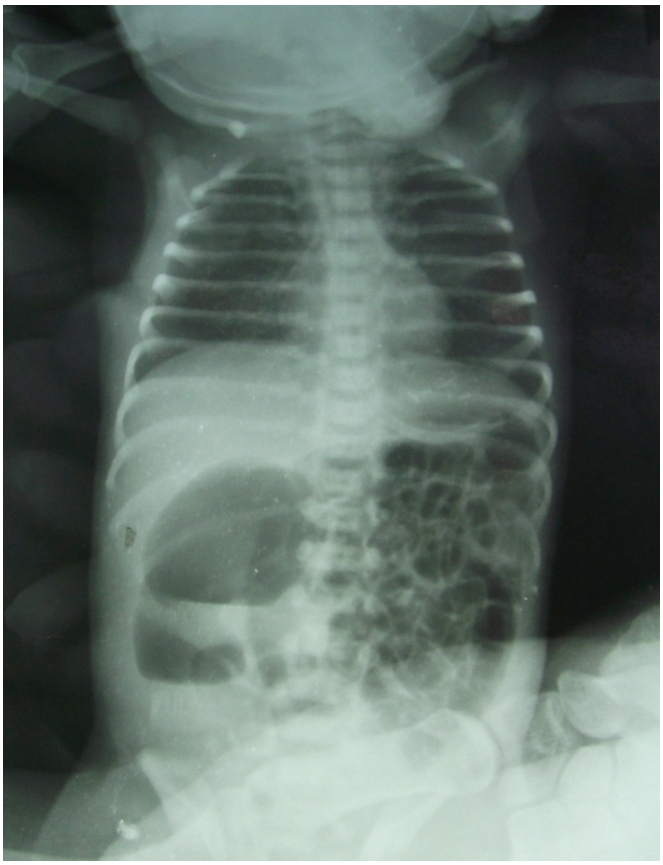

Figure 1: X-ray abdomen showing multiple gas filled small bowel loops on right side with large dilated loops seen on the right of the abdomen

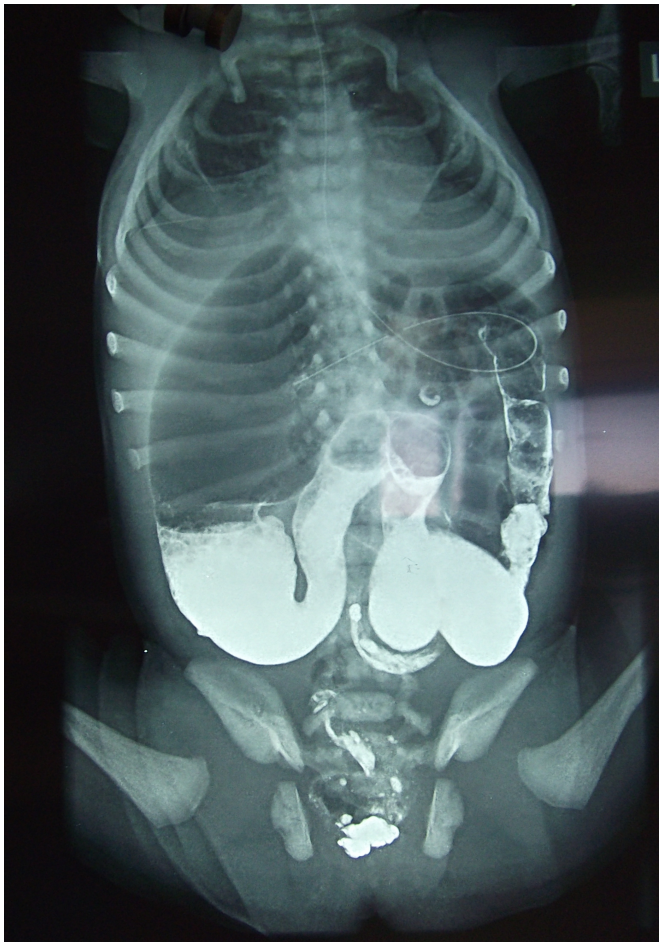

Figure 2: Upper gastrointestinal contrast showing massively dilated bowel loop on right side of abdomen with air contrast level

cried immediately after birth and passed meconium on first day of life. Around one week after birth the baby started developing abdominal distension, poor oral intake and bilious vomiting. With these symptoms the child was referred to our institute as a case of neonatal intestinal obstruction. On examination the child's activity was fair and vitals were stable although the child was dehydrated. Nasogastric tube aspirate was bilious and the abdomen was grossly distended. Bowel sounds were absent. Plain radiograph abdomen revealed a grossly distended small bowel loop on right side of the abdomen, while rest of the small bowel was mildly distended. There was no pneumoperitoneum (**Figure 1**). Gastrointestinal contrast study revealed a confusing picture with the same persistently distended bowel loop on right side of the abdomen with two separately distended contrast filled bowel loops in the abdomen making us suspect intestinal duplication, although a small quantity of dye was reaching till rectum (**Figure 2**).

On laparotomy an 8 cm segment of ileum, 10 cm proximal to the ileocecal junction was found dilated. The diameter of dilated segment was 3 cm. There was perforation on the antimesentric

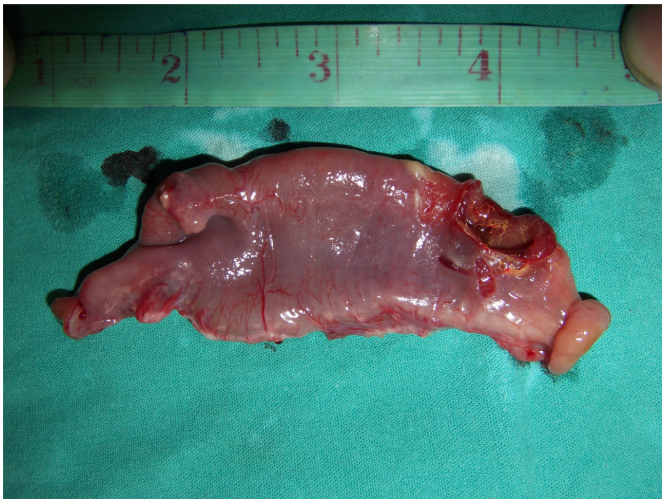

Figure 3: Resected dilated segment of ileum with perforation. Note the normal caliber intestine proximal and distal to the dilated segment

side of the dilated bowel. The small bowel proximal and distal to the dilated segment was of normal caliber and texture (**Figure 3**). The dilated segment of the ileum along with the perforation was resected and an end–to-end anastomosis of the normal

bowel was fashioned. The child was discharged uneventfully on eighth postoperative day. The histopathology of the resected specimen showed features of chronic inflammation with normal ganglion cells.

Discussion

Although neonatal intestinal obstruction due segmental intestinal dilatation is described in literature, etiology of this entity remains unknown.[1,2] It often manifests as an isolated, dilated small bowel segment, without evidence of intrinsic or extrinsic obstruction or abnormal neural innervation. In the neonatal period it presents with acute intestinal obstruction or can mimic Hirschsprung's disease, while in older infants it presents with anemia, malabsorption, chronic constipation or features of intermittent intestinal obstruction.[3] Swenson and Rathauser in 1959 established the criteria for the diagnosis of this rare entity.[4] Their criteria included, (i) limited bowel dilatation with a 3- to 4-fold increase in size, (ii) an abrupt transition between dilated and normal bowel, (iii) no intrinsic or extrinsic barrier distal to the dilatation, (iv) clinical picture of intestinal occlusion or sub-occlusion, (v) a normal neuronal plexus, and (vi) complete recovery after resection of the affected segment.

Around 150 odd cases of segmental intestinal dilatation are reported in literature but none of them provide any clues to the definite etiology of this disease.[5] The presence of hererotopic tissue like lung, pancreatic, esophageal, gastric, cartilage and striated muscle in the dilated segment is described by some authors.[6] Some authors suggest intrauterine vascular accidents or external compression to the fetal bowel as a probable cause of the intestinal dilatation.[7] Entrapment of the bowel, with incomplete intestinal obstruction within the omphalocoele during gestation, has also been postulated as a cause.[8] Cheng et al[9] demonstrated localized vacuolization of the intestinal smooth muscle in their case suggesting myopathy to the cause of dilatation. Partial or complete absence of muscularis propria in the dilated segment has also been reported by some authors but similar findings have not been observed by other authors.[10]

Although segmental dilatation can involve anywhere from duodenum to distal colon, ileum is the most commonly affected site.[11] The usual finding on laparotomy is localized dilation of an isolated, well defined segment of bowel with apparently normal bowel proximal and distal to this segment. The obstruction in these cases is a functional and non-mechanical because the lumen of the dilated segment is continuous with rest of the intestine, as was noted in our case. We could easily see the passage of feces and gas across the lumen of dilated segment while milking the bowel. The microscopic examination of the dilated segment also shows normal histology with normal innervations and normal distribution of ganglion cells.[7] The features of inflammation seen in our case were probably due to perforation of the dilated segment. Although resection of the dilated segment and end-to-end anastomosis of the normal bowel is the definitive curative treatment, the cause of this condition remains unexplained.

The diagnosis of segmental intestinal dilatation should be kept in mind while dealing with cases of neonatal intestinal obstruction. As the etiopathogeneis of the entity is still obscure, the resected dilated portion of the intestine should always be sent to appropriate experienced centers for a thorough histopathological and biochemical evaluation.

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References

1. Balik E, Taneli C, Yazici M, Demircan M, Herek O. Segmental dilatation of intestine: a case report and review of the literature. *Eur J Pediatr Surg*. 1993;**3**:118–20.
2. Ben Brahim M, Belghith M, Mekki M, Jouini R, Sahnoun L, Maazoun K, et al. Segmental dilatation of the intestine. *J Pediatr Surg*. 2006;**41**:1130–3.
3. Manikoth P, Paul J, Zachariah N, Vaishnav A, Sajwani MJ. Congenital segmental dilatation of the small bowel. *J Pediatr*. 2004;**145**:415.
4. Swenson O, Rathauser F. Segmental dilatation of the colon: a new entity. *Am J Surg*. 1959;**97**:734–8.
5. Ben Brahim M, Belghith M, Mekki M, Jouini R, Sahnoun L, Maazoun K, et al. Segmental dilatation of the intestine. *J Pediatr Surg*. 2006;**41**:1130–3.
6. Rovira J, Morales L, Parri FJ, Juliá V, Claret I. Segmental dilatation of the duodenum. *J Pediatr Surg*. 1989;**24**:1155–7.
7. Ratan SK, Kulshrestha R, Ratan J. Cystic duplication of the cecum with segmental dilatation of the ileum: report of a case. *Surg Today*. 2001;**31**:72–5.
8. Thambidorai CR, Arief H, Noor Afidah MS. Ileal perforation in segmental intestinal dilatation associated with omphalocoele.

Singapore Med J. 2009;**50**:e412–4.
9. Cheng W, Lui VCH, Chen QM, Tam PK. Enteric Nervous System, interstitial Cells of Cajal, and smooth muscle vacuolization in segmental dilatation of jejunum. *J Pediatr Surg*. 2001;**36**:930–5.
10. Huang SF, Vacanti J, Kozakewich H. Segmental defect of the intestinal musculature of a newborn: evidence of acquired pathogenesis. *J Pediatr Surg*. 1996;**31**:721–5.
11. Saha S, Konar H, Chatterjee P, Basu KS, Chatterjee N, Thakur SB, et al. Segmental ileal obstruction in neonates— rare entity. *J Pediatr Surg*. 2009;**44**:1827–30.

Cryptococcal meningitis in a patient with hepatitis C virus related decompensated cirrhosis: co-incidental or immunologically related?

Introduction

Systemic fungal infections are a less recognized complication of decompensated cirrhosis.[1] Recurrent behavioural abnormalities in cirrhotics are considered as episodes of hepatic encephalopathy. Meningitis is rarely suspected in these patients especially with lack of meningeal signs.[2] We present a patient with hepatitis C related decompensated cirrhosis of liver who developed recurrent altered mental status secondary to cryptococcal meningitis. We also discuss the possible immunological circumstances related to hepatic failure that may predispose to severe cryptococcal infection.

Case report

A 48-year-old lady presented with altered sensorium after an episode of generalized tonic-clonic seizures. There was no preceding headache, vomiting, fever or previous seizures. She had been diagnosed 2 years ago with hepatitis C virus related cirrhosis of liver with portal hypertension and grade 3 esophageal varices. On examination, she was in altered mental status (Glasgow coma scale (GCS) 9/15) (E2M4V3). Oral candidiasis was noticed. Neck rigidity was absent. On per abdominal examination, liver span was 10 cm. Spleen was just palpable below the left costal margin. Moderate ascites was noticed. Rest of the systemic examination was unremarkable. Her investigations revealed a haemoglobin of 8.5 gm/dl, TLC 3500 per mm^3, neutrophils 60%, lymphocyte 40%, normal platelets, ESR 30 mm first hour (Westergren method) and microcytic hypochromic general blood picture. Renal and liver function tests, prothrombin time, serum ammonia and thyroid functions were normal. Serum proteins/albumin was 5.9/2.3 gm/dl. ELISA for HIV was negative. CD4+ count was 750/mm^3. Thyroperoxidase antibodies were negative. Cerebrospinal fluid (CSF) examination revealed protein 55 mg/dl, cells 210 /mm^3 and sugar 65 mg/dl. CSF light microscopy with Gram, Ziehl Neelsen and India ink staining were negative. CSF culture for mycobacteria and fungi were sterile. CSF PCR for *Mycobacterium tuberculosis*, Varicella zoster, Herpes simplex type 1 and 2, and West Nile virus were negative. Blood and CSF ELISA for Japanese encephalitis and cryptococcal antigen were negative. MRI brain and electroencephalography was normal. By 14th day, she became afebrile and showed improvement in consciousness on treatment with broad spectrum antibiotics, fluconazole (for oral thrush) and supportive care and a repeat CSF examination revealed 20 cells (lymphocytes), protein of 140 mg%, and normal sugar. A possibility of nonspecific viral encephalitis was considered and the patient was discharged.

One month later, the patient returned with altered mental state again. There was no history of preceding headache, fever, behavioural changes or seizures. On examination, she had a GCS score of 9/15 (E3M4V2). Focal neurological signs and neck rigidity were absent. Systemic examination was unremarkable. Blood counts and biochemical examination was within normal limits. CSF revealed protein 133 mg/dl, sugar 56 mg/dl, cells 50/cumm (all lymphocytes), positive cryptococcal antigen and numerous cryptococcal cells on India ink preparation. *Cryptococcus neoformans* was recovered on CSF fungal culture. CSF bacterial and BACTEC culture were sterile. Ascitic fluid (transudative) was sterile on cultures. MRI brain showed dilated Virchow Robbin spaces in bilateral basal ganglia and numerous cryptococcomas in the central semiovale (**Figures 1 & 2**). The patient was instituted on conventional amphotericin B at 1 mg/kg body weight and 5-flucytosine at 2 gm/day. After one week, she developed spontaneous left hydropneumothorax which was drained by intercostal drainage. The pleural fluid was exudative but bacterial, BACTEC and fungal cultures were sterile. Anti-tubercular treatment was added on an empirical basis. The chest tube was removed after 2 weeks with no recurrence of hydropneumothorax. Despite improvement in CSF cell counts, the patient remained stuporous. Repeat CSF fungal cultures at 2 weeks showed heavy growth of *Cryptococcus neoformans*. Repeat MRI did not reveal hydrocephalus or infarcts. Amphotericin B and 5-flucytosine was continued along with the four drug anti-