Bowel Gangrene in Congenital Mesenteric Defects: Not Always Due to Volvulus or Strangulation

A 7-month-old female baby weighing 7.4 kg presented with abdominal distension, bilious vomiting, low urine output and fever for two days. Examination revealed pallor, dehydration and feeble peripheral pulses. The abdomen was distended and tender. Plain abdominal *X*-ray showed multiple air-fluid levels. Ultrasound scan of the abdomen showed some free fluid and there was no intussusception. Hemoglobin was low and renal functions were normal. Clinical setting of shock with tender abdomen suggested a bowel catastrophe. Emergency laparotomy after adequate resuscitation revealed 70 mL of hemorrhagic fluid in the peritoneal cavity, and on evisceration and simply splaying the intestines, we found two large 'V' shaped mesenteric defects in the jejunal mesentery approximately 30 cm from duodeno-jejunal flexure, each measuring 10x12cm with a short strip of intact intervening mesentery containing a feeding vessels in between the two defects (***Fig.* 1**). The jejunum overlying the defects had multiple impending perforations and necrotic patches. There was no marginal vascular arcade along both the jejunal mesenteric defects. Another small mesenteric defect measuring 2x4 cm was noted in the ileal mesentery but the overlying ileum was normal. There was no evidence of malrotation. Rest of the bowel was normal. Fifty centimeters of necrotic jejunum overlying the defects was excised and jejuno-ileal anastomosis was performed. Jejunal and ileal mesenteric defects were closed. Post-operative recovery was uneventful. The histopathology of the resected jejunum showed scattered areas of mucosal ulceration, hyperemia and villous atrophy with hemorrhagic necrosis and leucocytic infiltration suggestive of ischemic enteritis. There was no evidence of fibrosis or stricture.

Common causes of bowel gangrene in infants are intussusception and midgut volvulus. Congenital mesenteric defects (CMDs) are usually accompanied by corresponding bowel atresia and present acutely at birth. CMD not associated with bowel atresia are rare and present with intestinal obstruction or bowel gangrene due to internal herniation or volvulus at a later age with or without bowel gangrene [1]. First reported in 1960 [2] CMDs are rare but an important cause of intestinal obstruction in all age groups; they may be asymptomatic, can present with shock or can be a cause of unexpected death [3]. CMD can also present with recurrent

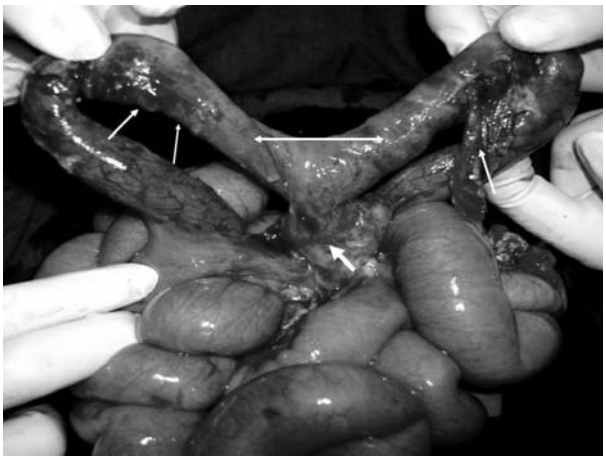

FIG. 1 *Intraoperative photograph showing two large jejunal mesenteric defects with an intervening strip of intact mesentery with a feeding vessel (thick arrow). Absence of marginal vascular arcade along both the defects and distribution of necrotic patches in the mid portion of the jejunal segment overlying each defect (white arrows). Few Impending perforations are sealed by omentum, and a very pink segment of jejunum being supplied by feeding vessel (double head arrow).*

constipation, recurrent abdominal pain or recurrent intestinal obstruction due to intermittent internal herniation, those are often misdiagnosed [3,4]. CMDs can be either segmental defects or basilar defects, and mostly located in the ileocecal mesentery [5]. Rarely, segmental defects can occur both in jejunal and ileal mesentery as in our index case. Most CMDs are 2 to 3 cm wide and usually have a marginal vessel, unlike this case where defects were quite large and marginal vessels were conspicuous by their absence. Adjacent bowel might get trapped in the defect resulting in strangulation, or bowel overlying the defect might twist on its supplying vessel leading to volvulus, both causing occlusive ischemia leading to bowel gangrene. However, contrary to the above fact, CMD in our case were too large to trap adjacent bowel and there was no volvulus found intraoperatively, however the possibility of a spontaneously reduced volvulus cannot be ignored. Large CMD with absent marginal vessels and long segment of growing bowel overlying the CMDs runs a significant risk of vascular insufficiency. In the absence of occlusive phenomenon, a non-occlusive ischemic trigger (like infections especially viral, dehydration or shock) to a bowel, which already has a diminished blood supply, can lead to enterocolitis. However, the exact etiology of the necrosis in our case is not known and it could be due to either an infection, dehydration, shock or a spontaneously reduced volvulus.

T RENUKUMAR and *M ANANTH SAGAR
*Department of Pediatric Surgery and *Pediatrics, Vaatsalya Hospital, Three Lamps Junction, Vizianagram, Andhra Pradesh 535 002, India.*
drtrk2007@rediffmail.com

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Barium Toxicity - A Rare Presentation of Fireworks Ingestion

Barium toxicity is reported in exposure to explosives, fireworks, chemical compounds and presents as severe hypokalemia due to redistribution of potassium in the body. We report a 16-year-old boy who ingested fireworks and presented with acute quadriparesis and respiratory failure due to severe hypokalemia. He had gastroenteritis, pain abdomen, difficulty in breathing, and generalized weakness. Investigations showed serum potassium of 0.2 meq/L. He was ventilated and received rapid potassium correction. He subsequently developed ventricular tachycardia, which reverted with rapid potassium infusion.A total of 360 meq of potassium was supplemented in first 20 hours. He regained complete muscle power by day 3. Barium nitrate, commonly used in fireworks, is highly toxic and can lead to hypokalemia. We suspected barium poisoning in our index case and samples of blood and urine were sent for toxicological analysis.Blood barium levels were 98.5 mcg/dL (normal 3-20 mcg/dL).

Barium compounds are highly toxic when ingested with the exception of barium sulphate which is not absorbed from gut and hence commonly used as radiographic contrast. Fireworks contain Barium chlorate and nitrate which gives the yellow green flame on igniting [1]. Barium interferes with potassium transport, causing intracellular sequestration of the ion and severe hypokalemia. Effects of barium include gastroenteritis, cardiac instability, wide complex arrhythmias, muscle weakness, hypertension and respiratory failure [1-4]. Renal toxicity with ingestion of large amounts of barium is also reported [5]. Barium carbonate poisoning from rodenticide ingestion has been reported to cause acute rhabdomyolysis and hypophosphatemia. No specific antidote is known for barium toxicity. Patients requires large doses of potassium supplementation and respiratory support [1-5]. Use of oral magnesium sulphate to form barium sulphate prevents further absorption of barium from GI tract. In refractory cases with cardiac instability or renal toxicity hemodialysis can be effective [5].

B DEEPTHIRAJU AND PRK VARMA
Department of Pediatrics, Varma Hospitals, JP Road, Bhimavaram 2, Andhra Pradesh 534 202, India.
drdeepthiraju@gmail.com

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