Case Report

Bleeding oesophageal varices in a 9 months old infant as a complication of neonatal umbilical catheterization – A case report

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ABSTRACT

Esophageal variceal bleeding following portal hypertension (PHT) is rare in children but associated with significant morbidity and mortality. Neonatal umbilical catheterization is a risk factor for portal vein thrombosis and subsequent development of PHT. We report the case of a 9-month-old male infant that presented with upper gastrointestinal bleeding from esophageal varices. He was delivered at 32 weeks of gestation and had neonatal umbilical catheterization for intravenous fluids and antibiotics administration. Barium meal done after resuscitation revealed esophageal varices. He had a blood transfusion while on admission and was discharged home on oral propranolol. This case report highlights a rare case of bleeding esophageal varices secondary to PHT occurring in an infant who had neonatal umbilical catheterization.

Key words: Bleeding esophageal varices, Portal hypertension, Umbilical catheterization

Esophageal variceal bleeding, though rare in children, remains a major cause of significant morbidity and mortality [1,2]. It may results in rapid depletion of the circulatory volume in children, as a result of their relatively small total blood volume [3]. Hence, effective resuscitation followed by prompt diagnosis, controlling of bleeding, and prevention of complications are important steps in the management of acute variceal bleeding [4].

In children, portal hypertension (PHT) from portal vein thrombosis (PVT) is the most common etiology of esophageal varices [1,5]. Risk factors for this include neonatal umbilical catheterization, omphalitis, neonatal sepsis with abdominal focus, and dehydration [6,7]. These factors result in PVT and subsequent disruption of blood flow throw the portal system leading to PHT [8].

The umbilical venous access (through umbilical catheterization) is one of the most commonly used routes for blood transfusion, intravenous fluids, and drug administration as well as parenteral nutrition in preterm neonates [9]. Catheter tip malposition, infection, and prolonged duration increase the risk of PVT [1]. We, therefore, report a case of bleeding esophageal varices in a 9-month-old infant who had umbilical catheterization during the neonatal period. This highlights a rare case of PHT occurring in an infant; hence, the need for screening for PHT in all infants presenting with upper gastrointestinal (GI) bleeding.

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CASE REPORT

A 9-month-old male infant, a product of preterm delivery, presented with complaints of fever 4 days duration, vomiting of the blood of 12 h duration, and passage of blood per rectum of 6 h duration. Fever was noticed a few hours after immunization (measles, yellow fever, and meningococcal). He had three bouts of bloody vomitus; volume estimated to be 20 ml each and contains blood clots. The passage of blood per rectum was five bouts and contains altered blood. There was associated paleness of the body and body weakness. No bleeding from any other body orifices. He was admitted for 2 weeks during the neonatal period on account of prematurity with respiratory distress syndrome (delivered at 32 weeks of gestation). He had umbilical catheterization during the neonatal period which was used for intravenous fluids and antibiotics administration. He is also awaiting hydrocelectomy for bilateral congenital hydrocele by the pediatric surgical unit. Since the onset of this current illness, he was given oral artemisinin-based combination therapy, paracetamol, and cefixime before presentation to the emergency pediatric unit.

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Physical examination revealed an acutely ill-looking child with high-grade pyrexia (temperature = 39.0°C), severe pallor, moderate dehydration, moderate wasting, tachycardia (heart rate = 148 beats/min), tachypnea (respiratory rate = 44 breaths/min), and blood pressure = 80/50 mmHg, apex beat on the left fourth intercostal space, mid-clavicular line, and a normal first and second heart sounds. The spleen was palpable 5 cm along its longest axis. There is a presence of bilateral scrotal swelling which was transilluminating. Digital rectal examination revealed good anal sphincteric tone, empty rectum with normal mucosa, and no masses palpable. The examination finger was stained with blood.

A diagnosis of upper GI bleeding secondary to esophageal varices was made with a differential diagnosis of sepsis with disseminated intravascular coagulopathy.

Laboratory investigations showed packed cell volume of 24% and hemoglobin concentration of 7.4g/dL with a normal platelet count of 200,000 cells/mm^3. The white blood cell parameters were within normal limit. The serum electrolytes, urea, and creatinine were normal (sodium= 142 mmol/L, potassium = 4.9 mmol/L, bicarbonate = 20 mmol/L, chloride = 104 mmol/L, urea = 6.6 mmol/L, and creatinine = 30 μmol/L).

He was commenced on intranasal oxygen, intravenous fluids (normal saline and subsequently 4.3% dextrose in one-fifth normal saline), Vitamin K, octreotide, amoxicillin (100 mg/kg/day in three divided doses), and paracetamol. He was also transfused with fresh whole blood twice within 48 h. Hematemesis subsided following admission, however, he continued to pass altered blood per rectum for 72 h and then stopped.

Barium meal done (after the resolution of symptoms) showed opacified esophagus with persistent multiple serpiginous intraluminal filling defects suggestive of varices (Fig. 1). An abdominal ultrasound scan showed moderate splenomegaly measuring 79 mm (with uniform parenchymal echogenicity). The liver was normal in size and shows normal parenchymal echogenicity and regular outline.

He was subsequently discharged home on oral propranolol after 5 days on admission to continue follow-up in pediatric gastroenterology clinic.

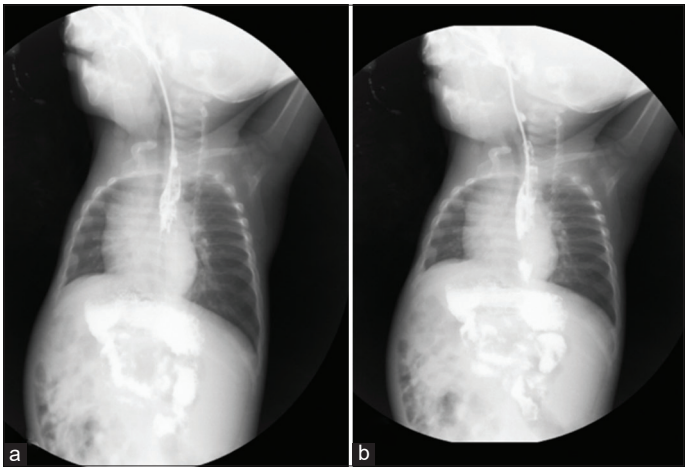

Figure 1: (a and b) Barium meal showing opacified esophagus with multiple serpiginous intraluminal filling defects suggestive of varices

DISCUSSION

PHT and the complications associated with it have remained a major cause of significant morbidity and mortality in children [2]. Its main complication is upper GI bleeding from esophageal varices, which may present as a challenging clinical spectrum warranting inpatient care and emergency medical management [6,10]. Children with PHT usually present with upper GI bleeding and splenomegaly without hepatomegaly [11]. In India, esophageal varices were found in 40–95% of children presenting with significant upper GI bleeding [12,13]

The significant increase in the number of premature deliveries has made umbilical vein catheterization a common bedside procedure in the neonatal intensive care unit [14]. Although it is important vascular access in neonate for the administration of intravenous fluid and drugs, exchange blood transfusion and parenteral nutrition are associated with several complications such as thrombus formation, embolism, vessel perforation, hemorrhage, infection, cardiac arrhythmias, pericardial, and pleural effusion [14-16].

Our index case was a product of preterm gestation delivered at 32 weeks. He had umbilical catheterization during the neonatal period for intravenous fluids and drug administration. He subsequently presented with hematemesis and passage of melena stools at the age of 9 months. Similar cases of upper GI bleeding following umbilical catheterization were reported by Rahman *et al*. [17], Rogvi *et al*. [18], and Ugwu *et al*. [1] who presented at the ages of 2.6 years, 7 years, and 9 years, respectively.

The factors associated with increased risk of developing PVT following neonatal umbilical catheterization include lack of use of anticoagulant, malposition of the catheter tip, and prolong catheterization period [15]. Pre-treatment of the umbilical catheter with anticoagulant was not done in our index patient and the position of the umbilical catheter tip was not determined. Although the patient spent 2 weeks on admission, the catheter indwelling time could not be ascertained.

The detection of esophageal varices is important in the diagnosis of PHT [19]. Although upper GI endoscopy is the gold standard for the diagnosis of esophageal and gastric varices, barium swallow/meal can detect varices in up to 90% of cases [20]. In our index case, barium meal was used to demonstrate the presence of esophageal varices due to the unavailability of pediatric endoscope at the time of writing this report.

Effective fluid resuscitation including blood transfusion and controlling of bleeding precedes the definitive management of acute variceal bleeding. A study by Ferri *et al*. [6] showed that 79.5% of their patients presented with bleeding esophageal varices following PHT required blood transfusion. Our index case was transfused with two units of fresh whole blood. The definitive treatments include endoscopic procedure (sclerotherapy or band ligation) and surgery (portosystemic shunts) [6]. The indications for surgical treatment include recurrent bleeding after appropriate endoscopic treatment, massive splenomegaly and/or severe

hypersplenism, growth retardation, and symptomatic portal biliopathy [6].

Although rebleeding may occur in 50% of cases, the overall prognosis in patients with variceal bleeding from PHT is determined by the amount of blood loss and the status of the liver before the bleeding [17]. The prevention of variceal bleeding/ rebleeding in children with PHT can be achieved by the use of non-selective β-blockers such as propranolol and carvedilol [21].

CONCLUSION

Neonatal umbilical catheterization, though a useful route for administration of intravenous fluid and drugs in neonates, is associated with several complications such as PVT and subsequent development of PHT. Esophageal variceal bleeding, a common complication of PHT, is associated with significant morbidity and mortality which can occur even in infants. Therefore, a high index of suspicion is required to detect esophageal variceal bleeding in infants, especially those with a history of neonatal umbilical catheterization.

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