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TEACHING FILES (GRAND ROUNDS)

HYPERPLASTIC DUODENAL AND JEJUNAL POLYPS IN A CHILD WITH PORTAL CAVERNOMA

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Clinical Problem

A 7-year-old boy presented with abdominal pain for 2 days. He was diagnosed to have extrahepatic portal hypertension with portal cavernoma at the age of 1 year. At that time, he had hematemesis and malena. However, colonoscopy and esophagogastroscopy (OGDscopy) showed no varices. Ultrasound abdomen at that time showed multiple channels at porta with hepatopetal flow suggestive of portal cavernoma. There was no history of umbilical sepsis, jaundice or umbilical catheterization. Subsequently he had no further bleeding episodes. On presentation to us, height was 116 cm and weight was 18.3 kg. He had a 10 cm splenomegaly. Other systems were normal. Investigations showed hemoglobin of 8.8 gm/dL, white cell count 2600 cells/cumm (56% polymorphs, 31% lymphocytes) and platelets of 50,000 cells/cumm. Liver function tests were normal. Ultrasound abdomen showed normal liver with portal cavernoma with periportal, pericholecystic, peripancreatic, retroperitoneal and rectal collaterals. Spleen measured 17.3 cm and splenic vein was normal. He was started on propranolol (1 mg/kg), folic acid and iron supplements and advised OGDscopy which showed grade 3 varices for which band ligation was done. One month later, he had malena and again OGDscopy was done. Presence of duodenal polyps was noted. Five months later, he again had malena and OGDscopy was done that showed grade I-II esophageal varices, portal gastropathy and multiple duodenal and proximal jejunal polyp. One jejunal polyp was actively oozing which was controlled with saline adrenaline injection and bipolar probe coagulation. Biopsy of duodenal polyp was done which on histopathology was suggestive of hyperplastic polyps. He was advised colonoscopy to rule out colonic polyps, which has not been done yet due to financial constraints. He is on regular follow up.

What is the cause of duodenal and jejunal polyps?

Discussion

In children and neonates, portal vein obstruction can be due to intra-abdominal infection, neonatal sepsis with umbilical catheter placement, congenital anomalies of the portal venous system, often associated with cardiovascular anomalies and biliary tract abnormalities.[1] Patients with chronic portal vein thrombosis develop collateral blood vessels that bring blood in a hepatopetal manner around the area of obstruction, known as cavernous transformation of the portal vein or portal cavernoma.[2] The development of collateral circulation, with its attendant risk of variceal hemorrhage, is responsible for most of the complications and is the most common manifestation of portal vein obstruction.[1] In addition, less common features include portal hypertensive gastropathy, portal colopathy and portal hypertensive enteropathy (PHE).[3] Polypoid enteropathy is a rare manifestation of PHE and has been associated with occult or overt gastrointestinal bleeding.[4]

Portal hypertensive polypoid enteropathy is a rarer manifestation of PHE and polypoid lesions are less common in the small intestine (0.3%) compared to polypoid gastropathy (0.6%).[5] PHE can present as anemia, melena, hematochezia, hematemesis or may be asymptomatic. PHE should be suspected when there is gastrointestinal bleeding or anemia not otherwise explained by more common etiologies, along with signs of portal hypertension such as ascites, splenomegaly, thrombocytopenia, or hepatic venous pressure gradient more than 8 mmHg.[5,6] Endoscopically, polypoid lesions can have varied manifestations and may occasionally mimic adenomatous polyps. Typical features of portal hypertensive polyps reportedly include foveolar hyperplasia of the epithelium as well as proliferating, ectatic capillaries in the lamina propria; this indicates their portal hypertensive nature, distinguishing them from inflammatory polyps.[7] Treatment of portal hypertensive polypoid enteropathy depends on number of polyps and endoscopic accessibility. A polypectomy can be safely performed if the polyp is accessible and amenable for endoscopic removal. Endoclip can be used at the stalk to achieve complete hemostasis.[8] Argon plasma coagulation can be used on the inflamed surface of bleeding polyp to achieve hemostasis, but recurrent bleeding has been reported.[5] Non-selective beta blocker, transjugular intrahepatic portosystemic shunt (TIPS), surgical small bowel resection, and liver transplantation have all been reported anecdotally to be successful treatment for portal hypertensive polypoid enteropathy.[5,8]

In summary, duodenal polyps secondary to PHE is

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uncommon. The finding of multiple polyps in a child with portal hypertension should raise suspicion for this entity and management should be individualized based on acuity and severity of the hemorrhage, endoscopic accessibility of the lesion, surgical risk of the patient, patency of portal vein, available therapy and expertise of each institution.[4]

Compliance with ethical standards

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