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Bleeding Colonic Varices as a Presentation of Malrotation in a 21-Year-Old: A Case Report and Review of the Literature

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1. Abstract

1.1. Background

Intestinal malrotation is a congenital anomaly resulting from incomplete rotation of the midgut during fatal development. Most cases present in the neonatal period and adult presentations are rare. Colonic varices as a complication of malrotation have rarely been reported in the literature.

1.2. Case Presentation

We present a 21-year-old female with a history of malrotation who presented with hematochezia and was found to have bleeding colonic varices in the ascending colon and terminal ileum. Initial hemostasis was achieved endoscopically with Hemospray. Following recurrent episodes of lower GI bleeding, the patient underwent a robotic Ladd's procedure, after which no further bleeding occurred.

1.3. Conclusion

This case represents a rare presentation of malrotation causing colonic variceal hemorrhage in an adult, likely through mesenteric venous outflow obstruction related to Ladd's bands and narrow mesenteric pedicle. Definitive anatomic correction with a Ladd's procedure led to resolution of the varices.

2. Introduction

Intestinal malrotation is a congenital anomaly resulting from the partial or complete arrest of normal gut rotation during fetal development, leading to abnormal orientation of the bowel and mesentery. Normal midgut rotation in utero involves 270 degrees of counterclockwise rotation around the superior mesenteric artery (SMA) axis, occurring between the 4th and 10th weeks of gestation [1,2]. There are several subtypes of malrotation, including non-rotation, incomplete rotation, reverse rotation, and anomalous fixation of the mesentery [1].

The estimated incidence of malrotation is approximately 0.2% of live births. However, approximately 75% of cases are identified within the first year of life, and adult presentations are uncommon [3]. About 33% of intestinal malrotations occur within the first month of life and 60% within the first year [3]. The true incidence in adults is estimated to be between 0.00001% and 0.19%, as many adults remain asymptomatic [3]. In symptomatic adults, abdominal pain is the most common complaint, occurring in up to 87% of patients, and some patients experience symptoms for years prior to diagnosis [3]. Gastrointestinal (GI) bleeding as a presenting manifestation

Figure 1: Varices in the ascending colon (left and center) and in the terminal ileum (right).

of malrotation is not a recognized classic presentation and is largely confined to isolated case reports.

Colonic varices are a rare cause of lower GI hemorrhage. In most cases, they occur in the setting of portal hypertension secondary to liver cirrhosis. However, it is estimated that only 3.4% of patients with cirrhosis-related portal hypertension develop colonic varices [13]. When varices occur outside the gastroesophageal region, they are referred to as ectopic varices. Ectopic varices carry a mortality rate of up to 70% during a bleeding episode and a four-fold increased risk of bleeding compared to gastroesophageal varices [13]. Idiopathic colonic varices in the absence of cirrhosis or portal hypertension are exceedingly rare, and their occurrence in the specific context of malrotation has been described only in a small number of cases in the literature [7,14].

We present a case of a 21-year-old female with known malrotation who developed massive bleeding from colonic varices, managed initially with endoscopic Hemospray and ultimately treated definitively with a robotic Ladd's procedure. This is the first case of bleeding from colonic varices resulting from malrotation-associated mesenteric venous outflow obstruction in an adult and supports the role of definitive anatomic correction in achieving durable resolution.

3. Case Presentation

A 21-year-old female who presented with hematochezia and was found to have intestinal malrotation on computed tomography angiography (CTA) imaging. She was referred to a surgeon for possible Ladd's procedure. However, due to her school schedule, she could not make that visit. She present- ed a few months later with another episode of hematochezia, and another CTA of the abdomen and pelvis was interpreted as showing inferior mesenteric vein (IMV) thrombosis, and so she was initiated on therapeutic heparin. This resulted in a massive GI bleed and syncopal episode. Colonoscopy was performed, during which 1.6 liters of blood was evacuated from the colon. Colonic varices were identified, but no active bleeding source was localized at that time. She was then transferred to the University Hospital for a higher level of care. On arrival at the University Hospital, her hemoglobin was 7.4 g/dL. She remained hemodynamically stable after resuscitation with blood products but continued to have bloody bowel movements. Repeat CTA of the abdomen and pelvis demon-

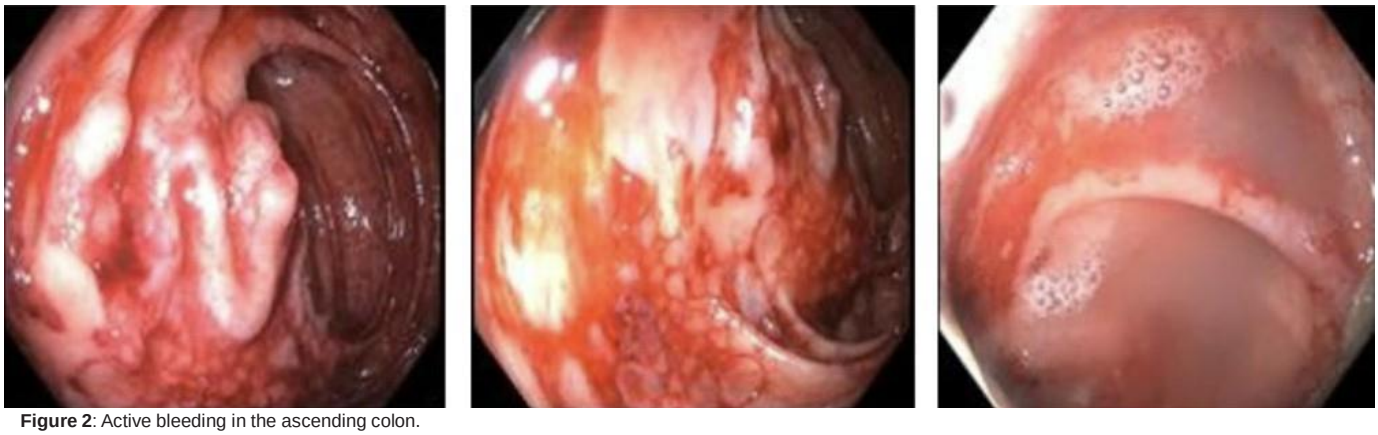

Figure 2: Active bleeding in the ascending colon.

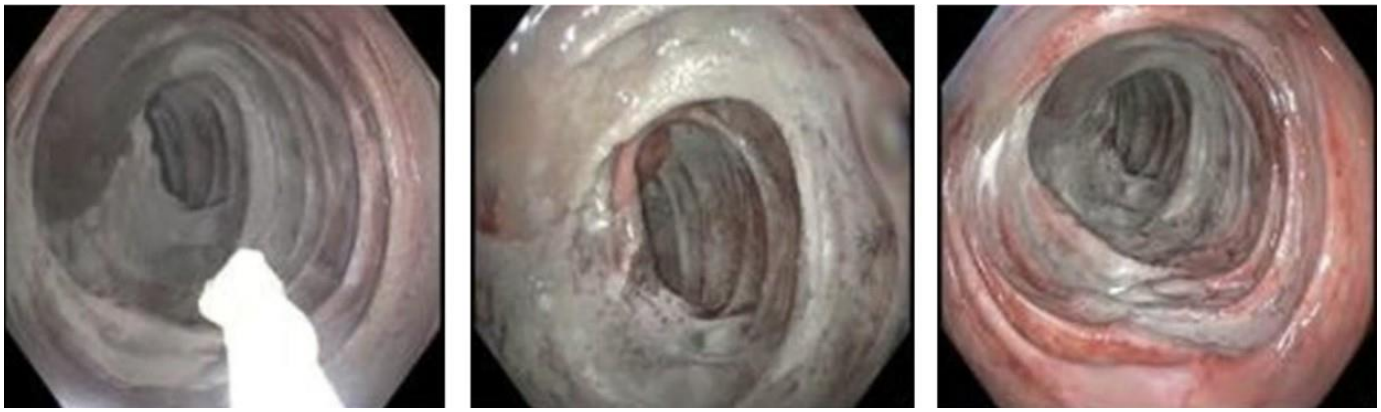

Figure 3: Post-Hemospray application demonstrating effective hemostasis.

strated the cecum in the left upper quadrant, consistent with her known malrotation. There was significant compression of the left renal vein, and prominent dilated vessels lining the colon and distal small bowel were noted consistent with varices. A repeat colonoscopy revealed extensive varices in the colon and terminal ileum, with active bleeding in the ascending colon. Hemospray was applied endoscopically as a temporizing measure, achieving effective hemostasis. Her hemoglobin and hematocrit remained stable. She was discharged and seen in follow-up two weeks later, at which time she reported doing well with normal daily bowel movements. After multidisciplinary discussion, a Ladd's procedure was recommended as definitive treatment. She was scheduled for surgery based on her school schedule. However, she was subsequently hospitalized twice following her index admission for recurrent lower GI bleeding, each time undergoing repeat colonoscopy with Hemospray application. She ultimately underwent a robotic Ladd's procedure and has experienced no further bleeding episodes in 15 months.

4. Discussion

Intestinal malrotation results from arrest of the normal 270° counterclockwise rotation of the midgut around the SMA axis during embryonic development [1,2]. The estimated incidence is approximately 0.2% - 0.5% of live births. It is usually diagnosed in the first year of life in the majority of affected individuals, making adult presentations uncommon [3]. The clinical spectrum in adults ranges from asymptomatic disease to acute or chronic abdominal pain, bowel obstruction, volvulus, and as in our patient GI bleeding [3]. A key anatomical feature of malrotation is the narrow mesenteric pedicle and the malfixation of the mesentery, which shortens the normally broad mesenteric attachment and predisposes the patient not only to volvulus, but also to progressive impairment of mesenteric venous outflow [3].

Only a few cases of colonic variceal bleeding have been published in the literature, but none were associated with malrotation. We believe the varices in our patient arose from venous outflow obstruction caused by Ladd's bands and the narrowed mesenteric pedicle, resulting in mesenteric venous hypertension and the formation of collateral vessels within the colonic submucosa. This pathophysiological mechanism has been established in prior literature, most directly by Park and Watkins, who described a pediatric case in which long-standing obstruction of the superior mesenteric vein (SMV) in the setting of midgut malrotation and volvulus produced intestinal variceal bleeding from the duodenum [7]. In a review of 62 cases of small and large bowel bleeding, Cappell and Price identified malrotation as a predisposing condition [13]. Walsh and Crombleholme described a 13-year-old girl with an 11-year history of recurrent abdominal pain who had been evaluated at 3 other institutions without a diagnosis. A CT scan of the abdomen demonstrated a calcified thrombus within the SMV. She was found at laparotomy to have malrotation with chronic 270° volvulus and evidence of mesenteric venous hypertension [15]. Dubovan et al. reported a 22-year-old patient with both intestinal malrotation and congenital absence of the proximal SMV who developed tortuous duodenal collaterals and submucosal varices causing chronic GI blood loss from infancy [16]. Another case report described a 31-year-old patient with a 4-year history of melena and hematemesis associated with abdominal pain. She had undergone a Roux-en Y Gastric Bypass (RYGB) 8 years earlier. Balloon enteroscopy revealed ileal submucosal varices that were treated with endoscopic sclerotherapy. She continued to bleed and upon review of her CTA, she was found to have large intraabdominal

venous collaterals from the SMV, which was obstructed by a Petersen's hernia. At surgery, the internal hernia was found to cause SMV obstruction leading to formation of varices. The varices decompressed intraoperatively once the hernia was repaired [17]. Collectively, these cases corroborate the fact that malrotation-associated mesenteric venous compromise whether from Ladd's bands, volvulus, or anatomic SMV anomaly can cause variceal pathology and subsequent GI hemorrhage.

Idiopathic colonic varices are a rare cause of lower GI hemorrhage. Only a small number of cases have been published describing idiopathic colonic variceal bleeding in the absence of cirrhosis or classic portal hypertension. In our case, the varices arose not from liver disease but from mesenteric venous outflow occlusion resulting from malrotation.

Among published cases, one involved a 32-year-old male in Brussels who presented with recurrent hematochezia and anemia from idiopathic, non-cirrhotic varices of the rectum and sigmoid colon [4]. Upper GI endoscopy showed no stigmata of portal hypertension and liver function tests were normal. Colonoscopy demonstrated large tortuous submucosal veins extending from the dentate line up to 40 cm. CT angiography confirmed significantly dilated sigmoidal and rectal veins, and the authors ultimately opted for surgical resection [4]. A second case involved a 54-year-old male with no prior GI history who presented with rectal bleeding [5]. Colonoscopy revealed large tortuous colonic veins without active bleeding, and portal hypertension was excluded by hepatic venous pressure measurement. This patient was managed non-operatively [5]. A third case involved a 38-year-old female with alcoholic liver cirrhosis and massive hematochezia in whom CT demonstrated ascending colonic varices that was subsequently treated with venous coil embolization [6]. Sunkara et all also reported a case of a 71-year-old female who underwent a screening colonoscopy and was found to have large submucosal varices extending from the sigmoid colon to the cecum. She recalled a few episodes of hematochezi earlier. CTA of the abdomen and pelvis showed no abnormalities [14]. Most recently, Jonnagaddala et al. described a 2025 case of colonic variceal bleeding without underlying liver disease, noting the diagnostic and therapeutic challenges posed by non-cirrhotic cases [18].

There are no randomized trials or formal clinical guidelines governing the management of colonic variceal hemorrhage, and treatment strategies have been largely extrapolated from upper GI variceal bleeding management and individual case series [14]. Reported options include endoscopic, endovascular, and surgical approaches.

Hemospray is an inorganic powder delivered endoscopically that absorbs moisture from an active bleeding site, forming a mechanical barrier, concentrating clotting factors, and promoting hemostasis [9]. While effective in achieving rapid hemostasis in both upper and lower GI applications, some series report non-trivial rates of rebleeding following its use [10]. In our patient, Hemospray provided effective initial hemostasis on multiple occasions but was not curative, consistent with its established role as a temporizing rather than definitive intervention.

Endovascular options include mechanical coil embolization of variceal vessels and transjugular intrahepatic portosystemic shunt (TIPS). TIPS is effective for portal hypertension-related varices from liver disease but would not be mechanistically appropriate for malrotation-associated mesenteric venous hypertension, in which the pathology is in distal segmental veins and not hepatic in origin [11]. Splenic vein stenting has been described for colonic varices secondary to pancreatitis-induced splenic vein thrombosis [19]. For endoscopic management of gastric and esophageal varices, cyanoacrylate injection, band ligation, and sclerotherapy remain established modalities [11].

For malrotation specifically, the definitive treatment is a Ladd's procedure. The procedure involves reducing the volvulus by counterclockwise rotation of the bowel, dividing Ladd's bands, broadening the mesenteric base, repositioning the small bowel on the right and large bowel on the left, and performing an appendectomy [3,12]. The Ladd's procedure has been performed successfully via laparoscopic approach, with the largest modern series from the Mayo Clinic demonstrating that among 44 adult patients, more than 90% achieved partial or complete symptom resolution at a median follow-up of 8 years, with median hospital stay of 2 days [20]. Laparoscopic repair has demonstrated a statistically significant reduction in length of stay compared to open repair, with comparable complication profiles [20]. A recent systematic review and meta-analysis of laparoscopic versus open Ladd's procedure, including 14 studies concluded that laparoscopic Ladd's procedure was superior to open Ladd's procedure in operative time, hospital stay, time to full feeds, overall complications

and postoperative adhesive small bowel obstruction [22]. Robotic-assisted Ladd's procedure is exceedingly rare in the literature. Gibson et al. described the first documented robotic-assisted laparoscopic Ladd's procedure in a 22-year-old female with malrotation [21]. Published experience with robotic Ladd's in adults remains limited to isolated case reports since that initial description. The robotic platform may offer technical advantages in the confined mesenteric dissection required, with improved visualization and instrument articulation. Our patient underwent a successful robotic Ladd's procedure with no further bleeding, making this among the very few documented cases of robotic Ladd's in adults and the first in the context of variceal hemorrhage.

In our patient treating the malrotation, which was the underlying cause of the colonic varices resulted in complete resolution of hemorrhage without bowel resection, in contrast to idiopathic colonic variceal cases where subtotal colectomy was required [4,13]. Our case provides strong evidence that Ladd's procedure should be considered not only for classic indications of obstruction and pain, but as curative therapy when malrotation is identified as the etiology of variceal hemorrhage.

5. Conclusion

We present a 21-year-old female with intestinal malrotation who developed massive lower GI hemorrhage from colonic varices of the ascending colon and terminal ileum. The patient was initially misidentified as having IMV thrombosis and treated with therapeutic heparin, resulting in clinical deterioration. Repeat imaging and endoscopy established the diagnosis of colonic variceal bleeding in the context of malrotation. Hemospray provided effective temporary hemostasis on multiple occasions, but the patient experienced recurrent episodes of hemorrhage until definitive anatomic correction was performed with a robotic Ladd's procedure, after which no further bleeding occurred. Though rare, colonic varices can develop from mesenteric venous outflow obstruction from Ladd's bands, volvulus or the narrow mesenteric pedicle associated with intestinal malrotation. Definitive anatomic correction with a Ladd's procedure can resolve the underlying vascular pathology and achieve durable hemostasis.

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