Stomal Varices

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Background

Patients with surgically created stomas can start with or develop liver disease in their lifetime, often as a result of the same pathology that required fecal or urinary diversion. When these patients develop gastrointestinal bleeding, the astute clinician must always be aware of the possibility of variceal hemorrhage originating from the stoma's mucocutaneous junction.

Stomal varices were first reported in 1968 by Dr. Reznick et al. in a randomized controlled trial of patients undergoing colonic bypass to reduce hepatic encephalopathy. In this study, cirrhotic patients underwent an internal bypass with an ileosigmoid anastomosis in an attempt to reduce the amount of ammonia produced by the colon, and the terminal ileum distal to the anastomosis was brought out as a mucus fistula. Not surprisingly, the morbidity and mortality for this procedure was quite high, and the authors noted stomal varices with hemorrhage in 15% of patients.

Since its initial description almost 50 years ago, our knowledge of stomal varices has improved, but the current literature still lacks a reliable algorithm for management, and there is disagreement among experts regarding the best approach to acute and chronic variceal hemorrhage. The aim of this chapter is to provide an updated summary of the clinically relevant aspects of stomal varices, and develop a reliable algorithm for the diagnosis and management of variceal bleeding.

Etiology

A stomal varix is an acquired communication between the portal and systemic circulation that occurs due to portal hypertension. The bowel submucosa contains veins that drain into the portal venous system, and because of increased pressure, they develop collateral communications with veins of the abdominal wall. Since this process requires portal hypertension, it is almost always secondary to liver failure, which can occur due to many reasons including Hepatitis C, alcohol abuse, primary sclerosing cholangitis (PSC), or extensive hepatic tumor burden in patients with Stage IV malignancies.

Stomal variceal hemorrhage is usually due to local trauma related to pouching or other components of stoma care, but it can also occur spontaneously due to vessel erosion through the stoma's mucocutaneous border.

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Epidemiology

Most studies describe bleeding stomal varices as rare, with a 1990 review finding only 75 cases in the literature and a 2013 review reporting 235 cases. However, the incidence is likely underreported, and other case series estimate the incidence among patients with liver failure and intestinal stomas to be 5–30%.

Stomal varices are more common in men, but it is unclear if this is due to a specific predisposition to varices, or if it simply reflects the higher incidence of liver disease among males. The most common cause of liver failure among patients with stomal varices is Primary Sclerosing Cholangitis, which can be partially attributed to the relationship between Ulcerative Colitis (UC) and PSC, with many of these patients requiring temporary or permanent stomas. Indeed, the most common abdominal pathology leading to stoma formation in this group is UC (58%), followed by rectal cancer (23%) and cancer of the urinary tract (9%). Of note, the incidence of stomal varices is similar between patients with PSC and other causes of liver failure.

Variceal hemorrhage can be associated with any type of intestinal stoma, including ileostomies, colostomies, urostomies, loop stomas, and defunctionalized stomas or "mucus fistulas". In general, about 70% of variceal bleeds occur from ileostomies, 20% from colostomies, and 10% from ileal conduits.

Clinical Presentation

Refer to Algorithm in Fig. 80.1

Stomal varices usually take time to form, and variceal hemorrhage typically occurs 2–4 years after stoma creation. However, it has also been described in the immediate postoperative period (range 1–480 months).

A. Patients may present with intermittent low-volume bleeding, or they may experience high-volume bleeding with associated anemia. Blood may be bright red or dark purple in color. The bleeding usually occurs from the mucocutaneous junction, and consists of a brisk, non-pulsatile flow. The bleeding can at times be quite dramatic depending on the degree of portal hypertension, and some case reports include descriptions of blood squirting long distances from the skin edge.

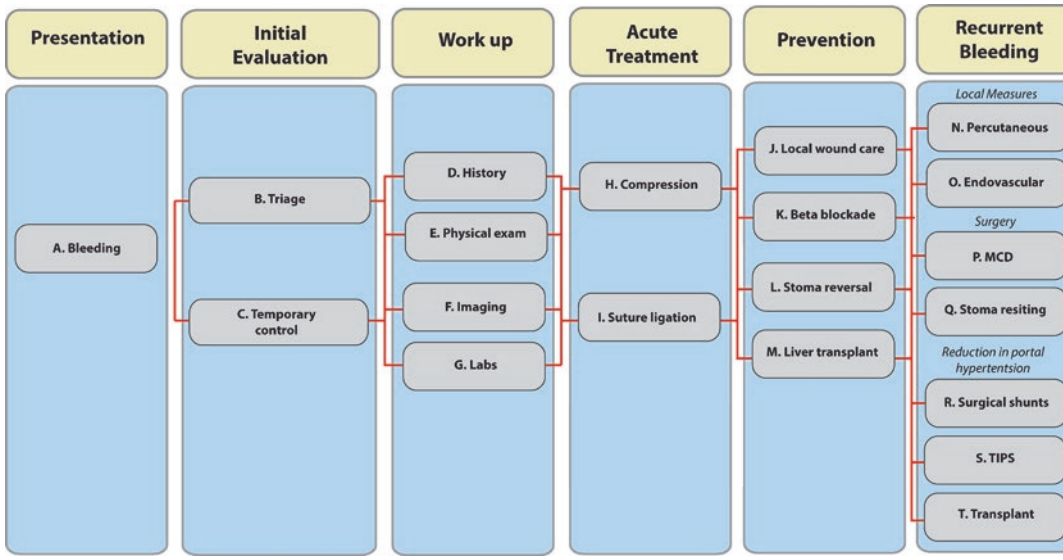

Fig. 80.1 Algorithm for the diagnosis and treatment of stomal varices

Unfortunately, this bleeding is often attributed to the gastrointestinal tract rather than the skin edge, and a lengthy and fruitless evaluation including expensive imaging and pan-endoscopy can occur if the clinician does not remove the stoma appliance and inspect the stoma itself. One case series reported this to be the case in 25% of patients with bleeding stomal varices.

Because of their comorbidities, patients may also present with decompensated liver failure at the time of stomal hemorrhage. Some patients may also describe similar episodes of bleeding in the past, regardless of whether or not the bleeding was previously localized to the stoma.

Evaluation

Triage and Temporary Control of Bleeding

B. Stomal varices should be treated similar to other causes of GI hemorrhage. Specifically, the clinician should ensure that the patient is in a location with sufficient resources such as emergency room, intensive care unit, or operating room, has adequate intravenous access, and receives ongoing resuscitation during the workup. It is important to remember that this patient population tends to have several other comorbidities that require attention, and the clinician should assess the patient's degree of global dysfunction.

C. While more information must be obtained from the patient, the first step is to obtain temporary control of the bleeding with direct pressure applied to the site of hemorrhage. This prevents unnecessary blood loss during a detailed evaluation.

D. History: The clinician should determine the cause of the patient's liver disease, as well as the reason and timing of stoma creation. Medications and comorbidities which may be contributing to coagulopathy and hemorrhage should be reviewed.

E. Physical examination: Patients may exhibit other signs of liver failure including jaundice, cachexia, caput medusae, hepatomegaly, and hepatic encephalopathy. A comprehensive head-to-toe exam is warranted, and it is essential that the primary survey include complete removal of the stoma appliance and a detailed stomal exam, as this can save an expensive workup for other sources of hemorrhage.

Fig. 80.2 Ileostomy with classic appearance of the peristomal skin. (Courtesy of Patricia Roberts, MD)

After removal of the stomal appliance, the clinician will encounter brisk non-pulsatile bleeding from the mucocutaneous junction, typically in a single location. The surrounding peristomal skin often has a bluish discoloration (Fig. 80.2), but this is only present in one-third of patients with stomal hemorrhage.

F. Imaging: Specific imaging is not necessary for patients with stomal hemorrhage.

G. Laboratory tests: Complete Blood Count, International Normalized Ratio, and a Comprehensive Metabolic Panel are warranted to determine the extent of hepatic disease, coagulopathy, and anemia. These values will guide resuscitation.

Treatment

Acute Treatment

Acute bleeding almost always responds to local bedside measures, but recurrent hemorrhage is universal. The best definitive treatment is to correct the patient's underlying portal hypertension, as any measure aimed specifically at the offending vessel is temporary, and new collaterals will form.

H. The immediate goal of bedside intervention is to stop active hemorrhage. The simplest way to achieve this is through direct pressure, often with the assistance of epinephrine-soaked gauze. This results in temporary source control for the majority of patients.
I. When direct compression is unsuccessful, bleeding can be controlled with suture ligation. An absorbable suture is typically used to eliminate the need for future removal. A tapered needle should be employed, with braided and monofilament sutures having equal efficacy. The purchase should go across the mucocutaneous junction in a simple interrupted or figure-of-eight manner.

Prevention of Recurrent Bleeding

J. Pouching issues often contribute to local trauma, and the only effective mechanical measure for the prevention of recurrent bleeding is modification of the stoma appliance. This often involves a stoma nurse to help the patient get re-fit for a new appliance with less potential for stomal trauma.
K. Beta-blockade is well-described for the primary prevention and treatment of bleeding esophageal varices, and it has also been described for the prevention of recurrent stomal hemorrhage with mixed results. A single case series reported long-term success in three patients treated with propranolol, with dosage aimed at a 25% reduction in heart rate. At this point, there is inadequate evidence to support routine use of beta-blockade, but it is certainly an attractive alternative to larger interventions.
L. The stoma's utility should also be determined, and if the stoma is no longer necessary, the surgeon may consider stoma takedown as an effective means to prevent future hemorrhage, as this effectively interrupts the portosystemic communication. Of note, many patients will have comorbidities that limit their ability to tolerate an elective surgery. Their surgical history is also important, as loop stomas are easier to reverse than end stomas, and a hostile open dissection in a coagulopathic patient may not be the best path to choose.
M. In addition to consideration for stoma reversal, the surgeon must also assess the patient's candidacy for liver transplant. This is a complex decision based on the patient's severity of disease and overall prognosis, and liver transplant should not be considered solely for the treatment of variceal hemorrhage.

Treatment of Recurrent Bleeding

When bleeding recurs, repeat suture ligation can be employed, and there are several more aggressive local measures, which will be described below. The best approach to recurrent bleeding is not well-defined, and existing literature is mostly in the form of small case series. Many of these studies are old, without much activity in the last 20 years. As stated before, local measures have only temporary efficacy because the main underlying issue is portal hypertension. It is also important that the clinician cater the treatment to the patient, as they may present with fulminant liver disease and a limited liver-specific life expectancy, in which situation smaller interventions are preferable. Most patients will succumb to their liver disease prior to experiencing life-threatening stomal hemorrhage.

Local Measures

N. Percutaneous interventions: Overall, percutaneous treatments are safe with reasonable short-term outcomes, but many complications have been reported, and case series are small. In addition, multiple applications may be necessary to achieve the desired effect.

Injection sclerotherapy has been well-described for the local obliteration of stomal varices. It can be done to treat acute bleeding or to prevent recurrent hemorrhage after temporary hemostasis. In general, a sclerosing agent is injected directly into the offending vessels, either through the stomal mucosa or

percutaneously through the peristomal skin. In some newer series, ultrasound guidance has been employed with good short-term outcomes. Several sclerosing agents have been described, similar to what is used for hemorrhoids, and there is no evidence that one sclerosing agent is superior. Success with this technique is modest, and described solely in small case series. Skin necrosis and stomal stenosis have been reported with this technique as well.

Percutaneous embolization has also been described using both ultrasound and fluoroscopy. A combination of endovascular coils and Histoacryl glue is typically employed. Of note, coil migration has been reported. This technique is newer and has less supporting evidence than sclerotherapy.

O. Endovascular interventions: Angiography with endovascular coil embolization has also been described in small series, and is a viable option for patients who continue to bleed, and are not candidates for surgical intervention. Sclerosing agents can also be injected in this manner.

Surgery

P. Mucocutaneous disconnection (MCD) was popularized in 1988 by Drs. Beck, Fazio, and Grundfest-Broniatowski. This technique interrupts the portosystemic collaterals surgically without the need to re-site the offending stoma. The authors conceded that new collaterals would eventually form in most patients, but opined that this technique often results in a sustained period of hemostasis, which is adequate for most patients with advanced liver disease.

The original description of this technique included an incision at the mucocutaneous border, direct variceal ligation down to the level of the anterior rectus sheath, and repeat maturation of the stoma in the same location. The authors reported universal technical success with roughly 200–300 ml of blood loss. In this author's experience, a helpful caveat is to include a small (1 mm) rim of skin so as to approach slightly proximal from the site of bleeding, and have a bipolar energy device available. Great care must be taken not to injure the adjacent bowel, and subcutaneous dissection should remain very close to the serosa if possible.

Q. Stomal re-siting is listed for historical purposes, but is generally not a preferable technique, as it does not provide any additional hemostasis or decrease in rebleeding rates compared to MCD. However, if there is a heavily symptomatic parastomal hernia, relocation may be necessary. If the stoma's location and profile are suboptimal, and this is contributing to local trauma and hemorrhage, then relocation is also reasonable.

Reduction in Portal Hypertension

Since portal hypertension is the true cause of stomal varices, techniques aimed at reducing portal pressures have the lower reported rates of rebleeding.

R. Surgical shunts: In the 1980s, the most commonly described method of portal decompression was surgical portosystemic shunting, including mesocaval and portacaval splenorenal shunts. Of note, rebleeding was very uncommon after these procedures, and usually occurred when the shunts occluded. However, these are very morbid procedures, and alter the anatomy making future transplantation more difficult, so they have a very limited role now that Transjugular Intrahepatic Portosystemic Shunting (TIPS) is available.

S. Transjugular Intrahepatic Portosystemic Shunt: TIPS has long been known to reduce portal hypertension without requiring major abdominal surgery or altering the patient's anatomy. It was first described for the treatment of variceal hemorrhage in the early 1990s, and it has become increasingly prevalent since then. When technically feasible, TIPS is associated with nearly-universal short-term success, and the lowest reported

rate of rebleeding (20% for TIPS vs. 80–90% for local measures).

Rebleeding can occur after TIPS, typically due to shunt occlusion and recurrent portal hypertension, so patients should be monitored closely for recurrent symptoms. TIPS can also worsen hepatic encephalopathy, so patients must be selected carefully, and TIPS is not appropriate for all patients with variceal hemorrhage.

T. Liver transplantation is discussed above (M), and is an effective means of reducing portal hypertension as well.

Summary

Stomal varices can result in significant hemorrhage. Local hemostatic measures are usually effective, but are associated with high rates of recurrent hemorrhage. Surgeons should be familiar with the technique of mucocutaneous disconnection for severe, recurrent bleeding. The most durable way to prevent rebleeding is to reduce portal hypertension, and TIPS is slowly emerging as the most promising non-invasive technique for portal decompression. Clinicians must balance interventions with the patient's liver-specific prognosis, as patients frequently succumb to their liver disease rather than to stomal hemorrhage.

Suggested Reading

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