Stomal Varices: A Rare Cause of Stomal Hemorrhage. A Report of Three Cases

Muhammad A. Kabeer, MRCS; Lucina Jackson, MB, MRCPI, PhD; Adam L. Widdison, DM, FRCS, PGCE; Giles Maskell, MA, MRCP, FRCR; and Joseph Mathew, FMCPATH, FRCPATH

Stomal varices secondary to portal hypertension are a rare but potentially fatal cause of hemorrhage. Management, determined by the site of the bleeding, centers on preventing additional bleeds and may include providing local pressure, applying silver nitrate, injection sclerotherapy, suture ligation of the bleeding point, and/or the placement of transjugular intrahepatic portosystemic shunts and refashioning the stoma. Two patients (60- and 69-year-old women) had panproctocolectomy for inflammatory bowel disease and presented at the authors’ hospital with bleeding from the ileostomy 1 and 19 years, respectively, following the creation of their stomas. A third patient (a 72-year-old man) bled from an end colostomy following an abdominopereineal resection for Duke’s C rectal adenocarcinoma performed 3 years previous. All three patients had recurrent admissions for stomal bleeding and stomal varices secondary to portal hypertension and were initially treated with local measures (pressure, silver nitrate, and suture ligation). Two had undergone revision of their stomas prior to current treatment. One patient responded to local treatment but later died due to liver failure, one stopped bleeding after transjugular portosystemic shunt placement, and one died from metastatic cancer. Clinicians should maintain a high index of suspicion of stomal varices in patients with underlying liver disease who present with recurrent stomal bleeds and provide appropriate treatment to stop active bleeding and reduce portal venous pressure.

KEYWORDS: stomas, case study, portosystemic shunts, gastrointestinal hemorrhage, portal hypertension


Stomal bleeding is usually caused by trauma,12 skin excoriation, and stomal ulceration1 and typically controlled by local measures that include local pressure, silver nitrate application, injection sclerotherapy, and suture ligation of the bleeding point.2,6 Hemorrhage from stomal varices is a rare but potentially fatal cause of stomal bleeds. To increase clinician awareness about this complication, three recent cases involving patients with recurrent stomal bleeding secondary to stomal varices are described and management strategies are discussed.

Literature Review

Physiology. Variceal development at sites of portosystemic anastomoses is usually associated with portal hypertension.5 7 Varices typically develop at the gastro-esophageal junction. Varices elsewhere (ectopic varices) can develop anywhere along the entire gastrointestinal tract; they have been reported at unusual sites such as the gallbladder, urinary bladder, uterus, vagina, umbilicus, and retroperitoneum.10 Ileostomies, colostomies, and ileal conduits11,14 provide additional sites for the formation of what are called stomal varices.5

Mr. Kabeer is a Research Registrar, Department of Histopathology; Dr. Jackson is a Consultant Gastroenterologist, Department of Medicine; Mr. Widdison is Consultant, Department of Surgery; Dr. Maskell is a Consultant, Department of Clinical Imaging; and Dr. Mathew is Consultant, Department of Histopathology, Royal Cornwall Hospital, Truro, UK. Please address correspondence to: Dr. Joseph Mathew, Department of Histopathology, Royal Cornwall Hospital, Treliske, Truro, Cornwall, TR1 3LJ, UK; email: joe.mathew@rcht.cornwall.nhs.uk.
Stomal varices were first described in 1968. All variceal tissue is friable and bleeds easily if traumatized. Studies have shown that bleeding can occur in up to 27% of patients with stomal varices and is potentially fatal. Case series data suggest that the interval between fashioning the stoma and the first bleed ranges from 5 months to 29 years (average 48 months). The reported mortality rate from a stomal variceal bleed is 3% to 4% — a relatively low rate compared with the 20% mortality associated with esophageal variceal bleeds.

**Diagnosis.** Diagnosing stomal varices is challenging. Clinicians should be highly suspicious of stomal varices in patients with recurrent stomal bleeding. Assessment of patients’ coagulation status, liver function, and stage of liver disease are important. Liver disease leads to impaired vitamin K production and prothrombin activity (ie, clotting) is impaired. Portal hypertension, a result of liver disease (eg, cirrhosis, extrahepatic portal vein occlusion, intrahepatic veno-occlusive disease, and occlusion of the main hepatic veins) leads to the formation of varices at sites of portosystemic anastomosis. The site of the bleeding (eg, the lumen, the mucosa, or the muco-cutaneous junction) is a factor in determining immediate and future management strategies. The stoma may look normal or have characteristic bluish surrounding skin with excoriation and streaks of vessels possibly visible around the stoma (see Figure 1). The mucosa may appear red and engorged in the presence of hemorrhaging and narrowed by annular scarring due to repeated bleeds. An endoscopy is needed to confirm that other conditions such as arteriovenous malformations, polyps, or ulcers are not cause for concern. Selective mesenteric angiography, computed tomographic angiography, endoscopic ultrasonography, and magnetic resonance angiography help diagnose portal hypertension.

**Treatment.** Treatment is directed at preventing further bleeding and ranges from local measures to portosystemic shunt placements and liver transplantation. Local measures are usually temporary; the underlying cause of the bleed is not treated.

Local measures include adjusting the face plate to reduce local trauma, applying firm pressure to the bleeding point with or without epinephrine-soaked gauze (1:100,000), gel foam, chemical cautery (eg, silver-nitrate), injection sclerotherapy (using 0.5% to 1% polidocanol, 5% phenol in almond oil, sodium tetradecyl sulphate, absolute ethanol, and strong saline solution), correcting coagulation abnormalities, and suture ligation of the bleeding point. All of these strategies have been tried (and may be repeated) with varying rates of success. Each carries a variable risk of peristomal ulceration, infection, stricturing, and necrosis depending on patient factors such as nutrition, disease state, and technique used. The patients should be referred to a wound and ostomy care nurse for education regarding preventive measures and immediate treatment.

**Figure 1.** Stoma showing characteristic visible peristomal vessels and narrowing due to fibrosis from repeated bleeds.

**KEY POINTS**
- Stomal bleeding, especially if recurrent and in patients with liver disease, may be caused by stomal varices and may be life-threatening.
- Diagnostic studies to determine the presence of portal hypertension are needed to help guide optimal treatment.
- As occurred with the patients described, most patients are managed conservatively (eg, by applying pressure) or surgically with placement of a transjugular portosystemic shunt.
Although mucocutaneous disconnection\(^1\) and stoma re-siting have been suggested,\(^2,4,15\) these approaches did not stop the bleeding in the patients described at the author’s facility.

Beta-blockers also are used as an attempt to lower the portal pressure but their efficacy in preventing subsequent bleeds has not been evaluated.\(^15,24\)

Percutaneous transhepatic embolization of stomal varices also has been used successfully, although the long-term efficacy of this procedure is unknown. Complications of embolization include bleeding, bile leak, liver trauma, portal vein thrombosis, and bowel infarction.\(^5,12,16,24\)

Surgical portosystemic shunts present a definitive treatment option to reduce the portal pressure\(^4,23,30,31\) but are associated with significant morbidity and mortality\(^7\) and may compromise potential liver transplant at a later stage.\(^15,25\) Hence, they should be performed only in carefully selected patients.\(^7\)

A transjugular intrahepatic portosystemic shunt (TIPSS) is currently the treatment of choice for portal hypertension.\(^10,32-37\) First used in 1989, TIPSS involves the creation of a portosystemic shunt (bypass) between the hepatic vein and the intrahepatic portal vein using a transjugular approach; thereby, reducing portal venous pressure. Under direct x-ray vision using a C-arm, a stent is placed at the site and dilated until a gradient between the systemic and portal pressures falls to \(<12 \text{ mm Hg}^\text{10}\). The procedure can be performed even in patients who are Child-Pugh class C (see Table 1).\(^21\) A success rate of up to 90% for TIPSS placement has been reported; TIPSS was successful in treating and preventing re-bleeding in one of the patients presented herein. Most authors presenting on the subject consider a post-procedure reduction of portocaval pressure of \(\leq 12 \text{ mm Hg}\) or a 20% reduction to be sufficient to prevent variceal rebleeding.\(^10,32\)

The incidence of early life-threatening complications after TIPSS is 1% to 2% and hepatic encephalopathy procedures are less commonly performed than surgical shunts.\(^10\)

Although the occlusion rate following this procedure is approximately 50% at 1 year, it can be reduced by the use of polytetrafluoroethylene (PTFE)-covered stents.\(^10,26\) Regular surveillance with portography or Doppler ultrasound is indicated.\(^32,38\)

**Case Reports**

**Patient 1.** A 60-year-old woman presented with recurrent, occasionally heavy ileostomy bleeding that appeared to be of venous and mucosal origin. She had undergone total proctocolectomy and ileostomy in 1997 for ulcerative colitis. Between 1998 and 2002, she was admitted to the hospital six times with stomal hemorrhage. She was managed initially with under-running sutures and silver nitrate application to the tissue around the stoma but later underwent excision and revision of her ileostomy. The histology report of her stomal stump showed dilated vessels.

When examined on presentation in 2002, 5 years after the initial procedure, her stoma was stenosed due to scarring, appeared cyanosed, and the surrounding skin was bluish and surrounded by streaks of tiny blood vessels. Further investigation and an ultrasound scan revealed an enlarged liver with some nodularity. A mesenteric angiogram showed an occluded superior mesenteric vein and lower esophageal and stomal varices. A portogram (portal venogram) showed signs of portal hypertension and a transjugular liver biopsy confirmed micronodular cirrhosis; she was in Child-Pugh Class A (see Table 1) for liver disease. Contrast-enhanced CT of her abdomen revealed large stomal

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**TABLE 1**

**CHILD-PUGH CLASSIFICATION**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>A</th>
<th>B</th>
<th>C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Albumin (g/dL)</td>
<td>&gt;3.5</td>
<td>3.0–3.5</td>
<td>&lt;3.0</td>
</tr>
<tr>
<td>Bilirubin (µmol/L)</td>
<td>&lt;25</td>
<td>25–40</td>
<td>&gt;40</td>
</tr>
<tr>
<td>Prothrombin (sec &gt;normal)</td>
<td>&lt;4</td>
<td>4–6</td>
<td>&gt;6</td>
</tr>
<tr>
<td>Prothrombin (%)</td>
<td>&gt;64</td>
<td>40–65</td>
<td>&lt;40</td>
</tr>
<tr>
<td>Ascites</td>
<td>None</td>
<td>Controlled</td>
<td>Refractory</td>
</tr>
<tr>
<td>Encephalopathy</td>
<td>None</td>
<td>Minimal</td>
<td>Advanced</td>
</tr>
</tbody>
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This classification is used to define the severity of liver disease and the following parameters are taken into consideration. Each positive parameter is given 1 point in category A, 2 points in category B, and 3 points in category C. Child-Pugh Classes are based on total points scored: A= 5≤7 points; B= 8≤10 points; C=≥11
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varices anastomosing with cutaneous vessels (see Figure 2), a dilated splenic vein with splenomegaly, and a possible portal vein stenosis at the hilum of the liver. The patient underwent a transjugular intrahepatic portosystemic shunt (TIPSS) placement in 2002 and her bleeding stopped.

**Patient 2.** A 72-year-old man had an abdominoperineal resection for a Duke’s C rectal adenocarcinoma in 2002. He presented through the emergency department in September and December 2005 with massive episodes of bleeding at the muco-cutaneous junction of the stoma. The bleeding stopped with pressure to the stoma with epinephrine-soaked gauze and he was managed conservatively. He also was found to have abnormal liver function tests — a CT scan showed extensive hepatic and pulmonary metastasis and prominent stomal varices. A colonoscopy revealed only mild mucosal inflammation. He was terminally ill and the bleeding stopped with pressure on each occasion. He died later from his metastatic disease.

**Patient 3.** A 69-year-old woman presented with a massive bleed from the lumen of her ileostomy in April 2006, 19 years after the initial procedure. She had a total proctocolectomy and ileostomy in 1987 for ulcerative colitis. An earlier CT had shown stomal varices with signs of portal hypertension and a subsequent liver biopsy showed sclerosing cholangitis with micronodular cirrhosis; she was in Child-Pugh Class B (see Table 1) for liver disease. An ileoscopy confirmed peristomal varices. The bleeding stopped with pressure to the stoma with epinephrine-soaked gauze and she was discharged. She had no further bleeds from her stoma but died in July 2006 due to liver failure.

**Discussion**

These three patients had typical presentation of liver disease and stomal hemorrhage. All had been managed initially with local measures and responded well. One of these patients had multiple episodes of stomal hemorrhage and each time responded to local measures of pressure to the stoma site, under-running sutures, and silver nitrate application; however, she had revision of her stoma and later had TIPSS placement. Two other patients responded to local measures to stop stomal bleed but were terminally ill and did not undergo any further interventions. The procedures used are well recognized in the literature but whether one would have been better than the other is a matter of debate as evidence in this regard is lacking. Furthermore, performing TIPSS earlier in the management of the first patient would have prevented subsequent readmissions with bleeding.

**Conclusion**

Stomal varices are a rare cause of stomal hemorrhage. The bleeding is recurrent and occasionally hemodynamically significant. Patients with underlying liver disease presenting with recurrent stomal bleeds should be considered at risk. Treatment includes local measures to stop active bleeding and interventions to reduce portal venous pressure such as TIPSS. Guidelines for the optimal management of these patients are not available. Further research to evaluate each of the measures suggested to weigh their risks and benefits is warranted.
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Acknowledgment

The authors acknowledge the consent of one patient to discuss the case. The other two patients are deceased. - OWM

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