Special Considerations in the GI Bleeding Patient

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Abstract

Keywords

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- radiation proctitis
- rectal varices

Lower gastrointestinal bleeding (LGIB) is an increasingly common problem in patients with comorbid medical conditions that place them at higher bleeding risk. This discussion of some special considerations in the GI bleeding patient encompasses an overview of the elderly patient, and selects comorbid conditions that place patients at higher risk of developing intestinal bleeding. The discussion lends itself to exploring the challenges of and new advancements in anticoagulation therapy. Radiation induced proctitis and rectal varices as sources of LGIB will also be addressed.

The incidence of lower gastrointestinal bleeding (LGIB) has an annual estimate of 20 per 100,000 patients and increases in proportion to age. This discussion of special cases in GIB, therefore, inevitably calls for a discussion of the elderly population and the comorbid conditions that place these patients at higher risk, which naturally leads into discussing the challenges of, and new advancements in anticoagulation therapy. Radiation induced proctitis and rectal varices as sources of LGIB will also be addressed.

GIB in the Elderly

With increasing age, the propensity for a GIB to localize to a lower GI tract source increases. Therefore, elderly patients presenting with GIB should undergo a swift evaluation to rule out a source proximal to the ligament of Treitz and undergo a preferential evaluation for a colonic source of bleeding. Understanding that the causes of LGIB in the elderly are likely diverticular or malignant in nature, will assist in narrowing the differential diagnoses.

Several physiologic derangements can complicate the care of the elderly patient. In the context of a discussion about GIB, it is important to point out that the elderly patient is both at greater risk of thrombotic complications from chronic vasculopathy and vascular stasis, and also at greater risk of bleeding complications from malnutrition, liver dysfunction, or from medication use. The complexity of the elderly patient's physi-

ology is further compounded by progressive renal insufficiency with its predisposition to inconsistent clearance of drugs, polypharmacy plaguing makes many elderly patients to run into problems of unforeseen drug interactions, and the overall physical and psychological frailty that the elderly patients are at higher risk of complications from medications, such as anticoagulation, drug interactions, and noncompliance.

It is imperative that a thorough history to be solicited from reliable caretakers to identify pre-existing cardiopulmonary disease, liver disease, renal failure, substance, and medication use. A detailed surgical history is also useful in considering vascular etiologies of bleeding, including ischemic bowel or vasculocenteric fistula.

The strongest predictors of mortality in LGIB are advanced age, intestinal ischemia, and patient comorbidities.¹ This speaks to the fact that the morbidity and mortality, associated with GIB, are often related not to blood loss, but the end organ damage brought on by hemodynamic instability, infectious complications, and the exacerbation of existing comorbidities. Advanced age and increased comorbidities tend to go hand in hand; therefore, the elderly are doubly at risk of developing complications from a GIB compared to their younger counterparts. In addition, as the population ages, there has been a rise in chronic conditions, such as arrhythmias, venous thromboembolism, and orthopedic pathologies, requiring major procedures which often necessitate systemic anticoagulation. The management of these patients can become complex, especially with the

introduction of newer anticoagulant classes of medications which are starting to replace the more familiar vitamin K antagonists (VKA) and heparin products.

Anticoagulation: VKA

The most frequently used long-term anticoagulation is the VKA and warfarin. VKA-treated patients are at increased risk of bleeding, the most common site being the GI tract. The first step in treatment of patients presenting with an acute bleeding episode, after withholding the VKA, is reversal.² While a therapeutic goal International Normalization Ratio (INR) of 1.5 to 2.5 should be obtained for most anticoagulation indications, there is still some controversy on the urgency with which to administer products for reversal, and how it should relate to timing of endoscopic interventions. Some authors recommend that endoscopy proceeds regardless of coagulopathy, but the general consensus is to postpone endoscopic intervention, if possible, until coagulopathy is reversed. A national audit from the United Kingdom showed that elevated INR or prothrombin time were associated with failure to achieve endoscopic hemostasis in nonvariceal active upper GIB patients.³ While a similarly designed study is not available for LGIB, it is reasonable to extrapolate the principle of achieving normalization of coagulopathy prior to intervening for active bleeding in the LGIB patient.

VKA Reversal Options

Reversal of VKA can be achieved with vitamin K, fresh frozen plasma (FFP), prothrombin complex concentrate (PCC), and recombinant activated factor VII. Vitamin K promotes the hepatic synthesis of clotting factors II, VII, IX, and X. Intravenous infusion of 5 to 10 mg vitamin K leads to normalization of INR within 24 hours.⁴ The use of vitamin K produces a sustained normalization of coagulation, and should be utilized in conjunction with other factors to bring about more immediate reversal.

FFP consists of plasma taken from whole blood frozen within 8 hours of collection, and contains all coagulation factors in physiologic concentrations, including the vitamin K-dependent clotting factors. Intravenous infusion of 15 mL/kg leads to immediate effect in 10 minutes, and full reversal by 9 hours. The major disadvantages associated with FFP are similar to those of any blood product, including the time needed for cross matching and thawing the product, and the volume of fluid necessary for administration. It also does not produce a sustained level of normalization, as once the coagulation factors are consumed, there may be a return to anticoagulation. Therefore, ongoing transfusions may be required which compounds the volume of fluid received by the patient.

PCC is an inactive complex of factors II, IX, and X, with variable amounts of factor VII, derived from cryoprecipitate supernatant of plasma pools. PCC dosing is based on the concentration of factor IX, and an intravenous administration of 25 to 50 IU of factor IX/kg can be rapidly achieved in 20 to 30 minutes. Its use has been shown to be superior to FFP for rapid reversal, achieving INR below 1.3 within

30 minutes.⁵ A small but acceptable risk of thromboembolism has been identified.⁶

Recombinant factor VII has the theoretic advantage of rapidly correcting coagulopathy via the extrinsic pathway, but its use has shown in small studies to result in unacceptably higher risk of thromboembolism. Its routine use should be avoided until larger studies can demonstrate its safety.

In hemodynamically significant GIB, rapid reversal of coagulopathy should be initiated with the concomitant administration of IV vitamin K and PCC. Monitoring for effect can be done by repeating INR levels 20 to 30 minutes postadministration, and a second dose of PCC was given, if needed. Ideally, endoscopy can be deferred until INR is below 2.5 to minimize risk of uncontrolled hemorrhage, and procedure-exacerbated bleeding.

For hemodynamically stable patients, administration of vitamin K and allowing the plasma levels to "drift" may be sufficient to reverse coagulopathy enough to proceed with endoscopy on a semielective basis. For patients who have a supratherapeutic INR, PCC, or FFP can be infused and timed to optimize effect for endoscopy. An identifiable anorectal source of minor bleeding may warrant outpatient management of VKA dosing with follow-up monitoring of INR and administration of vitamin K if INR exceeds five. Close monitoring and a low threshold for endoscopic evaluation must be maintained.

Anticoagulation: Direct Oral Anticoagulants (DOACs)

Newer anticoagulant therapies, DOACs, have been emerging, which have more predictable pharmacokinetics that allow fixed dosing, obviating the need for drug level monitoring, and causing less adverse interactions with other drugs. DOACs have been approved for the same therapeutic use as VKAs to prevent embolic complications in atrial fibrillation and in the treatment of thromboembolic disease. The three more widely adopted DOACs to be discussed are the direct thrombin inhibitor dabigatran (Pradaxa, Dabigatran), and the activated factor X inhibitors rivaroxaban (Xarelto, Rivaroxaban) and apixaban (Eliquis, Apixaban).

Three pivotal trials comparing DOACs to warfarin have been performed. RE-LY (randomized evaluation of long-term anticoagulation therapy) was a multicenter randomized trial comparing two fixed doses of dabigatran to warfarin in patients with atrial fibrillation. The lower, 110 mg, dose of dabigatran was associated with similar rates of stroke and systemic embolism and lower rates of hemorrhage compared to warfarin. The higher, 150 mg twice daily, dose of dabigatran was associated with lower rates of stroke and systemic embolism and similar rate of major hemorrhage as warfarin but with a higher rate GIB.⁷ Major GIB occurred in the lower GI tract in 53%, and the upper GI tract in 47%, compared to 25% lower and 75% upper in the warfarin arm. The higher rate of major GI hemorrhage coinciding with the lower rate of intracranial hemorrhage has been hypothesized to be due to the incomplete absorption of DOACs across the GI tract and increased intraluminal drug availability for topical activity, whereas warfarin is almost completely absorbed from the gut mucosa and has no

topical anticoagulant activity.⁸ This study identified increased risk of LGIB using the higher dose, which will help to guide management of patients with a known higher risk of LGIB, to either avoid this particular DOAC or to use the lower dose.

ROCKET-AF (rivaroxaban once daily oral, direct factor xa inhibition, compared with vitamin k antagonism for prevention of stroke and embolism trial in atrial fibrillation) was a multicenter, randomized trial that studied the effectiveness of rivaroxaban to prevent stroke or systemic embolism in patients with nonvalvular atrial fibrillation. It demonstrated rivaroxaban's noninferiority to warfarin. There was an observed higher GIB rate in the rivaroxaban arm, but lower rates of intracranial bleeding, and, hence, lower overall critical bleeding complications.⁹ Similar to dabigatran, then, in patients with a known GI pathology that places them at higher risk of GIB, may be steered away from the use of rivaroxaban.

ARISTOTLE (apixaban for reduction in stroke and other thromboembolic events in atrial fibrillation) was a multicenter, randomized trial comparing the factor Xa inhibitor, apixaban, to warfarin in reducing stroke and systemic embolism in patients with atrial fibrillation, and at least one additional risk factor for stroke.¹⁰ Rivaroxaban was superior to warfarin in preventing stroke and systemic embolism, caused less fatal, intracranial, and GI hemorrhage, and resulted in lower overall mortality.¹¹

While none of these trials were head-to-head comparisons, until such trials are completed, their conclusions can help infer guidelines for management decisions. For instance, a patient presenting with an acute GI bleed on high dose dabigatran can be managed by either decreasing the dose, switching to warfarin, or switching to apixaban with its demonstrated decreased risk of GIB as compared to warfarin.

Management of GIB in Setting of DOAC Use

Major GIB should be managed in an inpatient setting, generally with discontinuation of the DOAC, as well as any other anticoagulant or antiplatelet agents and NSAIDs (nonsteroidal anti-inflammatory drug). Laboratory evaluation of PT, PTT and thrombin time can be used to assess the qualitative effect of dabigatran or rivaroxaban. There are no effective reversal agents for DOACs; however, they are reliably cleared from the system over 12 to 24 hours. With supportive care and resuscitation, withholding the agent will allow the system to re-establish normal coagulation to allow semielective endoscopy. In the case of the hemodynamically stable patient, presenting with either occult bleeding or minor anorectal bleeding, an elective endoscopy may be performed with continuation of the DOAC, just as for patients on VKAs.

If a patient is hemodynamically unstable, emergent endoscopic or angiographic intervention is priority. The tools available for controlling any GIB apply to patients being treated with DOACs, including thermal coagulation, balloon tamponade, clip application, and angiographic embolization. The use of topical agents, such as thrombin and fibrin, are not well delineated in patients being treated with DOACs.

There are several systemic pharmacologic measures to be pursued concomitantly with endoscopic or angiographic

intervention. If the most recent intake of the DOAC is known, within 2 to 3 hours of ingestion, gastric lavage, or activated charcoal may be used to bind the agent and prevent further absorption. Tranexamic acid's antifibrinolytic activity may attenuate DOAC-associated mucosal bleeding and could be used in an unstable bleeding patient. Likewise, the use of PCC or recombinant factor VII may be considered in cases of life-threatening bleeding, but the risk of thromboembolic complications must be weighed against the bleeding risk in less dire situations. In the setting of renal failure, dabigatran may be effectively removed with hemodialysis. Specific antidotes for DOACs include idarucizumab, a humanized antibody fragment that binds to dabigatran, and andexanet, an inactive factor Xa that binds to Xa inhibitors to inactivate them. Both idarucizumab and andexanet have shown promising results in clinical trials. Ciraparantag, a molecule that non-specifically binds to all DOACs and heparin products is currently in development.

Bleeding refractory to endoscopic, angiographic, or pharmacologic interventions surgery remains the final therapeutic option. In this setting, there is no role for minimally invasive approaches, and a laparotomy should be the initial approach. It is critical to maintain clear communication with the endoscopist or interventional radiologist, so as to maximize chances to localize the bleeding during operative exposure. Any sites of active bleeding discovered on endoscopy that cannot be controlled should be tattooed, or on angiography, a catheter left in place.

Resuming Anticoagulation

Reinstitution of anticoagulation will depend largely on the severity of the bleeding episode and the extent of intervention required to obtaining hemostasis. This is an area not extensively studied, but the existing data support the resuming of anticoagulation versus discontinuing it, as it improves survival of the underlying disease process. Resuming anticoagulation within 7 days of a GIB is associated with an increased risk of rebleeding, and waiting for greater than 30 days is associated with an increased risk of thromboembolic complications. Therefore, the ideal time to resume anticoagulation seems to be somewhere in between. In the absence of evidence and consensus, it is important for the individual provider to take into account the comorbidities that place the patient at higher risk of bleeding versus the risk of thromboembolism.

A consideration may also be given to the extent and invasiveness of the procedure required to obtain hemorrhage control. Minor endoscopic interventions (clipping, fulguration, and injection), at lower risk of rebleeding, may be followed by observation and reinstitution of anticoagulation sooner, within 7 days. Major endoscopic or surgical interventions (large polypectomy and segmental resection) may be followed by a longer period of observation off anticoagulation greater than 7 days.

In resuming anticoagulation, it is important to note that the use of DOACs have significantly impacted the time course of treatment. Traditionally, with the use of VKAs, it was necessary to bridge to therapeutic doses with unfractionated

heparin, or low molecular weight heparin, which may take several days. Because DOACs reach therapeutic levels within 12 to 24 hours, their use not only shortens length of stay but also could increase the risk of rebleeding in a shorter time course. These situations should place the physician at a higher level of alertness in discharging a patient on anticoagulation after resolution of a GIB.

In cases where the bleeding site could not be adequately identified or treated, risk factor modification may involve discontinuing noncritical agents, such as NSAIDs or prophylactic antiplatelet therapy, decreasing the dose of DOAC, switching to a different DOAC, or switching to warfarin. Each of these options carries unique rebleeding or thrombotic risks and must be tailored to the patient's situation.

Anticoagulation for Mechanical Heart Valves

A special consideration is given to patients who are anticoagulated for mechanical heart valves. The highest risk of complications is assigned to those with valve implantation within 6 months, valve in the mitral position, multiple valves, or history of prior stroke. The risk of and mortality associated with thrombotic complications are high with these patients, and minimizing interruption of anticoagulation is preferred. During the active bleeding phase, use of PCC in a patient with a mechanical valve has been under scrutiny, with the concern that thrombotic risk would be higher with its administration. However, in life-threatening bleeding episodes, the use of PCC over FFP is the current recommendation by the ACC/AHA.⁸ Initiating heparin bridge therapy as early as 72 hours after intervention and resuming full anticoagulation within a week is recommended.

Radiation Proctitis

Radiation proctopathy is a common complication of pelvic radiation delivered usually for the treatment of pelvic malignancy. Its incidence ranges from 5 to 65% of patients undergoing pelvic radiation. Early radiation changes commonly occur around the time of treatment and are self-limited. Late effects occur over the longer term, months to years, after cessation of treatment and can lead to stenosis due to fibrotic changes, fistulous disease, or bleeding from telangiectasia. Symptoms include rectal urgency, incontinence, pain, mucus discharge, and rectal bleeding. While the vast majority of patients undergoing pelvic radiation therapy experience rectal bleeding within a year of therapy, the vast majority resolve on their own. Only a minority of patients goes on to develop chronic radiation proctitis and treatment can be difficult, given the refractory nature of the disease. Treatment consists of medical therapy, endoscopic procedures, and operative interventions.

Medical Therapies

Formalin causes chemical cauterization when exposed to mucosal surfaces, and has been successfully used to stop bleeding associated with radiation proctopathy. Four percent

formalin can be applied as irrigation, alternating with saline flushes to avoid irritating the anoderm, or soaked on a cotton-tipped applicator, and applied via proctoscopy. Formalin is particularly useful in treating hemorrhagic proctitis, the success of which is dependent on localizing and targeting the bleeding site. A small risk of rectal necrosis, formation of fistulae, pelvic sepsis, or late strictures exists, and may be compounded in radiation-damaged tissue. These properties make formalin, the ideal treatment for active GIB or bleeding that has failed to respond to the other forms of therapy. Its use is less ideal in addressing other chronic symptoms associated with radiation proctopathy.

Anti-inflammatories have been used as first-line agents in radiation proctitis, based on their success in the treatment of proctitis associated with inflammatory bowel disease. These include 5-aminosalicylic acid (5-ASA), metronidazole, vitamin A, and steroids. The anti-inflammatory activity of 5-ASA initially showed some promise in symptom management, but mixed outcomes in subsequent studies have raised questions about the efficacy of these treatment options.¹² Oral metronidazole, when used in conjunction with 5-ASA products and steroid enemas, has shown to lower rates of rectal bleeding and severity of proctitis.¹³ Vitamin A's antioxidant properties have been used to attenuate the oxidative tissue damage induced by radiation. In a small placebo-controlled trial, symptoms improved in the study arm.¹⁴ Steroid enemas are often used in the treatment of ulcerative colitis, and has also been used in the setting of radiation proctitis. A comparison of hydrocortisone and betamethasone suggested no significant advantage of one over the other, although hydrocortisone was better tolerated.¹⁵

Sucralfate has been studied both as oral and enema formulations, and acts as a physical barrier protection of the mucosal surfaces with radiation damage. Better results and side effect profiles were shown with the use of sucralfate enemas, delivered as 20 mL aliquots of 10% concentration twice daily.¹⁶

Short-chain fatty acid (SCFA) delivered as enemas have shown to effectively treat diversion colitis, and its use has been extended to the treatment of radiation proctitis. SCFA is delivered directly into the rectum in 60 mL twice daily doses. Its effect is largely in providing nutrient support to the damaged rectal mucosa and improving mucosal ischemia through its vasodilatory effects. A small randomized study did not show significant effect on symptoms,¹⁷ and its use in the treatment of acute GIB is uncertain.

Hyperbaric oxygen has emerged as a technique for difficult wound healing, and the benefits conferred by improved tissue perfusion and bacterial inhibition could be useful for the treatment of proctitis. However, several studies have shown little effect in the setting of radiation proctitis.

Endoscopic Interventions

Bipolar and heater probe electrocoagulation makes use of the standard delivery of electrocautery to a bleeding source, with its inherent advantages and disadvantages. This widely used technique makes it readily available, and the directed application of cautery causes only localized tissue damage. However,

this means tissue charring and decreased effectiveness of coagulation that may lead to the need for repeat treatments. This form of therapy is limited to localized areas of involvement, as it is not practical to treat long segments of bleeding.

Neodymium/yttrium aluminum garnet argon (Nd:YAG) lasers coagulate deep vessels. Significant improvement in clinical symptoms and decrease in transfusion requirements were seen in a small group of patients.¹⁸ Rectal necrosis, fibrosis, and stricturing can complicate its use. The cost and sophistication of the equipment for this approach prevents it from being widely available, however.

Argon plasma coagulation (APC) of thermal coagulation has largely replaced laser therapy, and has the advantage of creating a shallower and more uniform area of coagulation of superficial mucosal vessels, to minimize the risks of complications associated with laser use. Major complications of rectal necrosis, perforation, and stenosis are rare. It has been shown to be effective in controlling bleeding symptoms in hemorrhagic proctitis with 80 to 90% success. It is, however, less reliable in the treatment of severe bleeding,¹⁹ due in part to the shallow penetration of the coagulating beam. A small study has shown that its use is equivalent to bipolar electrocoagulation, with a slightly improved minor and hemorrhagic complication rate, making it a reliable option for bleeding uncontrolled by other techniques.

Radiofrequency ablation (RFA) has emerged as a safe and effective way of achieving hemostasis with little risk of complications. A catheter delivers uniform thermal energy to a broad surface area, with the ability to control the depth, intensity, and duration of the energy supplied. In this way, RFA also targets the superficial mucosa but can reach broader and multiple areas of concern simultaneously. Treated areas have been shown to re-epithelialize, avoiding the mucosal damages and long-term changes that lead to strictures with other modalities.²⁰

Operative Strategies

Intractable symptoms related to proctitis may require surgical intervention, which will be necessary only in a minority of patients. For the bleeding patient, proctectomy may be required to remove the bleeding source. However, the decision to proceed with resection should be a measured one, as anastomotic and perineal wound complications can be prohibitively high when dealing with the irradiated pelvis. This option should be preserved as much as possible for cases of intractable bleeding, in which the only way of obtaining source control is to remove the offending segment.

Fecal diversion is an effective option for addressing symptoms of chronic pain, fistulous drainage, and tenesmus but is not as efficacious in addressing bleeding. There has been some evidence that diversion can improve refractory bleeding symptoms,²¹ so in those patients at prohibitively higher risk of more extensive surgical intervention, diversion may be worth pursuing as an initial operation.

Flap reconstruction may be a surgical option for rectovaginal or rectourethral fistulae to alleviate symptoms of drainage and chronic infection. However, given the irradiated tissue and

the resultant poor wound healing, the success rates of reconstructive attempts are low. In addition, reconstruction is not a viable option for bleeding.

Rectal Varices

A common complication of portal hypertension and rectal varices are dilated submucosal veins that facilitate flow between the iliac system (via the middle and inferior rectal veins) and the portal system (via the superior rectal veins). Its prevalence is as high as 56% in patients with cirrhosis, with clinically significant bleeding rate ranging from 0.5 to 5%.²² Despite the low rate of bleeding complications, uncontrolled bleeding from this site can have life-threatening consequences. It is a priority to properly establish the diagnosis of rectal varices, particularly as they relate to hemorrhoids. The presence of hemorrhoids is an independent phenomenon, and the diagnosis of one does not rule out the presence of the other.

In considering rectal varices as a diagnosis, it is important to obtain historical information about the duration of portal hypertension and any prior procedures to treat esophageal varices. It makes conceptual sense that in patients who have portal hypertension severe enough to lead to esophageal varices, rectal varices is also more likely. And after the successful treatment of esophageal varices, the high portal pressures must then be channeled away towards other ectopic variceal structures. This concept was bolstered by a large Japanese population study which confirmed that 95% of patients with rectal varices had a history of esophageal varices and the vast majority of them had undergone previous oblitative procedures for their esophageal varices.²³

Management of rectal varices falls into medical management, endoscopic interventions, radiologic interventions, and surgical interventions.

Medical Management

Medical management of bleeding rectal varices should focus on resuscitative efforts and correction of anemia and coagulopathies. Prophylactic antibiotics have shown to improve survival. The use of vasopressin and octreotide has been shown to be effective in esophageal variceal bleeding, and their use may be expanded to the setting of rectal variceal bleeding, although this has not been studied.

Endoscopic Interventions

Once a patient is medically stabilized, definitive therapies are most often endoscopically delivered. Injection sclerotherapy involves endoscopic injection of a sclerosant, such as ethanolamine oleate, preferably under fluoroscopic guidance, so as to avoid injecting into the systemic circulation. A major complication infrequently encountered with this approach is a pulmonary embolus, and can be avoided by the slow injection of the sclerosant under reliable visualization using imaging.

In the same vein, injection of cyanoacrylate glue to effect vascular obstruction and eventual extrusion has been attempted in the treatment of rectal varices, modeled after successful use in the treatment of gastric varices. Injection first of coils to act as a scaffold helps to keep the glue localized to the varix and prevent disastrous consequences in the event of systemic embolization.²⁴ For both sclerotherapy and glue injection, adjunctive use of endoscopic ultrasound (EUS) and the color Doppler is highly encouraged to localize injection only into the submucosal varices.

Band ligation is a well-studied procedure in the treatment of esophageal varices, and less so in gastric varices. Its use in bleeding rectal varices has shown promise, although recurrence rates are high. In a head to head trial, comparing sclerotherapy with banding, sclerotherapy appeared to be superior in preventing recurrent bleeding and in its side effect profile.²⁵

Radiologic Interventions

Transjugular intrahepatic portosystemic shunt (TIPS) can serve to acutely decompress portal pressures and adequately control hemorrhage during an acute bleeding episode from rectal varices. In the largest series of patients undergoing TIPS for acute bleeding, effective hemorrhage control was obtained using TIPS only in the majority of patients, without concomitant embolization.²⁶ Rebleeding rates are acceptable and are often due to shunt dysfunction, amenable, and responsive to shunt revision. TIPS is effective as both a bridge to transplantation and/or as a standalone therapy in nonoperative patients; hence, the actively bleeding patient with known portal hypertension as the source of ectopic variceal bleeding merits an urgent evaluation by an interventional radiologist.

Angiographic embolization can be performed alone or in conjunction with TIPS or other endoscopic procedures. Embolization to occlude the feeding vein to the varix may be performed with coils, ethanol, thrombin, collagen matrices, or autologous blood. Used alone, embolization has a high rebleeding rate,²⁷ so its use as an adjunct to one of the aforementioned endoscopic interventions or TIPS should be strongly considered.

Surgical Management

When endoscopic interventions fail, an operative approach may be necessary. Patients presenting with bleeding rectal varices are generally poor operative candidates due to their underlying liver failure, and carry a high baseline mortality rate. So while surgical decompression of the portal system does confer advantages in obtaining upstream control of hemorrhage, the incumbent risk to the patients are prohibitive. Local control of bleeding is often transient if successful, as the underlying portal hypertension is not addressed.

Suture ligation may be attempted but complete and adequate control of the submucosal venous network is often impossible. The apex of the bleeding tissue is ligated and the distal mucosa is oversewn. The ligated tissue is not excised but

it will slough after 7 to 10 days. Excision must be avoided as this can lead to uncontrolled, life-threatening hemorrhage. An alternative approach using a circumferential stapling device has been described in case reports and a small case series that demonstrated the safety and efficacy of obtaining hemostasis.²⁸ All patients underwent the procedure under emergent circumstances for the goal of obtaining hemorrhage control, using a technique that mirrors that of stapled hemorrhoidectomy. Familiarity with the circular stapling device and expertise in colorectal surgery are prerequisites.

Conclusion

As the U.S. population ages, chronic conditions and their treatments that carry higher risk of LGIB have become more prevalent. While many pharmacologic and interventional, particularly nonsurgical, advancements are emerging to address the challenges unique to the various special circumstances of LGIB, the importance of thorough history taking and understanding of the individual patient characteristics cannot be overemphasized in order to optimize patient care in increasingly complex situations.

Conflict of Interest

None declared.

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