

Clinical Characteristics and Causes among Patients with Lower Gastrointestinal Tract Bleeding

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ABSTRACT

Lower gastrointestinal bleeding (LGIB) is a common acute medical and surgical emergency. The incidence of LGIB is about 20-30 cases per 100,000 individuals. The clinical manifestations of LGIB differ and depend on the rate, quantity and location of bleeding. Diverticular bleeding remains the most frequent cause of bleeding followed by internal hemorrhoids, ischemic colitis and post-polypectomy bleeding. Diagnosis of the bleeding cause is sometimes difficult especially in the small bowel and non-surgical hemostasis is not always enough. The majority of LGIBs resolves spontaneously with conservative medical treatment and thus a lesion may remain undiagnosed until bleeding recurrence or other symptomatology in the future. Given that there is no specific treatment, the management of LGIB is still based on intravascular volume resuscitation, hemodynamic stabilization and close monitoring of the patients. Therefore, this study aimed to review a variety of clinical disorders and causes among patients with Lower Gastrointestinal Tract Bleeding.

Keywords: Gastrointestinal Tract Bleeding ; Diverticular disease; hemorrhoids

INTRODUCTION

Lower gastrointestinal bleeding (LGIB), defined as bleeding from the gastrointestinal tract distal to the ligament of Treitz is a relatively common emergency in acute state (1). LGIB can be further classified depending on the location of bleeding as small or large bowel (2). LGIB is a common acute medical and surgical emergency. Acute colonic bleeding occurring from the colon, rectum, or anus, and presenting as either hematochezia (bright red blood, clots or burgundy stools) or melena (3). The National Confidential Enquiry in Patient Outcomes and Death (NCEPOD) published a report on severe gastrointestinal bleeds that found that only 44% of patients received good care. This is at least partly the result of the lack of high-quality research into the management of patients that present with LGIB, a problem that this thesis aims to address (4).

Incidence of lower gastrointestinal bleeding:

Although LGIB is common condition, there are limited studies documenting its incidence. Annual incidence of LGIB hospitalization of approximately 36/100,000 population, about half of that for upper GI bleeding. The rate of hospitalization is even higher in the elderly (5). The incidence of LGIB is probably rising due to the aging of the population, the higher rates of comorbidities and the increasing use of aspirin, anticoagulants and non-steroidal anti-inflammatory drugs (NSAIDs)(6,7).

A group in Iceland calculated an incidence of 87/100,000 for LGIB by multiplying the number of endoscopy referrals for the investigation of rectal bleeding or melaena. This method also comprised a small number of patients and may be inaccurate as endoscopy is not the sole mode of investigation of LGIB. Historical studies estimate the incidence of LGIB as 20.5/100,000 but again this is based on data from a single center (3). A population-based study compared patients discharged with hospital codes consistent with GI complications. It was found that in comparison to the upper GI tract, where the incidence of complications fell from 87/100,000 to 47/100,000 over ten years, the incidence of complications in the lower GI tract increased from 20/100,000 to 33/100,000 (9).

Causes of lower gastrointestinal bleeding:

Colonic diverticulosis continues to be the most common cause of LGIB, accounting for about 30 % of lower GI bleeding cases requiring hospitalization. Internal hemorrhoids are the second-most common cause. Ischemic colitis and post-polypectomy bleeding are increasing in frequency, likely due to an increase in medical comorbidities and anti-platelet/anticoagulant use(10). Diverticular bleeding reported in several previous studies in the population group suffering from LGIB(3,5,11). Their co-existence with other causes of bleeding can make the differential diagnosis of LGIB challenging(7). The most common cause of small bowel GI bleed is a vascular lesion. 30-40% of bleeding in the small bowel is caused by abnormal vessels in the small bowel, with angiodysplasia being the most common. The vascular lesions may also be induced by NSAIDs. The other causes include tumors, aorto-enteric fistula, medications, small intestine ulcers, and non-specific enteritis (12).

LGIB can be categorized further into massive, moderate, and occult bleeding. Massive bleeding usually occurs in patients older than 65 years with multiple medical problems, and this bleeding presents as hematochezia or bright red blood per rectum.

The patient is usually hemodynamically unstable with a systolic blood pressure (SBP) equal to or less than 90 mmHg, heart rate (HR) less than or equal 100/min, and low urine output. Lab work reveals a hemoglobin equal to or less than 6 g/dl. Massive lower GI bleeds are mostly due to diverticulosis and angiodysplasias. The mortality rate may be as high as 21% (13). Moderate bleeding can occur at any age and presents as hematochezia or melena. The patient is usually hemodynamically stable. Many disease processes should be considered on the differential list including neoplastic disease, inflammatory, infectious, benign anorectal, and congenital (14). Occult lower GI bleeds can present in patients at any age. Lab work reveals patients with microcytic hypochromic anemia due to chronic blood loss. The differential diagnosis of these patients should include inflammatory, neoplastic and congenital (13).

i. Diverticular disease:

Diverticular disease is one of the most common conditions. The pathogenesis of diverticular disease is thought to be multifactorial and include both environmental and genetic factors in addition to the historically accepted etiology of dietary fiber deficiency. Symptomatic uncomplicated diverticular disease (SUDD) is a type of chronic diverticulosis that is perhaps akin to irritable bowel syndrome (15). It was the 16th common cause of death among gastrointestinal diseases with crude death rate of 0.9 per 100,000. Moreover, diverticulosis is increased in prevalence with increasing age (16).

A major predisposing factor for the formation of colonic diverticula is abnormal colonic motility (e.g., intestinal spasms or dyskinesia), resulting in exaggerated segmental muscle contractions, elevated intraluminal pressures, and separation of the colonic lumen into chambers. The increased incidence of diverticula in the sigmoid colon is explained by Laplace's law, such that pressure is proportional to wall tension and inversely proportional to bowel radius. As the sigmoid colon is the colon segment with the smallest diameter, it is also the segment with the highest intraluminal pressures (17).

Diverticulitis is the most common complication of diverticulosis, which occurs in about 10% to 25% of patients. The pathophysiology of diverticulitis is the obstruction of the diverticulum sac by irritation of the mucosa causes low-grade inflammation, congestion and further obstruction. Diverticulitis may be classified as uncomplicated and complicated. Complicated diverticulitis is characterized by the formation of abscesses, fistulas, obstruction and/or perforation (18). Segmental colitis associated with diverticulosis (SCAD) is now recognized as a distinct entity. It is

characterized by nonspecific segmental inflammation in the sigmoid colon surrounded by multiple diverticula. It does not necessarily involve the diverticular orifice (19).

Most bleeding associated with diverticulosis is self-limiting and not require intervention. In some cases, however, endoscopic, radiologic, or surgical intervention may be required to stop persistent bleeding, or mechanical devices. If a source cannot be determined in the case of recurrent bleeding, surgery may be considered to remove portions of the affected intestine. Similarly, in the case of a giant diverticulum, with an increased risk of infection and rupture, surgery is more likely to be considered (20).

ii.Hemorrhoidal Disease:

Hemorrhoidal disease is defined as a symptomatic enlargement and distal displacement of normal anal cushions (21). There is no data on the actual incidence of hemorrhoidal disease, and various studies report prevalence between 4% and 40% (22). This disease usually affects patients aging 45–65 years, but nowadays, younger patients are diagnosed with hemorrhoids. The common risk factors are considered to be pregnancy, constipation/chronic diarrhea, internal rectal prolapse, aging, obesity, depressive mood, prolonged straining, low fiber diet, spicy food, alcohol intake, chronic cough, strenuous exercises, weight lifting, long-standing, family history, etc. (23). Clinical symptoms vary from bleeding to ulceration and mucosal atrophy, and the manifestations depend on the grade of the hemorrhoidal disease (22).

The internal hemorrhoids arise from internal hemorrhoidal plexus when three soft hemorrhoids enlarge and transform into anal nodules. In turn, the external hemorrhoids arise from external plexus. A contradicting theory describes the external hemorrhoids as extensions of internal ones. However, the definition of the disease has certain incompleteness, since anal cushions are named” hemorrhoids” when they bleed and/or prolapse (24).

Surgical treatment is indicated for grade III and IV internal hemorrhoids and thrombosed external hemorrhoids with persistent symptoms. A perfect operation for hemorrhoids must have minimal post-operative pain and complications, with no risk of recurrence development (25). It has been shown that age itself is not correlated with postoperative complications (26). Treatment of hemorrhoids with the use of a stapling device. In stapled haemorrhoidopexy, the loose connective tissue of mucosa and the enlarged hemorrhoidal nodules above the dentate line contributing to the prolapse are excised. According to the technique, a special circular device a stapler excises a ring of redundant mucosa and fixes the hemorrhoidal plexus 4 cm above the dentate line without any sphincter tissue (27).

iii. Infectious Colitis:

Infectious colitis is diagnosed when diffuse inflammation of the colonic mucosa is caused by an inflammatory pathogen, which can be invasive or noninvasive. Diagnosis of colitis should be sought in acutely ill patients with diarrhea, especially if passing grossly bloody stools (dysentery), with tenesmus, fecal urgency or with laboratory evidence of fecal inflammatory markers (28). Most causes of acute colitis have an infectious etiology. When an immunosuppressed patient presents with diarrhea, fever and neutropenia typically within two to three weeks of receiving chemotherapy, neutropenic colitis known as typhlitis should be suspected. If a patient has recently received microbiome-depleting antibiotics or stayed in a hospital or nursing home for at least 3 days, colitis due to *C. difficile* infection (CDI) should be considered (29). Dysenteric colitis caused by invasive enteropathogens is often accompanied with fever at least 102°F and abdominal cramps or pain. *Yersinia* and *Campylobacter* colitides and typhlitis may be localized to ileocecal region presenting as acute right lower quadrant abdominal pain mimicking appendicitis (30).

Therapeutic recommendations by dysentery etiology are provided. Rehydration with fluid and electrolytes is recommended for all forms of diarrhea and to treat mild forms of dehydration. Encouraging patients to drink soups and broths and other nonalcoholic or caffeinated beverages is usually sufficient. Special care should be provided to elderly as they can more quickly develop dehydration and suffer complications when compared to younger patients. Anti-motility agents (e.g. Loperamide) should not be given routinely for patients with infectious colitis (31).

iv. Ulcerative Colitis

Ulcerative colitis (UC) is a chronic disease affecting the large intestine, with an increasing incidence worldwide. Nearly 1 million individuals each in the United States and Europe are affected by this condition and many more globally (32). Therapeutic management in UC should be guided by the specific diagnosis (i.e., Montreal classification), an assessment of disease activity (i.e., mild, moderate, or severe), and disease prognosis. A distinction made in this updated guideline is that treatment selection should be based not only on inflammatory activity but also on disease prognosis (33).

The precise etiology of UC is not known and 'cure' of this disease is not yet possible. Therefore, the primary therapeutic goal is to induce and maintain remission of the inflammation. The main treatment goals for UC include clinical remission, i.e. relief of symptoms, in combination with mucosal healing. According to the classic

step-up approach, treatment of UC starts with administration of 5-aminosalicylic acid (5-ASA) compounds, followed by short-term use of corticosteroids, immunomodulators and biologicals(34).

v. Colorectal Cancer

Despite increased screening efforts, up to 33% of patients with colorectal cancer present with symptoms requiring acute or emergent surgical intervention. Common emergency presentations include large bowel obstruction, perforation, and hemorrhage (35). GI bleeding is reported in up to 50% of patients with colorectal cancer. Bleeding is often an early symptom of a colorectal cancer associated with lower risk of advanced stage at diagnosis, and a shorter delay in presentation. Unlike the insidious onset of an obstructing cancer, patients often remember to the day when bleeding began (36).

Bleeding is complicated by the fact that most acute tumor bleeding is likely in the setting of chronic anemia of cancer and blood loss from the tumor. Acute massive gastrointestinal bleeding from a colorectal carcinoma is rare. The initial management is aimed at resuscitation, establishing large-bore IV access, and stabilization with crystalloid and correction of underlying coagulopathy or other metabolic abnormalities (35).

Surgery is the most effective and definitive approach for a hemorrhaging colorectal cancer. Some general indications for surgical intervention include hemodynamic instability despite transfusion of more than 6 units of blood products, slow bleeding requiring more than 3 units of blood products per day, inability to stop hemorrhage with endoscopic or endovascular techniques, or recurrent episodes of hemorrhagic shock (37).

CONCLUSION

Identification of a LGIB source is an important role that leading to increase the success of treatment intervention.

No Conflict of interest.

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