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Lower Gastrointestinal Bleeding: An Etiological Study

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Abstract

Background Lower gastrointestinal bleeding (LGIB) is defined as bleeding from a source below the ligament of Treitz. Most series which studied the etiology of the acute LGIB showed that Colonic diverticulae and angiodysplasia were the commonest etiology identified, ranging from (17- 40%, 2-30% respectively). Colitis including the inflammatory bowel disease, account for 6-30% of cases. Uncommon causes of LGIB include colonic neoplasia in 11-14% and anorectal lesions in 4-10% (mostly due to hemorrhoids,). Other less common cause is small bowel source in 2-9%; rare causes include Dieulafoy lesion and colonic ulcerations.

Objective To verify the etiology of LGIB in Iraqi patients.

Methods The study group included 100 patients who were suffering from acute lower gastrointestinal bleeding and referred to the endoscopy unit in the Gastroenterology and Hepatology Teaching Hospital. After the initial history, physical examination and laboratory studies were obtained; all patients with hemodynamic instability were resuscitated with intravenous fluid and blood transfusion. Colonoscopy performed within 12 hours of hospitalization (with or without an upper endoscopy), with the aim of reaching up to the cecum in all cases.

Results Of 100 patients (55 female, 45 male) 78% presented with haematochesia, 11% with red maroon stool with malaena, and 11% with malaena only. Bleeding due to colitis was the most frequent diagnosis, which is reported in 38 patients. The diagnosis of anorectal lesions and colonic neoplasia were the second and third most common diagnosis (21%, 12% respectively). Colonic diverticulae in 12 patients, colonic angiodysplasia in 11 patients and small intestinal source is 5 patients and 1 patient with colonic Dieulafoy lesion.

Conclusions This study showed that inflammatory bowel disease; colorectal polyps including post polypectomy bleeding, diverticulosis, angiodysplasia, and hemorrhoids were the most common causes of lower gastrointestinal bleeding.

Key words Lower gastrointestinal bleeding

Introduction

Lower gastrointestinal bleeding (LGIB) is defined as bleeding from a source below the ligament of Treitz, it occurs at a rate of 20 per 100,000 population, which is about one fifth as frequently as upper gastrointestinal bleeding [(1)]. The incidence of LGIB is higher in men and increases with age, presumably because of the high frequency of diverticulosis and vascular disease in older men [(2)]. Hematochezia, which is the most common presenting symptom for lower gastrointestinal hemorrhage, can be variously

described as bloody diarrhea, blood and clots per rectum, maroon-colored stool, or blood mixed with the stool, hematochezia can occur from bleeding anywhere in the gastrointestinal tract, and about 10% of patients who present with hematochezia have an upper gastrointestinal source of bleeding [3, 4].

Few data on prognostic variables for lower gastrointestinal bleeding exist; the following clinical data were associated with severe bleeding: heart rate ≥100/minute; systolic blood pressure ≤115 mm Hg; syncope; a non-tender abdominal examination; bleeding per rectum during the first 4 hours of evaluation; aspirin use; and more than two active comorbid conditions [5-7].

It is important to determine the color of the stool. Such information appears to be the most informative [8]. Bright red blood commonly indicates a distal colonic source or a rapidly bleeding proximal (small intestinal or gastric) source, whereas black stool indicates a slowly bleeding right colonic or more proximal source. Accordingly, in patients with apparent massive LGIB, it is important to exclude upper gastrointestinal hemorrhage by examining an aspirate from a nasogastric tube.

A thorough history and physical examination often point to the correct diagnosis. For example, LGIB in elderly patients is commonly caused by colonic diverticula or vascular ectasias, whereas in young patients, infectious or inflammatory conditions are more likely [9, 10]. A prior diagnosis of hemorrhoids or inflammatory bowel disease is important.

A history of NSAIDs intake, radiation therapy, previous surgery (particularly vascular surgery), and constipation, a change in bowel habits, and anorectal disease or trauma are important to consider in making a correct diagnosis [11]. Symptoms that are associated with bleeding, such as abdominal pain or diarrhea, suggest specific diagnoses. A recent history of anorexia or weight loss or an abdominal mass found during physical examination may indicate an underlying malignancy.

Historically, the two major causes of acute LGIB were thought to be diverticulosis and angiodysplasia [12-14].

Most series which studied the etiology of the acute LGIB showed that diverticular bleeding was the commenst etiology identified ranging from 17 to 40% of LGIB episodes [15,16]. Despite the fact that most diverticulae are located in the left colon in western individual , a number of series suggest that bleeding diverticulae occur more often in the proximal colon [17-20].

Angiodysplasia are ectatic blood vessels seen in the mucosa and the submucosa of the GI tract, with 1 to 2% incidence in the colon at autopsy and colonoscopy [21,22]. Angiodyslpasia are identified most commonly in elderly patients; more than two thirds of affected patients are older than 70 years of age [23-25]. Approximately three quarters of bleeding angioectasias are identified in the right colon [26-28].

Colonic neoplasia (including postpolypectomy bleeding) is uncommon cause of LGIB, if not rare, bleeding can be the presenting symptom of colonic neoplasia and the cause of LGIB in 11 to 14 % and are thought to bleed mainly from erosions on the luminal surface [29].

Postpolypectomy bleeding occur in 2-3% of patient undergoing polypectomy and is thought to occur from separation of the polyp before there is adequate coagulation of the blood vessel in the stalk, or from sloughing of the coagulum in case of delayed bleeding [30].

Colitis, including the inflammatory bowel disease, infectious colitis, radiation colitis and ischemic injury, can account for 6% to 30% of acute lower intestinal bleeding [31].

Severe GI bleeding is said to account for 6% to 10% of emergency surgical resections for ulcerative colitis. Ulcerative colitis and crohns' disease is responsible for 2% - 8% and 1% of cases of lower GI bleeding respectively [32,33].

Radiation therapy induces inflammatory changes in the bowel wall and can result in mucosal telangictases that bleed [34].

Colonic ischemia can also present with acute lower GI bleeding. Few series reported it in 3% of cases [35]. Reports of vasculitis as a cause of lower GI bleeding incude polyarteritis nodosa, wegeners granulomatosis, and rheumatoid vasculitis, this is caused by ulcerating necrotizing process resulting in hemorrhage [31].

Less common causes of acute LGIB include anorectal lesions in 4-10% (including hemorrhoids, solitary rectal ulcer syndrome, and rectal varices). Hemorrhoids prevalence rate of 76% have been reported in patients with acute LGIB, but an etiological relationship is established only in 2-9% of cases [36].

Other less common cause are small bowel source in 2-9%, rare causes include Dieulafoy lesion and colonic ulcerations [37]. Cases of aortocolonic fistula formation with bleeding have rarely been reported months or years after aortic graft surgery [38].

The source of bleeding cannot be identified definitively in a substantial number of patients [2, 39]. The diagnosis should be sought in all patients with lower GI bleeding unless their overall prognosis is too poor to warrant further tests.

Urgent colonoscopy has been defined variably as one performed within 12-48 hr of admission. Urgent colonoscopy enables a final diagnosis of colonic lesions in 74–90% of patients, this diagnostic accuracy is better than that of angiography [40,41].

The majority of episodes of acute LGIB cease spontaneously, regardless of source, but patients with continuing or recurrent bleeding require intervention to stop the bleeding [42].

Methods

The study group included 100 patients who were suffering from acute lower gastrointestinal bleeding and referred to the endoscopy unit in the Gastroenterology and Hepatology Teaching Hospital between February 2009 and October 2010. After the initial history and physical examination were performed, laboratory studies were obtained, including hematocrit, platelet count, prothrombin time, activated partial thromboplastin time and type, and cross match.

All patients with hemodynamic unstability (defined as either a resting heart rate over 100 beats per minute; resting systolic blood pressure below 90 mm Hg; or evidence of end-organ compromise, specifically lightheadedness, syncope, chest pain, or dyspnea at the time of hospitalization) were resuscitated with intravenous fluid and blood transfusion.

All patients were prepared with a polyethelyne-glycol-based purgative administered either orally (1 cup every 15 min) or by nasogastric tube (250 mL every 15 min) for patients unable to comply with oral intake. The goal was 4 to 6 L of purge requiring 3-4 hr. to clean the colon.

Patients then received conscious sedation with IV pethedine 50mg and diazepam 10mg while monitoring heart rate, blood pressure, and oxygen saturation.

Colonoscopy performed within 12 hr of hospitalization with or without an upper endoscopy. A standard video colonoscope (Olympus, Tokyo, Japan) was used for all patients with the aim of reaching up to the cecum in all cases.

When evidence of more than 1 potential site of bleeding was identified, the probable cause of bleeding was judged by the presence of ongoing hemorrhage or adjacent evidence of recent bleeding.

Push enteroscopy was performed if the source of bleeding remained undiagnosed after both colonoscopy and upper endoscopy. Other investigations were done in some patients in whom the source of the bleeding was not identified on initial endoscopic screen, these tests were included small intestinal barium study, mesenteric angiography (done by seldinger technique using JR catheter and iohexol contrast media) and intraoperative enteroscopy; this is judged according to the clinical situation of the patients and the haemodynamic state.

Statistical analysis was performed using the SPSS software package, Version 7.5. The chi square test was used to compare categorical data.

Results

Seventy eight patients (78%) were presented with haematochesia, eleven patients (11%) with red maroon stool with malaena and eleven patients (11%) with malaena only. Twelve patients (12%) had haeomodynamic instability at the time of the presentation. Multiple comorbidities were reported in 27% of the patients, which include (DM, cardiovascular disease, cerebrovascular disease and renal failure). Fifteen patients only had history of aspirin and NSAIDs use before the development of the bleeding.

The mean hemoglobin level of our patients at the time of presentation was 9.7±1.2 g/dl and the mean blood transfusion units received was 2.6±1.2. The demographic characteristics of the study group are shown in table 1.

Table 1. The demographic characteristics of the study group.

Feature		Number, %, Mean±SD
Total No. of patients		100
Age (Years)		36.1±20.1
Gender	Males	45
	Females	55
Aspirin & NSAID Use (%)		15%
Hemodynamic Instability (%)		12%
Comorbidities (%)		27%
Units of blood Transfused		2.6±1.2
Hb level at presentation		9.7±1.2
Length of the hospital stay(days)		6.7±1.3

All patients underwent total colonoscopy other diagnostic studies which had been done include OGD (esophagogastroscpy) in 22 patients, barium follow through in 10 patients, enteroscopy for 12 patients, angiography for 2 patients and 1 patient underwent lapratomy with intraoperative enteroscopy.

Bleeding due to colitis was the most frequent diagnosis, which is reported in 38 patients; ulcerative colitis in 28 patients, crohns colitis in 4 patients (assuming that there are no other causes of the bleeding), infective colitis in 4 patients, drug-induced colitis in 1 patient and ischemic colitis in another 1 patient).

The diagnosis of anorectal lesions and colonic neoplasia as the cause of the bleeding were the second and third most common diagnosis (21%, 12% respectively).

Other etiology included, in the order of frequency, colonic diverticulae in 12 patients, colonic angiodysplasia in 11 patients and small intestinal source is 5 patients and 1 patient with colonic Dieulafoy lesion. Table 2 showed the etiology of the bleeding.

Table 2. The Etiology of Bleeding

Cause		Frequency	Percent
Diverticulae		12	12%
Colonic Angiodysplasia		11	11%
Colitis (total = 38)	ulcerative Colitis	28	38%
	Crohn's Colitis	4	
	Infectious	4	
	Ischemic	1	
	Drug induced	1	
Colonic neoplasia (total = 12)	Colonic polyp	6	12%
	Colonic carcinoma	4	
	Post-Polypectomy bleeding	2	
Anorectal Lesions (total = 21)	Piles	8	21%
	Solitary Rectal Ulcer	4	
	Rectal polyp(non-neoplastic)	9	
Small Intestinal Source (total = 5)	Typhoid ulcer	2	5%
	T.B. ilietis	1	
	Intestinal Telengectasia	1	
	Meckls diverticulum	1	
Colonic Dieulafoy lesion		1	1%

Statistical analysis of the etiological data in different age group showed that the diverticular bleeding is the most frequent cause in patient's age more than 50 years (33.3%) followed by angiodysplasia (27.7%). Colitis was the most common cause of bleeding in age group below 50 years old (55.3%).

The children (age < 12 years), non- neoplastic rectal polyp is the most frequent cause of lower G.I.T bleeding (52.9%). The etiology in different age groups is shown in table 3.

During the hospitalization of the patients, spontaneous cessation of the bleeding occurred in 79 patients. Endoscopic control of the bleeding was attempted in the remaining 21 patients, and it was successful in 17 patients (6 patients with divrticular bleeding; 8 patients with angiosdysplasia; 2 with postpolypectomy bleeding and one patient with colonic dieulafoy lesion).

The remaining 4 patients in whom endoscopic therapy failed or cannot be done were referred for emergency surgery (1 with Meckles disease, 1 with intestinal telengectasia and 2 patients with diverticular bleeding).

The mortality was reported in 3 patients (1 with bleeding dieulafoy lesion, 1 patient with ischemic colitis and 1 patient with diverticular bleeding).

Discussion

Lower GI bleeding encompasses a wide clinical spectrum ranging from trivial bleeding to massive hemorrhage with shock. Lower GI bleeding is more common in men than in women, which is different from our study group which showed slight female predominance (55 versus 45 patients), and the incidence rate increases with age, with a greater than 200-fold increase from the 3rd to the 9th decades of life [(2)].

Table 3. The etiology in different age groups

Etiology	Age <12 (17 patients)	Age <50 (47 patients)	Age >50 (36 patients)	Total (100)
Diverticular disease	-	-	12(33.3%)	12
Colonic angiodysplasia	-	1(2.1%)	10(27.7%)	11
Colitis	7 (41.1%)	26 (55.3%)	5 (13.8 %)	
IBD	2	26	3	
Drug induced	1	-	-	38
Infective	4	-	1	
Ischemic	-	-	1	
Colonic Neoplasia	-	3 (6.3%)	9 (25%)	
Colonic cancer	-	1	3	12
Colonic polyp	-	1	5	
Post polypectomy	-	1	1	
Anorectal lesion	9(52.9%)	12 (25.5%)	-	
Solitary rectal ulcer	-	4	-	21
Pile	-	8	-	
Rectal polyp	9	-	-	
Small intestinal source	1(5.8%)	4(8.5%)	-	
Typhoid ulcer	-	2	-	
T.B ileitis	-	1	-	5
Telengectasia	-	1	-	
Meckls diverticulum	1	-	-	
Colonic Dieulafoy lesion	-	1(2.1%)	-	1

In a study done by peura DA, etal on patients with lower gastrointestinal bleeding showed that less hemodynamic instability than those with upper gastrointestinal bleeding and show less frequent orthostasis (19% versus 35%), need less frequent blood transfusions (36% versus 64%), and present with higher hemoglobin levels [6,7]. This is also seen in our patients were only 12% of them showed hemodynamic instability and 38% had received blood.

Another study done by Strate et al on 252 patients with acute lower gastrointestinal bleeding found predictive factors which increase the likelihood of a severe course or recurrence of bleeding: heart rate >100/min; systolic blood pressure <115 mmHg; history of acetylsalicylic acid use and more than two active comorbid conditions [5]. This is also seen in some of our patients, 3 of them died because of aspirin use and associated comorbidities.

The frequency of the source of colonic bleeding reported varies from one publication to the next. Epidemiologic and historical features should be considered. For example, in patients with lower GI bleeding over the age of 65, colonic angiodyslpasia, diverticular hemorrhage, or ischemic colitis, are most common, while in young patients, infectious or inflammatory conditions are more likely [9, 10]. This is comparable to our findings which showed that diverticular and angiodysplasia bleeding was more common in patients over the age of 50 year (33.3%, 27.7% respectively). While colitis was the most common cause in those below 50 years (55.3%).

We have found that the most common cause of lower GI bleeding in our patients was inflammatory bowel disease (32%), this is higher than what reported in the literature (16%), which can be attributed to young age of most of our patients [(9)]. However, the frequency of the colonic diverticulae and angiodysplasia as a cause of lower GI bleeding, in our study, was (12%) and (11%) respectively, this finding is less than those reported in the literature (17-40%) for diverticular bleeding and up to (30%) for angiodysplasia bleeding [(42)], which can be attributed to relatively small number of our patients were above the age of 50 years.

A striking finding in our study was that anorectal lesions were responsible for (21%) of causes of lower GI bleeding which is much more than reported in other series (4-11%), this can be explained by that our hospital is a tertiary referral center which receive large number of pediatric patients in whom rectal polyps are the most common cause of lower GI bleeding.

Other less frequent causes of lower GI bleeding were reported with lower frequencies in our study for example colonic neoplasia (including postpolpectomy bleeding) account for 12% of cases of haematochezia, hemorrhoids are reported to account for 8% of acute lower GI bleeding episodes, small intestinal source of bleeding is encountered in 5% of cases. These findings are comparable to those found in other series which reported incidence of 11-14%, 4-10% and 2-9% for bleeding from colon neoplasia, hemorrhoids and small intestinal source of bleeding respectively [(9,25,29,36,37)].

Prognosis in lower GI bleeding varies; however, since most acute lower GI bleeding is self-limited, outcomes are typically favorable. Spontaneous cessation of acute lower gastrointestinal bleeding is seen in about 79% of our patients, and it is similar to other studies which reported spontaneous cessation rate of 80 % [(43)].

Indeed, the mortality rate associated with lower GI bleeding is generally considered to be less than 5% and when it occurs, is often a result of comorbid conditions and the need for emergency surgery is in the range of 5% [(2)]. which is also reported in our study 3% and 4% for mortality and emergency surgery respectively.

In conclusion, there are numerous lesions that may be responsible for lower GI bleeding. Our study showed that inflammatory bowel disease; colorectal polyps including post polypectomy bleeding, diverticulosis, angiodysplasia, and hemorrhoids were the most common causes of lower gastrointestinal bleeding. Colonoscopy allows for diagnosis in most patients with lower gastrointestinal bleeding. Lower intestinal bleeding generally has a less severe clinical presentation and course and typically favorable outcomes.

References

1. Talley NJ, Jones M. Self-reported rectal bleeding in a United States community: Prevalence, risk factors, and health care seeking. Am J Gastroenterol. 1998; 93: 2179-83.
2. Longstreth GF. Epidemiology and outcome of patients hospitalized with acute lower gastrointestinal hemorrhage: a population-based study. Am J Gastroenterol. 1997 Mar; 92(3): 419-24.
3. Lanas A, Sekar C, Hirschowitz BJ. Objective evidence of aspirin use in both ulcer and non-ulcer upper and lower gastrointestinal bleeding. Gastroenterology. 1992 Sep; 103(3): 862-9.
4. Wilcox CM, Alexander LN, Cotsonis GA, et al. Nonsteroidal antiinflammatory drugs are associated with both upper and lower gastrointestinal bleeding. Dig Dis Sci. 1997 May; 42(5): 990-7.
5. Strate LL, Orav EJ, Syngal S. Early predictors of severity in acute lower intestinal tract bleeding. Arch Intern Med. 2003 Apr 14; 163(7): 838-43.
6. Peura DA, Lanza FL, Gostout CJ, et al. The American College of Gastroenterology Bleeding Registry: preliminary findings. Am J Gastroenterol. 1997 Jun; 92(6): 924-8.
7. Bramley PN, Masson JW, McKnight G, et al. The role of an open-access bleeding unit in the management of colonic haemorrhage. A 2-year prospective study. Scand J Gastroenterol. 1996 Aug; 31(8): 764-9.
8. Zuckerman GR, Trellis DR, Sherman TM, et al. An objective measure of stool color for differentiating upper

from lower gastrointestinal bleeding. Dig Dis Sci. 1995 Aug; 40(8): 1614-21.

9. Schmulewitz N, Fisher DA, Rockey DC. Early colonoscopy for acute lower GI bleeding predicts shorter hospital stay: A retrospective study of experience in a single center. Gastrointest Endosc. 2003 Dec; 58(6): 841-6.
10. Elta GH. Urgent colonoscopy for acute lower-GI bleeding. Gastrointest Endosc. 2004 Mar; 59(3): 402-8.
11. Bini EJ, Weinshel EH, Falkenstein DB. Risk factors for recurrent bleeding and mortality in human immunodeficiency virus infected patients with acute lower GI hemorrhage. Gastrointest Endosc. 1999 Jun; 49(6): 748-53.
12. Reinus JF, Brandt LJ. Vascular ectasias and diverticulosis: Common causes of lower intestinal bleeding. Gastroenterol Clin N Am. 1994; 23: 1-20.
13. McGuire HH, Hayness BW. Massive hemorrhage from diverticulosis of the colon: guidelines for the therapy based on bleeding patterns observed in fifty cases. Ann Surg. 1972; 175: 847-53.
14. Meyer MA, Alonso DR, Gray GF, et al. Pathogenesis of bleeding colonic diverticulosis. Gastroenterol. 1976 Oct; 71(4): 577-83.
15. Potter GD, Sillen GH. Lower gastrointestinal bleeding. Gastroentrol Clin N Am. 1988 Jun; 17(2): 341-56.
16. Casarella WJ, Kanter IE, Seaman WB. Right sided colonic diverticula as a cause of acute rectal hemorrhage. N Eng J Med. 1972 Mar 2; 286(9): 450-3.
17. Wong SK, Ho YH, Leong APK, et al. Clinical behavior of complicated right-sided and left-sided diverticulosis. Dis Colon Rectum. 1997; 40: 344-8.
18. McGuire HH. Bleeding colonic diverticula: A reappraisal of natural history and management. Ann Surg. 1994; 220: 653-6.
19. Schmulewitz N, Fisher DA, Rockey DC. Early colonoscopy for acute lower GI bleeding predicts short hospital stay. Gastrointest Endosc. 2003 Dec; 58(6): 841-6.
20. Stollman NH, Raskin JB. ACG guidline: Diagnosis and management of diverticular disease of the colon in adults. Ad Hoc Practice Parameters Committee of the American College of Gastroenterology. Am J Gastroenterol. 1999 Nov; 94(11): 3110-21.
21. Foutch PG. Angiodysplasia of the gastrointestinal tract. Am J Gastroenterol. 1993 Jun; 88(6): 807-18.
22. Baum S, Athanasoulis CA, Waltman AC, et al. angiodysplasia of the the right colon: a cause of the gastrointestinal bleeding. AJR Am J Roentgenol. 1977; 129: 789-94.
23. Foutch PG, Rex DK, Lieberman DA: Prevalence and natural history of colonic angiodysplasia among healthy asymptomatic people. Am J Gastroenterol. 1995 Apr; 90(4): 564-7.
24. Foutch PG. Angiodysplasia of the gastrointestinal tract. Am J Gastroenterol. 1993; 88: 807-18.
25. Zuccaro G. Epidemiology of the lower GI bleeding. Res Clin Gastroenterol. 2008; 22(2): 225-32.
26. Gupta N, Longo WE, Vernava AM. Angiodysplasia of the lower gastrointestinal tract: An entity readily diagnosed by colonoscopy and primarily managed nonoperatively. Dis Colon Rectum. 1995 Sep; 38(9): 979-82.
27. Zuckerman GR, Prakash C. Acute lower intestinal bleeding. Part II: Etiology, therapy, and outcomes. Gastrointest Endosc. 1999 Feb; 49(2): 228-38.
28. Bemvenuti GA, Jülich MM. Ethanolamine injection for sclerotherapy of angiodysplasia of the colon. Endoscopy. 1998 Aug; 30(6): 564-9.
29. Peura DA, Lanza FL, Gostout CJ. The American gastroenterology bleeding registry: preliminary findings. Am J Gastroenterol. 1997; 92: 924-8.
30. Rosen L, Bub DS, Reed JF. Hemorrhage following colonoscopic polypectomy. Dis Colon Rectum. 1993 Dec; 36(12): 1126-31.
31. Zuckerman GR, Prakash C. Acute lower intestinal bleeding: part II: clinical presentation and diagnosis. Gastrointest Endosc. 1999 Feb; 49(2): 228-38.
32. Robert JH, Sachar BD, Aufses AH. Management of severe hemorrhage in ulcerative colitis. Am J Surg 1990; 19: 550-5.
33. Richter JM, Christensen MR, Kaplan LM. Effectiveness of the current technology in the diagnosis and management of lower gastrointestinal hemorrhage. Gastrointest Endosc. 1995 Feb; 41(2): 93-8.
34. den Hartog Jager FC, van Haastert M, Batterman JJ, et al. The endoscopic spectrum of late radiation damage of the rectosigmoid colon. Endoscopy. 1985 Nov; 17(6): 214-6.
35. Wagner HE, Stain SC, Gilg M, et al. Systematic assessment of massive bleeding of the lower part of the gastrointestinal tract. Surg Gynecol Obstet. 1992 Nov; 175(5): 445-9
36. Vellacott KD. Early endoscopy for acute lower gastrointestinal hemorrhage. Ann R Coll Surg Engl. 1986 Sep; 68(5): 243-4.
37. Richard WO, Grove-mahoney D. Hemorrhage from a dieulafoy type ulcer of the colon. Am Surg. 1988; 54: 121-4.
38. Beach PM, Risly TS. Aortosigmoid fistulazation following aortic resection. Arch Surg. 1966; 92: 805-7.
39. Jensen DM, Machicado GA. Diagnosis and treatment of severe hematochezia. The role of urgent colonoscopy after purge. Gastroenterology. 1988 Dec; 95(6): 1569-74.
40. Dusold R, Burke K, Carpentier W, et al. The accuracy of technetium-99m-labeled red cell scintigraphy in localizing gastrointestinal bleeding. Am J Gastroenterol. 1994 Mar; 89(3): 345-8.

41. Kuo WT, Lee DE, Saad WE, et al. Superselective microcoil embolization for the treatment of lower gastrointestinal hemorrhage. J Vasc Interv Radiol. 2003 Dec; 14(12): 1503-9.

42. Green BT, Rockey DC, Portword G, et al. Urgent colonoscopy for evaluation and management of acute lower gastrointestinal hemorrhage: A randomized controlled trial. Am J Gastroenterol. 2005 Nov; 100(11): 2395-402.

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