

Endoscopic Management of Acute Lower Gastrointestinal Bleeding

Alberto Tringali

Alberto Tringali, Department of Surgery, Endoscopy Unit, Ospedale Niguarda, Piazza Ospedale Maggiore 3, 20162 Milan, Italy

Correspondence to: A Tringali, Department of Surgery, Endoscopy Unit, Ospedale Niguarda, Piazza Ospedale Maggiore 3, 20146 Milan, Italy

Email: alberto.tringali@ospedaleniguarda.it

Telephone: +390264442770

Fax: +390264442911

Received: December 2, 2014

Revised: January 15, 2015

Accepted: January 18, 2015

Published online: March 21, 2015

ABSTRACT

Lower gastrointestinal bleeding (LGIB) continues to be a problem for physicians. Acute LGIB is defined as bleeding that emanates from a source distal to the ligament of Treitz. Although 80% of all LGIB will stop spontaneously, the identification of the bleeding source remains challenging and rebleeding can occur in 25% of cases. Diverticular bleeding remains the most common cause of lower GI bleeding. Lower Gastrointestinal Bleeding encompasses a wide clinical spectrum ranging from occult bleeding to overt hematochezia until massive hemorrhage with shock requiring emerging hospitalization. Some patients with severe hematochezia require urgent attention to minimize further bleeding and complications. Colonoscopy is the diagnostic procedure of choice in most patients with Lower GI bleeding and its role in the treatment of lower GI bleeding has been shown to be an efficacious and safe method even if a therapeutic endoscopy occurred in about 30% of patients. The optimal timing of colonoscopy in LGIB remains to be determined. CT angiography is used in the setting of acute Lower GI bleeding correctly depicts the presence and location of active or recent hemorrhage, as well as the potential cause, in about 80-85% of case. Nuclear scintigraphy has been proposed as a diagnostic screening prior to angiography, increasing the likelihood of positive angiographic results or as a tool for localization for surgery but had multiple limitations. Superselective mesenteric angiography remains the cornerstone of management of patients with acute LGIB but it is an invasive and time-consuming procedure. Emergent surgery should be considered only as a last resort and is rarely needed to prevent death from exsanguination. The golden standard for surgical treatment of acute severe LGIB should be directed

segmental resection based on aggressive preoperative identification of the bleeding site. This article reviews the causes, clinical presentation, diagnostic methods, endoscopic treatment of LGIB and management of specific LGI bleeding lesions.

© 2015 ACT. All rights reserved.

Key words: LGIB-Gastrointestinal bleeding; Colonoscopy; Review-Hematochezia-Angiography; Lower GI bleed; Hemorrhage; Tagged red blood cell scan; Surgery; CT scans

Tringali A. Endoscopic Management of Acute Lower Gastrointestinal Bleeding. *Journal of Gastroenterology and Hepatology Research* 2015; 4(3): 1486-1500 Available from: URL: <http://www.ghrnet.org/index.php/joghr/article/view/1119>

DEFINITION

Acute Lower Gastrointestinal bleeding (LGIB) refers to blood loss from the gastrointestinal tract of recent onset emanating from a location distal to the ligament of Treitz and resulting in instability of vital signs, anemia and/or need for blood transfusion^[1].

INTRODUCTION

Lower Gastrointestinal bleeding (LGIB) is approximately one-fifth as common as upper GI bleeding and accounts for approximately 20 to 30 hospitalizations per 100,000 adults per year^[2,3].

The incidence of hospitalization for lower GI complications (primarily bleeding) has increased by >50 % in a decade, from 20/100,000 in 1996 to 33/100,000 person in 2005, whereas the incidence of hospitalizations for upper GI complications has decreased by almost 50%, from 87/100,000 to 47/100,000. In addition, lower GI complications had a higher mortality, longer hospitalization, and higher resource utilization than did upper GI complications. Thus, LGIB represents a serious and increasingly important problem for patients and gastroenterologists^[4]. LGIB is a more significant problem in males^[5] and elderly patients, with a greater than 200 fold increase in incidence in 80 years old compared with younger patients. The rise in incidence with age may be

explained by the increasing prevalence of diverticulosis, colonic angiodysplasia, neoplasms and ischemic colitis^[6]. Although 80% of all LGIB will stop spontaneously, the identification of the bleeding source remains challenging and rebleeding can occur in 25% of cases^[7]. Prognosis in LGIB varies.

However, since most acute LGIB is self-limited, outcomes are usually favourable. Indeed, the mortality associated with LGIB, is generally considered to be less than 5% compared with 23% in patients who developed LGIB while hospitalized for another reason. The mortality is often a result of comorbid conditions^[2,3].

ETIOLOGY

The causes of Lower GI Bleeding may be arbitrarily grouped into several categories: Anatomic (diverticulosis); Vascular (Angiodysplasia, Ischemic); Inflammatory (infectious, IBD), Neoplastic (colon adenocarcinoma) post-therapeutic intervention (post-polypectomy, post-surgical intervention). The commonest colonic causes are listed in table 1.

The small bowel or upper origin are less common. The diverticular bleeding are the most common causes of acute LGIB.

CLINICAL PRESENTATION

Lower gastrointestinal bleeding tends to be less severe in presentation than Upper GI Bleeding and 80-85% of patients with Lower Gastrointestinal Bleeding will stop spontaneously. Lower Gastrointestinal Bleeding encompasses a wide clinical spectrum

Table 1 Causes of Lower GI Bleeding.

	Causes	Frequency
Colorectal (80%)	Diverticular disease	5.2-42%
	Angiodysplasia	1.2-4%
	Neoplasm	2.9.19%
	Inflammatory bowel disease	
	Ischemic colitis	7-18%
	Infectious colitis	2.6%
	Radiation proctitis	9-13%
	Anorectal disease (haemorrhoids, fissurae)	20%
	Post-polypectomy/post-anastomotic bleeding	0-12.8%
	AV malformations	
Small bowel source (10%)	Meckel diverticulum	
	IBD	
UGI source (10%)	Neoplasia	
	Vasculitis	
	Ulcer	
	Neoplasm	

Table 2 Level of diagnostic certainty for acute colonic bleeding*.

Level I: Definitive diagnosis	A: Actively bleeding lesion found at endoscopy (anoscopy, sigmoidoscopy, or colonoscopy) or angiography; B: Stigmata of recent bleeding (nonbleeding visible vessel, adherent clot) found at endoscopy; C: Positive tagged red blood cell (TRBC) scan if verified by IA or IB.
Level II: Presumptive diagnosis/circumstantial evidence	A: Fresh blood localized to colon segment inhabited by potential bleeding source; B: Positive TRBC scan localizing to the colon and colonoscopy that shows potential bleeding site in area of positive scan; C: Bright red blood per rectum confirmed by objective color testing and colonoscopy that demonstrates single potential bleeding source in colon, complemented by negative upper endoscopy.
Level III: Equivocal diagnosis	A: "Hematochezia" or blood per rectum (without color specification) and colonoscopy that demonstrates 1 or more potential bleeding sources.

*Presurgical evaluation.

ranging from occult bleeding to overt hematochezia until massive hemorrhage with shock requiring emerging hospitalization. A mild-moderate Lower GI bleeding occur in about 85-90% of cases 10-15% of cases had a severe presentation with persistent or recurrent bleeding with hemodynamic effect (tachycardia, hypotension), drop in Hb levels (> 2 gr /dL) and need hospital admission.

LGIB can be classified as acute or chronic depending on the duration of symptoms.

Acute LGIB defined as bleeding of recent duration (<3 days) that may result in hemodynamic instability, anemia, and/or the need for blood transfusion and may be massive requiring urgent investigations and management.

Chronic LGIB is the passage of blood per rectum over a period of several days or longer and usually implies intermittent or slow loss of blood and can either present with episodic rectal bleeding or insidiously, with iron-deficiency anemia or positive FOBT.

Zuckermann *et al* has described a criteria to establish the diagnosis of acute lower GI bleeding distinguishing a level I as a definitive diagnosis, a level II as a presumptive diagnosis and level III with equivocal diagnosis^[8]. The criteria of diagnosing acute lower GI bleeding are presented in table 2.

MANAGEMENT OF LGIB

A) INITIAL EVALUATION AND TRIAGE

Initial evaluation of patient presenting with acute Lower gastrointestinal bleeding consists of a focused history and physical examination, ordering the appropriate blood tests, assessing the severity of bleeding, providing the necessary resuscitation, measures and blood transfusions, withholding particular drugs (eg, anticoagulant, antiplatelets drugs, NSAIDs), correcting coagulation defects and triaging the patient to the appropriate level of care (outpatient vs ward vs intensive unit care).

Elements in the history can direct the assessment toward a cause of probable or high likelihood such as post-polypectomy bleeding in a patient who recently underwent polypectomy, exacerbation of known inflammatory bowel disease or ischemic colitis in patients with known ischemic vascular disease. Although most overt LGI bleeding episodes will manifest as hematochezia (fresh blood and clots per rectum) indicating a distal source, a melanic stools can occur in the setting of bleeding from proximal source of right colon and cecum.

Most importantly hematochezia associated with hemodynamic instability should prompt consideration for brisk bleeding from an upper GI source, particularly when risk factors such as a prior history of bleeding peptic ulcer or NSAID use are present. Nasogastric tube lavage is performed and a positive or non diagnostic (non bilious, non blood) aspirate for blood prompt emergent upper endoscopy especially in risk patients. Upper endoscopy should be also performed in cases when a source is not identified at colonoscopy. The decision to manage in an outpatient setting or to admit to the intensive care unit depends on several factors and clinical judgement.

OUTCOMES

In contrast to UGIB, predictive factors of poor outcome in LGIB are not defined as well.

Due to most episodes of LGIB will stop spontaneously, the early identification of high risk patients would allow the selection of those patients most likely to benefit from urgent therapeutic interventions.

Strate and coworkers retrospectively collected data on 24 clinical variables available in the first 4 hours of evaluation in 252

A Tringali. Endoscopic management of LGIB

consecutive patients. Independent correlates of severe bleeding were as follows: heart rate 100 beats per min; systolic blood pressure no more than 115 mmHg; syncope; non-tender abdominal examination; bleeding per rectum during the first 4 hours of evaluation; aspirin use; and more than 2 active comorbid conditions^[9]. Independent risk factors of severe bleeding are summarised in table 3.

Thirty-seven (84%) of 44 patients with more than 3 risk factors, 85 patients (43%) of 197 with 1 to 3 risk factors and 1 (9%) of 11 with no risk factors experienced severe LGIB.

The primary and secondary outcomes of LGIB reported in different studies are shown in table 4.

Velayos and colleagues^[10] analysed prospectively all patients admitted at emergency department for lower gastrointestinal bleeding. Thirty-seven patients (39%) had severe lower gastrointestinal bleeding. Independent risk factors for severe lower gastrointestinal bleeding were initial hematocrit <35%; presence of abnormal vital signs (systolic blood pressure <100 mm Hg or heart rate >100/min) 1 hour after initial medical evaluation; and gross blood on initial rectal examination. Nineteen patients (20%) experienced a significant adverse outcome, including three deaths. The main independent predictor of adverse outcomes was severe lower gastrointestinal bleeding (OR 5.3; 95% CI, 1.7-16.5). The incidence of severe LGIB increased with the number of independent risk factors. Severe LGIB occurred in 11 of 14 patients (79%) with 3 risk factors, 20 of 35 (57%) patients with 2 risk factors, 6 of 36 (17%) with 1 risk factor, and 0 of 9 patients with no risk factors.

The prognostic model presented by Strata^[9] was externally validated on 275 patients and it successfully stratified patients into risk groups-patients with three or more risk factors were at high risk of severe bleeding (80%), those at 1-3 risk factors were at moderate risk (45%) and those with no risk factors at low risk (<10%). In addition this study showed a higher risk of surgery and death in high risk patients as shown in table 5^[11]. Therefore high risk patients can be identified early and triaged to urgent investigations and management.

Finally in a national US audit presented by Strate *et al*^[12] were identified a risk factors of death that are summarised in table 6.

Two other scores have been proposed. The first was published by Kollef and coworkers^[13] where patients were stratified into low and high risk according to the established criteria that were ongoing bleeding, low systolic blood pressure (>100 mmHg) elevated prothrombin time (> 1.2 times the control value), altered mental status and presence of an unstable comorbid disease. It is a cohort study evaluating an outcome prediction tool for clinical use in patients with either acute upper and lower GI bleeding. The main outcome measure was the occurrence of an in hospital complication, the source of haemorrhage and hospital mortality. This score may be used at the time of initial evaluation in emergency room to assign patient risk for the development of in hospital complication but it is not valid for discharge patients.

Another more recent study, evaluating a new score model, was published by Das *A et al*^[14]. They used, on the basis of positive results of other studies, an artificial neural network (ANN) as an accurate and reliable method in diagnosis and outcome prediction in acute lower gastrointestinal bleeding.

An artificial neural network (ANN) is a computer-based decision support systems characterized by a set interconnected equations that use a statistical analysis to reveal previously unrecognized relationships between given input variables and an output variable.

The network is "trained" to recognize patterns when presented with input variables from a representative population with a known outcome. Once appropriate training is completed, the network attempts to predict with a high degree of accuracy outcomes that may not have been apparent by conventional predictive models.

ANN models were constructed by use of a commercial neural network program (Statistical Neural Networks, version 5.5; Statsoft, Tulsa). Two different sets of ANN were built, each with 27 clinical measures as input variables and two different output variables (major SRH and need for endoscopic therapy).

They have proposed the use of ANN for predicting clinical outcome in a group of patients presenting with acute lower GI bleeding by using of 27 clinical non endoscopic parameters which are not routinely available to clinicians at the time of triage. Anyway there are some limitations to the ANN-based model. First ANN was

Table 3 Independent risk factors for severe Lower GI tract bleeding.

Predictor	OR (95% CI)	P Value
Heart rate >100 /min	3.67(1.78-7.57)	<0.001
Systolic blood pressure < 115 mmHg	3.45 (1.54-7.72)	0.003
Syncope	2.82 (1.06-7.46)	0.04
Non-tender abdominal examination	2.43 (1.22-4.85)	0.01
Bleeding in the first 4 h of evaluation	2.32 (1.28-4.20)	0.005
Aspirin use	2.07 (1.12-3.82)	0.02
>2active comorbid conditions	1.93 (1.08-3.44)	0.02

Table 6 Risk of death.

Risk factors	OR
Age > 70	4.9
Intestinal ischemia	3.5
>2 comorbidity	3.0
Nosocomial bleed	2.4
Coagulopathy	2.3
Hypovolemia	2.2
Transfusion	1.6
Men	1.5

Table 4 Outcomes in different studies.

Author/year	N	Continued o rebleeding	Died	Surgery	RBC transfusion	LOS (days)
Strate 2003	252	7%	2.4%	3.6%	2.0 (3.0)	4.3
Schmulewitz 2003	565	11%	3%	5%	3.1 (3.9)	6.7
Das 2003	332	19%	5%	Nr	2.2	4.4
Strate 2005	275	nr	4%	2.6%	2.5 (4.5)	

Table 5 Outcomes based on risk stratification.

	Severe bleeding	Surgery(%)	Death (%)	LOS (days)	Transfused RBC (median units)
Low risk=0 factors	<10%	0	0	2.8	0
Moderate risk= 1-3 risk factors	45%	1.5	2.9%	3.1	1
High risk ≥ 3 risk factors	80%	7.7%	9.6%	4.6	3

not tested prospectively in a community Hospital. Second ANN is more expensive to use than some of simpler numerical scoring systems. Finally reason was the low positive predictive value in the prediction of mortality.

It is vitally important to understand that the use of ANN based computer models is only meant to assist in the decision-making process of the physician and not to replace the physician. Moreover, the use of neural network models is not meant to be a substitute for the judgment of experienced clinicians. On the contrary, it is paramount that the final medical decision is left up to the experienced clinician.

Triage and optimal length of stay

Data are scarce compared with upper GI bleeding. Expert opinion suggest to admit in ICU for 24 hrs and in ward for 72 hours high risk patients; hospital for 24-48 hrs with early refeeding patients with moderate risk and feed and early discharge patients with low risk.

A proposed approach to LGIB that are admitted in emergency room (ER) is shown in figure 1.

DIAGNOSIS: LOCALIZING THE SOURCE OF BLEEDING

A) Colonoscopy

Timing and Preparation for Colonoscopy

Historically, colonoscopy was performed electively due to the need for colon preparation and concern regarding complications. Over

the last two decades, a number of studies have indicated that urgent colonoscopy, defined as colonoscopy performed within 12-24 h of admission, is safe and may facilitate the identification and treatment of bleeding lesions^[15-17].

However, studies comparing this approach to delayed colonoscopy for LGIB are limited.

Moreover, urgent colonoscopy is logistically complicated, stigmata of hemorrhage are arguably difficult to identify and there a number of other potential diagnostic tools to choose (radionuclide scintigraphy, CT multidetector). Colonoscopy is attractive in LGIB because it provides the best opportunity for early diagnosis, triage and treatment. The possibility is supported by two studies demonstrating that the length of time from presentation to colonoscopy is an independent predictor of hospital length of stay^[18]. Nonetheless, there is a great controversy about the timing of colonoscopy. Urgent colonoscopy appears to be safe, and provides a specific diagnosis in a high proportion of patients (range from 69-89%)^[15-18,20-22] but notably the source of bleeding cannot be definitively identified in up to 25% of patients^[2,18] as reported in table 7.

However, outcome data supporting its use are lacking.

In a randomized controlled trial designed to address this issue, urgent colonoscopy was compared with a standard care^[15]. In this study a definitive source of bleeding was found more often in urgent colonoscopy patients than in the standard care group but there were no difference in terms of important outcomes including hospital stay, transfusion requirements, early or late rebleeding, surgery or mortality. It is also noteworthy that the definition of “urgent” and timing of procedures vary greatly both in clinical practice and in published reports.

If abnormalities are more likely to be found during colonoscopy when it is performed urgently than when it is performed expectantly, it would follow that endoscopic therapy would be more likely in this circumstances. Surprisingly only 10-15% of patients undergoing urgent colonoscopy had some form of endoscopic therapy^[22]. Recently Laine *et al*^[24] present a randomized trial of urgent vs elective colonoscopy in patients with LGIB.

Seventy-two patients with severe LGIB were randomized 1:1 to colonoscopy within 12 h of admission or elective colonoscopy within 36-60 h. The authors found no differences in the primary outcome, rebleeding during hospitalization or secondary outcomes, including number of units of blood transfused, number of diagnostic and therapeutic interventions, length of hospital stay and conclude that urgent colonoscopy does not improve outcomes in patients with serious LGIB. However the limited number of patients and the fact that patients in the urgent colonoscopy arm appeared to have more severe bleeding than those undergoing elective examinations make it

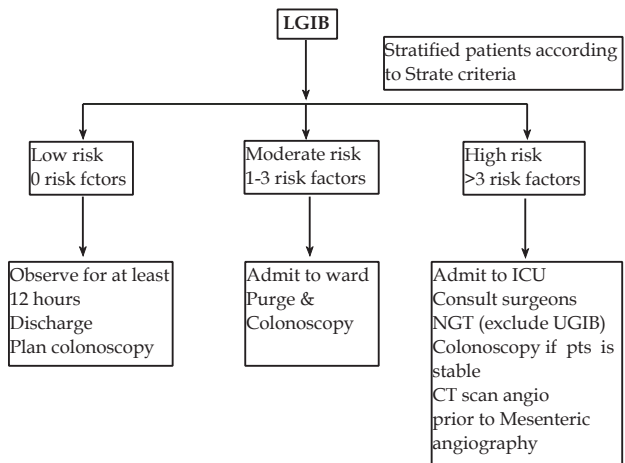


Figure 1 A proposed Approach to LGIB.

Study (year)	N	Specific diagnosis, N (%)	Endoscopic therapy, N (%)	Complications
Caos ^[24] , J Clin Gastroenterol; 8(1): 46-9	35	24/35 (77%)	11/35 (31%)	0%
Jensen DM ^[15] , Gastroenterology 1988; 95: 1569-74	80	59/80 (74%)	31/80 (39%)	nr
Chaudhry ^[20] , Am Surg 1998; 64(8): 723-8	85	82/85 (97%)	17/27 (62%)	1%
Kok ^[19] 1998	190	190 (100%)	19/190 (10%)	0%
Jensen ^[25] , Gastroenterology; 2000	121	116/121 (96%)	12/121 (10%)	nr
Ohyama T ^[26] , Digestion 2000; 61(3):189-92	345	307/345 (89%)	227/345 (67%)	22/345 (6%)
Angtuaco TL ^[22] , AMJ gastroenterol 2001; 96(6): 1782-5	39	28/39 (74%)	4/39 (10%)	-
Al Qahtani ^[27] , Worl J Surg 2000	152	59/152 (45%)	NR	NR
Strate ^[9] , Arch Intern med 2003; 163(7): 838-843	144	128/144 (89%)	NR	NR
Schulmlewitz ^[18] , GIE 2003; 58(6): 841-6	415	369/415 (89%)	21/42 (50%)	Nr
Green ^[15] , AM J Gastroenterol 2005	50	21/25 (84%)	17/25 (68%)	2%
Laine ^[24] , AM. J gastroenterol 2010; 105(12): 2636-41	36+36, 72	27/36 (75%)	4/36 (12%)	nr
Total	521	1410/1667 (85%)	363/940(38%)	0-6%

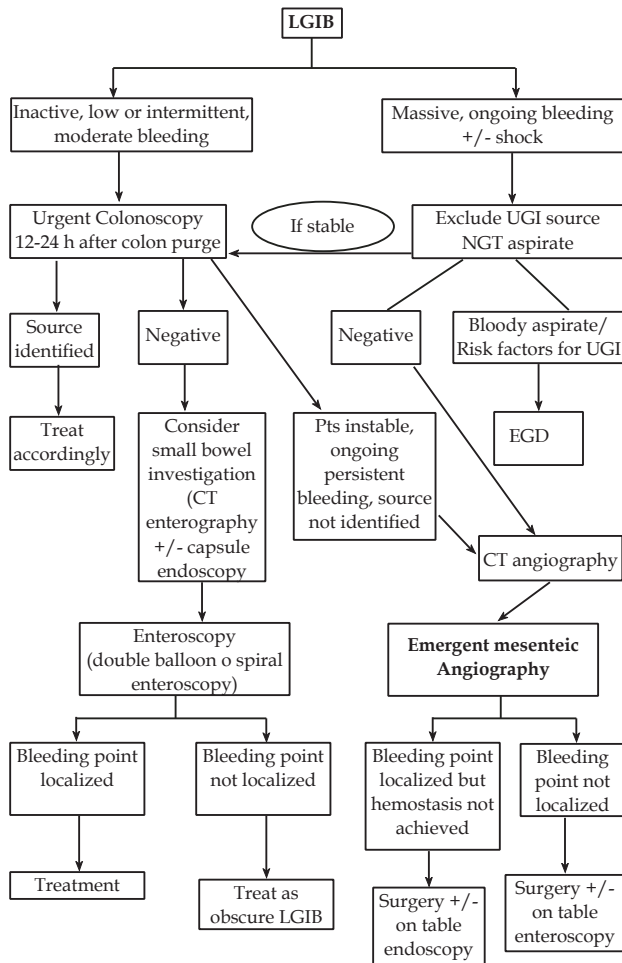


Figure 2 A proposed algorithm for management of acute LGIB.

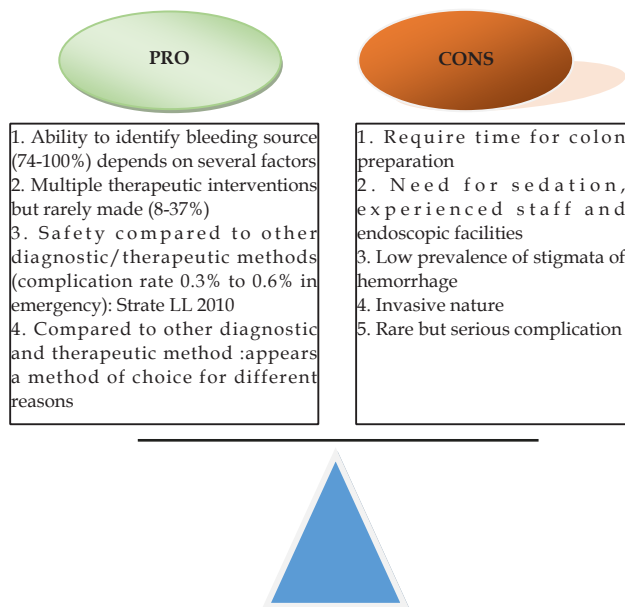


Figure 3 Pros and Cons of Colonoscopy in the management of Lower GI bleeding.

difficult to draw conclusion regarding the utility of urgent vs elective colonoscopy in LGIB. The optimal timing of colonoscopy remains to be determined. The diagnostic yield of colonoscopy ranged from 45% to 97% (median 88%).

Advantages and disadvantages of colonoscopy in the management of lower gastrointestinal bleeding are summarised in figure 3.

Many studies have reported the specific diagnosis during colonoscopy and also the availability to perform endoscopic treatments^[9,15,16,18-20,22,24-27] as seen in table 6. Regardless of the timing of colonoscopy, according to ASGE guideline, the consensus opinion is that colonoscopy is the diagnostic procedure of choice in most patients with Lower GI bleeding^[1,28].

COLONOSCOPIC PROCEDURE

Usually a standard colonoscope is used because of the larger (3.7 mm) working channel facilitates clearing of blood, clots and residual stool material and enables passage of larger diameter hemostatic devices. The goal of the procedure is to identify a bleeding site more than perform a cecal intubation. Meticulous examination is performed both during insertion and withdrawal of the scope because of intermittently bleeding lesion.

Areas containing or accumulating fresh blood or clots are vigorously washed and investigated as they are a potential bleeding site. Maximum dexterity is required when performing colonoscopy in patients with acute severe LGIB to negotiate the scope through the blood, clots and diverticular segments to avoid perforation. Examination under water immersion has been demonstrated useful technique to identify the nature and site of a bleeding lesion^[29]. The irrigation device is useful for removing adherent material from the mucosa and/or pinpointing a bleeding site for targeted therapy. Unless a definitive bleeding colonic lesion is identified or a specific cause is sought, every attempt should be made to intubate the terminal ileum. The colon should be rapidly purged with 4-6 L of polyethylene glycol (PEG) based solution given at rate of 1 L every 30 minutes by nasogastric tube. Time required to obtain a bowel preparation is 3.4 hours. Unless contraindicated, the prokinetic drug metoclopramide (10 mg iv) is administered prior to the purge to hasten bowel transit and control nausea and vomiting. Colonoscopy can generally be performed within 1-2 h upon completion of the preparation.

Nuclear scintigraphy

Nuclear scintigraphy has been proposed as a diagnostic method prior to angiography, increasing the likelihood of positive angiographic results or as a toll for localization for surgery. The limitation of scintigraphy depends on several factors. First is a variable technique, second has a variable threshold for performing study, third variables times to angiography or surgery and finally present variable criteria for determining "accurate" localization. We identified 26 studies evaluating the accuracy of scintigraphy in the LGIB and showing a 78% accuracy (range 40-100%) as shown in table 8.

A study by NG and coworkers evaluating scan as a prelude to angiography in 160 patients from 1989 to 1994, showed that an early blush (< 2 min) identify during the scan has a higher angiographic positive rates (92%) compared to late blush (after 2 min) (60%)^[30].

Computes Tomography Angiography (CTA)

CT has been evaluated as highly effective to detect vascular ectasia. The potential advantages are that it is non-invasive, simple to use and less costly than conventional angiography. CT angiography performed in the emergency setting in patients with acute lower

intestinal bleeding is feasible and correctly depicts the presence and location of active or recent hemorrhage, as well as the potential cause, in about 80-85% of cases^[31,32,33].

Therefore although just emerging as the diagnostic option the advantage of CT scan is that it can localize the source of bleeding rapidly and guide therapy including endoscopy, mesenteric angiography and eventually surgery.

A comparison of different methods of diagnosis is shown in table 9.

MANAGEMENT OF LGIB

Endoscopic Treatments: Endoscopic treatment modalities include injection, contact thermal coagulation, argon plasma coagulation, clipping and band ligation. The use of one or a combination of these techniques depends on the site and features of the bleeding lesions, operator choice and familiarity with the device and type of access to the bleeding site. Hemostatic spray has been demonstrated efficacy method to stop bleeding either in upper and lower GI bleeding. Some case report or case series have been published. The more recent studies as a preliminary data using hemospray in lower GI bleeding showed that Hemostatic spray can be effective in the management of LGIB, but suggest cautious use for patients on antithrombotic therapy and spurting bleeds^[34]. Thus hemostatic spray remain a method that need further investigation before firm conclusion can be drawn. Advantages and disadvantages of endoscopic treatment are shown in table 10.

Management of Specific LGI Bleeding Lesions

Diverticular bleeding

Diverticular bleeding is a common cause of acute LGIB accounting for approximately 40% of cases^[35]. The incidence of bleeding ranges from 5% to 50% in patients with diverticulosis^[17,35], while in those with stigmata of recent bleeding, the risk of rebleeding is about 53% with need of emergency surgery in about 35%^[17,36,37]. Bleeding is arterial occurring either at the neck or dome of the diverticulum (Figure 7). It is usually associated with painless hematochezia.

Table 8 99m Tc RBC for LGIB: recent studies.

Author Year	Total scan	Positive scans	Correct localization	Positive angiograms
Alavi 1980	43	54%	48%	43%
McKusick KA 1981	-	-	83%	-
Winzelberg GG 1982	-	-	83%	-
Markisz 1982	50	34%	91%	36%
Winn 1983	82	16%	100%	38%
Kester RR 1984	62	60%	67%	22%
Bunker TG 1984			95%	
Szasz JJ 1985			81%	
Orecchia 1985	76	34%	94%	-
Nicholson ML 1989	43	72%	97%	-
Leitman 1989	28	43%	86%	50%
Hunter 1990	203	26%	41%	44%
Voeller 1991	59	32%	64%	-
Bentley DE 1991	-	47%	52%	-
Miller GA 1991	-	-	52%	-
Bearne P 1992	-	78%	82%	-
Whitaker SC 1993	-	-	80%	-
Gupta N 1995		-	78%	
Rantis 1995	80	48%	73%	-
Suzman 1996	224	51%	78%	44%
NG 1997	160	54%	-	43%
Gutierrez C 1998	105	40%	88%	-
Levy 2003	40	70%	47%	0%
Olds 2005	127	39%	48%	42%
Rantis 1995	80	48%	73%	-
Suzman 1996	224	51%	78%	44%
NG 1997	160	54%	-	43%
Gutierrez C 1998	105	40%	88%	-
Levy 2003	40	70%	47%	0%
Olds 2005	127	39%	48%	42%

Table 9 Comparison by different method of diagnosis of LGIB.

Procedure	Diagnosis	Therapy	Early rebleeding	Major complication	Colon prep	Require active bleeding
Colonoscopy	74-100%	8-37%	0-24%	0-2%	Yes	No
Multidetectors scan	24-94%	-	-	0-11%	No	Yes
Radionuclide scan	40-73%	.	.	Rare	No	Yes
Angiography	23-72%	14-100%	1-57%	0-60%	No	Yes

Table 10 Advantages and disadvantages of endoscopic treatment.

	Advantages	Disadvantages	Setting
Injection therapy			
Adrenaline	Easy to use	Rapidly disappear	
Thermal therapy			
Contact (Bicap/Goldprobe)	Easy to use and to apply also in difficult location	Higher risk of perforation	7-10 watt 1-2 s pulse duration 15-40 watt 1 l/min (argon flow)
Non contact (APC)	Easy to use and apply also in difficult location	No for diverticular bleeding, risk of perforation in thin wall (cecum, small bowel)	10-30 pulsed 2 (thinner wall precise mode) Brief and repeated aspiration of gas should be performed throughout the procedure to avoid overdistension
Mechanical therapy			
Band ligation	efficacy	Need to remove and reinsert the scope and tattooing the site	
Ovesco	efficacy	Need to remove and reinsert the scope	
Clip	efficacy	Difficult to place in some locations	
New technique			
Hemospray	Easy to use and apply also in difficult situation	Occlusion of delivery system	

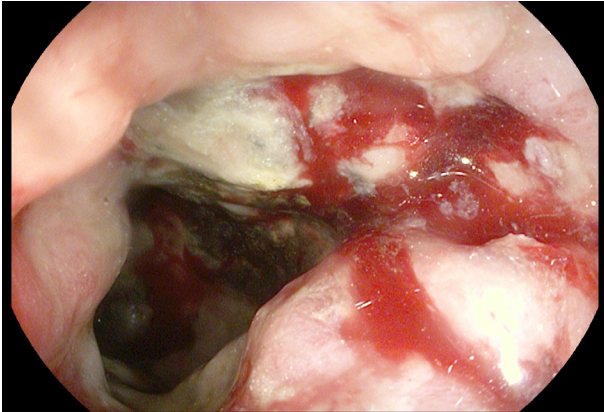


Figure 4 Colon cancer bleeding.

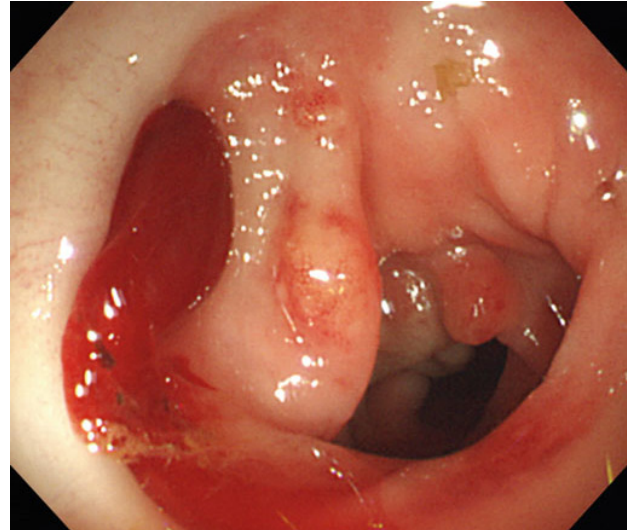


Figure 7 Diverticular bleeding.

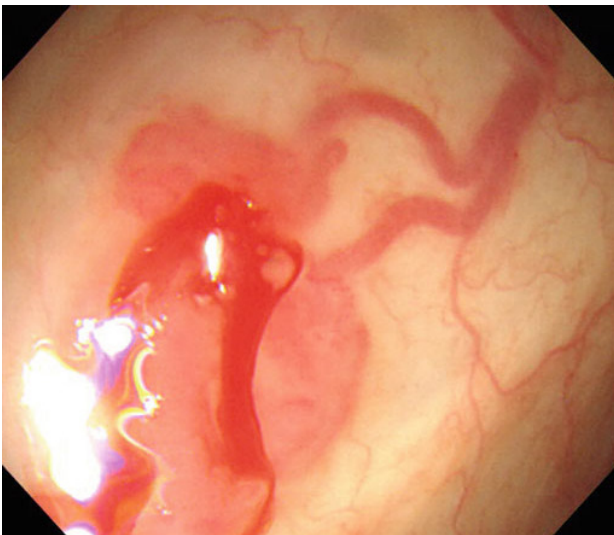


Figure 5 Bleeding from Angiodysplasia.

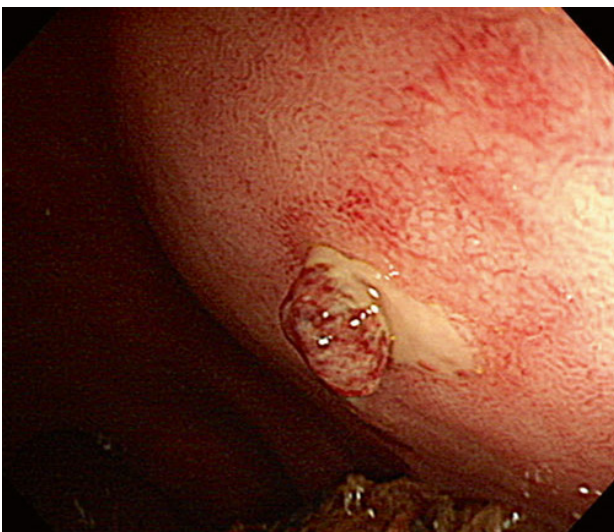


Figure 6 Dieulafoy.

Diverticular bleeding ceases spontaneously in about 75-80% of cases at the time of endoscopy but can recur in 10% to 40% of cases^[38].

Despite the frequency of diverticular bleeding there is a paucity of prospective clinical data on specific treatment strategies, and moreover, the ones that are published are primarily case series, and small, often nonrandomized studies.

Endoscopic treatment of diverticular bleeding with active bleeding, non bleeding visible vessel or an adherent clot is treated with paradiverticular injection of a 1:10,000 dilution of epinephrine in 0.5 aliquots. A submucosal injection of 1-2 mL in 2-4 site around the diverticulum is often sufficient^[39-42]. If the bleeding vessel is identified should be cauterized with bipolar probe (large probe 10-15 W- 1-2s pulse) with light-to moderate tamponade pressure avoiding the risk of perforation which is substantially higher when the vessel is located at the base of the diverticulum. In this circumstance, clipping of the visible vessel or entire diverticulum might be a safer alternative^[43].

Adherent clot is usually washed and suctioned or, if resistant, removed with a basket or snare with prior injection of epinephrine at the base of the clot. The exposed vessel is then coagulated or clipped. More recently, the efficacy of band ligation for diverticular bleeding has been demonstrated. Farrell *et al*^[44], based on previous work of Witte *et al*^[45], have reported cases of diverticular bleeding treated by elastic band ligation and suggested that this might be a promising method for the hemostasis. A study by Setoyama showed that endoscopic band ligation is superior to endoclip for the treatment of diverticular haemorrhage in term of rebleeding rate (33% vs 6%). The study was a prospectively non-randomized and compared 18 patients treated by EBL versus 48 patients treated by endoclip. Despite the better results in EBL groups, the limitation of the study does not permit a firm conclusion^[46].

It is useful to tattoo the site of the diverticular bleeding for future localization in case of rebleeding or in case of reinsertion of the scope to perform band ligation which could be useful in case of narrowed lumen associated to diverticular disease that may impair use of clipping and thermal coagulation probe.

Angiodysplasia: Colonic arteriovenous malformation is thought to result from intermittent low-grade obstruction of submucosal veins, because they penetrate the muscle layer of the colon and cause small

arteriovenous communications^[47]. Sporadic vascular ectasia accounts for approximately 10% of cases and are commonly found in elderly patients localized in the right colon^[48]. Intestinal AVM are classified into 3 types based on angiographic characteristics, localization, age of the patient and family history.

Type 1 AVMs are solitary, localized lesions within the right side of the colon and usually occur in older patients. Type 2 AVMs are larger, occasionally visible, most commonly in the small intestine, and probably of congenital origin. Type 3 arteriovenous malformations are punctate angiomas causing gastrointestinal hemorrhage.

Typically the bleeding caused by angiodysplasia is chronic, slow and intermittent as shown in figure 5. The bleeding stops spontaneously in 85-90% of cases but it recurs in 25-85%^[49]. The characteristic endoscopic appearance is of a red, flat lesion (2-10 mm diameter) with dilated blood vessels radiating from a central feeding vessel. The use of narcotics during colonoscopy may mask the detection of these lesion by reducing mucosal blood flow^[50,51].

Endoscopic management of actively bleeding angiodysplasia is straightforward, decision making may be more difficult in patients who have stopped bleeding and are found to have non bleeding angiodysplasia.

In this case seems to be reasonable to ablate angiodysplasia in the absence of another definitive bleeding site.

Contact and non contact thermal techniques are effective at treating vascular ectasias.

Bipolar probe should be used with lower power setting about 10-12 watt, 2-3 pulses duration, mild probe-tissue contact pressure and application are repeated until the lesion is completely obliterated and haemostasis achieved.

Coagulation complications can be expected in 5% of treated patients and they include recurrent delayed haemorrhage due to post-coagulation ulceration and post-coagulation syndrome.

APC is the preferred method due to its easy of use and more speedy application especially when multiple lesions are present. It is important to avoid thinning of the colonic wall with excessive air insufflation as this increases the risk of perforation during therapy. Although some recommend ablation starting at periphery I usually prefer target the center vessel as ensuing coagulation and edema will efface the peripheral branches and limit the extent of coagulation.

Post-polypectomy bleeding: May occur during the procedure or may be delayed up to 1 month after the polypectomy. Risk factors include the removal of large sessile right sided colon polyps as well as resumption of anticoagulation. Approximately 70% stop spontaneously. A recent case control study including 4,592 patients who underwent colonoscopy with polypectomy showed that a polyp diameter and resuming anticoagulation were strongly associated with increased risk of severe delayed post-polypectomy bleeding^[52]. Another study by Watabe^[53] showed that hypertension is a significant risk factors for delayed colorectal post-polypectomy haemorrhage.

Therefore based on evidence that some groups of patients are at higher risk of delayed post-polypectomy bleeding it has been suggested that endoscopic clipping is a useful technique for prevention bleeding in high risk patients.

The role of endoscopic clipping in average risk patients is still controversial. One randomized controlled trials^[54] showed that clipping did not decrease the occurrence of delayed bleeding after endoscopic polypectomy.

A cost efficacy decision analysis of prophylactic clip placement after endoscopic removal of large polyp showed that endoscopic clipping after polypectomy appears to be a cost-effective strategy

for patients who receive anti-platelet or anticoagulation therapy. The use of prophylactic clipping in other situation are based on personal opinion or experience but are not evidence based approach.

In my unit we placed clip not only in high risk patients but also after resection of large sessile polyp located in the right colon with evident visible vessel after resection.

In case of bleeding the choice method is based on clipping of the bleeding point, with or without epinephrine injection because it does not extend tissue injury as with thermal therapy.

When feasible, a tangential rather than perpendicular approach to the lesion and the application of suction prior to clip closure are maneuver that allow the capture of more tissue between the two prongs. In circumstances with limited scope maneuverability and difficulty in assessing a particular lesion, the use of rotatable clip allow orientation of the clip in a favorable position.

Futhermore hemospray has been showed to be efficacious and safe procedure but need more studies.

Finally the use of endoloop has been proposed as efficacious and safe method to prevent bleeding before performing resection of pedunculated polyp^[55-58] especially useful in high risk patients.

Colitis: Several colitis can present with acute LGIB, including inflammatory bowel disease, infectious disease and ischemic colitis. Features in the history, clinical presentation, biochemical data and endoscopic assessment with biopsy typically lead to the definitive diagnosis.

Ischemic colitis: Ischemic colitis is caused by sudden, often temporary, reduction in mesenteric blood flow secondary to hypoperfusion, vasospasm, or occlusion of the mesenteric vasculature.

A recent review of 313 patients with ischemic colitis reported involvement of the sigmoid colon in 20.8%, descending colon to sigmoid colon in 9.9%, transverse colon to sigmoid colon in 4.2%, and pancolonic involvement in 7.3% of patients^[59]. Mesenteric occlusion related to cardiac thromboembolism has been reported in up to one-third of patients with ischemic colitis^[60] whereas hypercoagulable states, vasculitis, and medications are less common risk factors^[61].

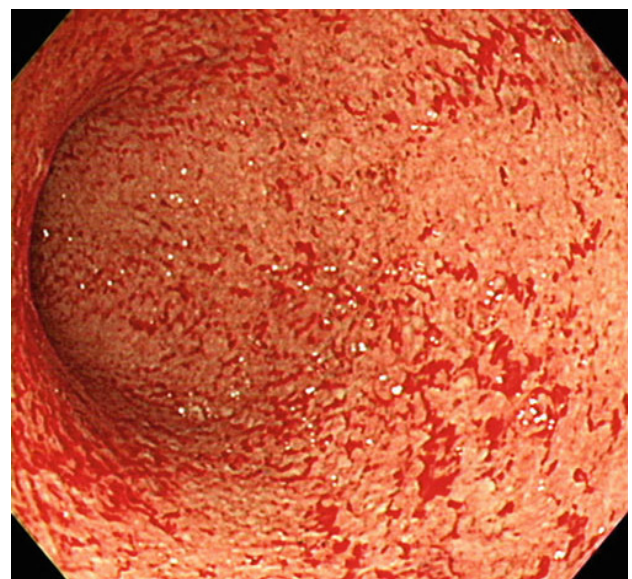


Figure 8 Ulcerative Colitis.

A Tringali. Endoscopic management of LGIB

The clinical presentation of ischemic colitis is characterized by the sudden onset of cramping abdominal pain, followed by hematochezia or bloody diarrhea within 24 hours^[62].

Typical endoscopic findings are submucosal hemorrhage and ulcerations in the colon with a characteristic segmental distribution (abrupt transition between abnormal and normal mucosa).

The rectum usually is spared, because of its dual blood supply^[63].

A single linear ulcer that runs along the longitudinal axis of the colon on the antimesenteric border (“single-strip” sign) also may indicate colon ischemia^[64]. None of these endoscopic findings are pathognomonic of ischemic colitis, however, and infectious and inflammatory colitis should remain in the differential diagnosis. Anyway endoscopy in this setting has just a diagnostic role.

IBD

Patients with inflammatory bowel disease commonly present with LGIB as shown in figure 8.

Although GI bleeding is a common manifestation of inflammatory bowel disease, acute severe hematochezia is infrequent. Acute severe hematochezia accounts for up to 6% of hospitalizations for patients with Crohn’s disease and 1.4% to 4.2% of patients with ulcerative colitis. Usually patients with acute Lower GI bleeding from IBD are older and had longer duration of IBD. Patients taking aspirin or non steroidal anti-inflammatory drugs may induce exacerbations of IBD and contribute to the morbidity of the patients taking them. The site of bleeding in Crohn’s disease is equally distributed between isolated small bowel, ileocolonic or colonic disease. The patients with Ulcerative colitis (UC) had pancolitis.

Bleeding resolves spontaneously in up to 50% of patients, but there is a recurrence rate of up to 35%^[65] and when it occurs, surgery is required for control of hemorrhage in about of 50% of cases.

The median time to recurrence hemorrhage has been reported to be 3 days, which might represent the minimum safe duration of hospitalization, although some patients may have recurrence more than 1 week after the initial episode.

Medical management includes supportive care with no change in baseline medications, the addition of corticosteroids and/or aminosalicilates. Biological therapy can be effective in the management of these patients, especially in Crohn’s disease^[66,67].

Because of the diffuse nature of the bleeding associated with these diseases, endoscopic treatment of bleeding is rarely appropriate and therefore these lesions are not amenable for endoscopic treatments.

NSAID colitis: NSAID-induced colopathy usually involves the right colon due to a higher concentration of the drug at this site, but the rectum may also be involved. NSAIDs possibly damage the normal large intestine, which presents as ulceration, colitis and stricture. Long-term drug use is not a requirement for the development of NSAID-induced colopathy. Short-term therapy can be just as significant and colitis can occur even after a few days of NSAID use^[68,69].

NSAIDs given in both the oral and parenteral forms have been found to cause colonic ulcerations^[70].

Colonic perforation, as an adverse effect of NSAIDs, has been reported in sporadic cases in the literature^[71,72]. Several studies found that NSAIDs can cause diverticular bleeding and perforation, flare-up of inflammatory bowel disease, and play a role as an etiologic factor in lymphocytic colitis. Inflammatory bowel diseases, malignancy and infectious colitis must be ruled out before establishing the diagnosis of NSAID-induced colopathy.

NSAIDs have numerous deleterious effects on the colon. NSAID-

induced colopathy is not a rare condition. Severe lower GI bleeding due to NSAID-induced colopathy is a rare diagnosis. Discontinuation of the offending drug is mandatory. There is no obvious evidence for the effectiveness of medical treatment in NSAID-induced colopathy. In some case reports, steroids have been used in the treatment of NSAID colonic stricture which failed to resolve after discontinuation of the offending drug^[73].

Infectious colitis: Massive lower gastrointestinal bleeding due to intestinal TB was once an uncommon complication of TB, but recent reports indicate an increased incidence especially in developing Countries.

In HIV patients, lower gastrointestinal bleeding may occur due to opportunistic infections, such as cytomegalovirus (CMV). Bleeding is reported to occur in 35-70% of CMV colitis^[74], but hemodynamically significant hemorrhage is rare. Taken together, the co-infection of CMV colitis and intestinal TB appears to contribute to the massive hemorrhage as reported in the case report^[75].

Surgical resection is usually not indicated for intestinal TB^[76], however, mortality is high when intestinal TB is complicated with bleeding^[77].

Previous reports indicate that surgery remains the definitive management for the treatment of patients with massive gastrointestinal bleeding due to intestinal TB^[78,79]. Indeed, timely resection dramatically improved the condition of patients. Therefore, early surgical intervention is recommended for lower gastrointestinal bleeding with intestinal TB.

Infective colitis can be a cause of massive lower gastrointestinal bleeding requiring acute surgical intervention. Causative organisms include Entamoeba and Histoplasma species. However, concurrent colonic infection with both these organisms is very rare.

Neoplasm: Colon adenocarcinoma may present with acute LGIB. Occasionally there is a focal bleeding from the tumor (Figure 4) that can be temporized with coagulation therapy but bleeding is generally slow and diffuse.

Recently hemospray has emerged as a new hemostatic technique for this gastrointestinal bleeding. The available data demonstrated the potential for hemostatic spray as a definitive or bridge therapy particularly in oozing lesion while in brisk arterial bleeding the efficacy is unknown^[80]. Several single case reports and small series have described the efficacy of Hemostatic spray in acute bleeding in various GI conditions (eg Mallory Weiss; Dieulafoy lesion, gastric antral vascular ectasia, post-polypectomy, radiation proctopathy and tumor bleeding^[81]).

Dieulafoy lesions: A bleeding Dieulafoy lesion (Figure 6) is caused by an exposed artery arising with a minute mucosal defect and, unless actively bleeding, may be difficult to detect. When seen the endoscopic treatment are identical at those of the upper GI tract such as thermal method, clipping and band ligation for eradication.

Rectal ulcers: Rectal ulcers have been reported in 8% of patients who present with severe hematochezia^[82] and in 32% of patients who develop LGIB after intensive care unit admissions for other critical illnesses^[83].

The terms acute hemorrhagic rectal ulcer (AHRU) was first used by Soenho *et al* and was established as disease entity in Japan by Hirooka *et al*. It has been described as a bleeding lesion that frequently occur in elderly patients who have severe underlying disorders and characterized by the presence of either irregular or

nearly round ulcer formation at lower part of the rectum presented as abrupt fresh rectal bleeding as initial symptom^[84].

Patients often have major medical comorbidities of end stage renal disease on hemodialysis, respiratory failure requiring mechanical ventilation, decompensated cirrhosis, critically ill^[83] or malignancy. Endoscopic findings range from clean-based ulcers (82%) to adherent clots (17%), non bleeding visible vessels (33%), and active bleeding (50%)^[82].

Early rebleeding after endoscopic treatment has been reported in 44% to 48% of patients, and a mortality rate of 33% to 48% has been reported in patients with high-risk stigmata who have multiple comorbidities^[82,83].

Post-operative LGIB: Lower GI bleeding can also occur in the postoperative course after colon resection^[85]. Generally is characterized by a limited hematochezia, but severe bleeding can occur in about 1% of patients and diagnostic and therapeutic manoeuvres can be challenging^[86].

Although there are some reports about the importance of comorbidities in the outcome of LGIB, there is no information about their influence on colorectal anastomosis bleeding.

The median time from primary surgery to LGIB was 6.5 h and varied from 30 min to 9 days after surgery, but more than 85% had this complications in the first day. It is extremely rare to occur as much as 10 days after surgery. In case of postoperative bleeding it is well known that nonoperative treatment is usually successful in almost all cases^[86].

The Cochrane group analyzed data regarding the colorectal anastomosis technique and its complication rate^[87].

The conclusion was that there is no scientific evidence to demonstrate any increased risk of hemorrhage of the stapled over hand-sewn technique. The endoscopy is the preferred approach in case of persistent/recurrent bleeding because allows the location of the bleeding and treatment. The presence of clot in the suture of the anastomosis may be responsible for the persistence of bleeding and therefore its removal could expose the vessel permitting the definitive treatment. The management of patients with ileocolic anastomosis is the most challenging situation.

The utility of endoscopy has been suggested and efficacy and safety of sclerotherapy, clips or electrocoagulation to stop bleeding

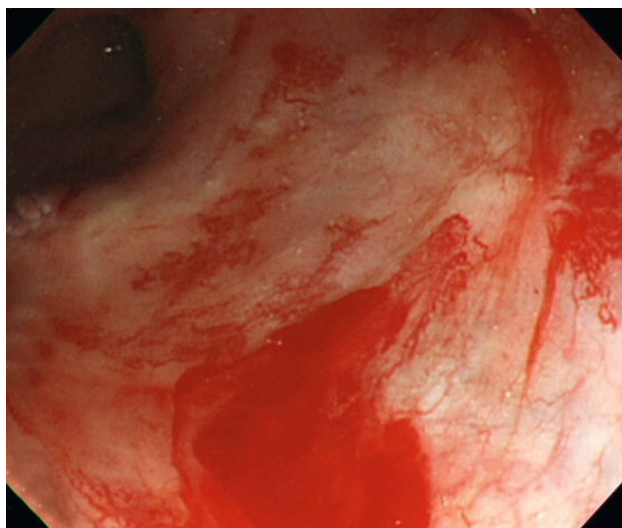


Figure 9 Radiation proctitis.

has been investigated^[85,86,88,89]. However the use of angiographic localization and control of bleeding could be a safe alternative. Surgery should be considered in patients with hemodynamic instability despite aggressive resuscitation and in case of endoscopic or angiographic failure.

Radiation proctopathy: Lower Gastrointestinal Bleeding has been reported in 4% to 13% of patients with radiation proctopathy.

A typical endoscopic appearance of radiation proctopathy is reported in figure 9.

Chronic radiation proctopathy (CRP) can occur from 9 months to 30 years after pelvic radiation injury, although patients typically present within 2 years after radiation^[90]. Can cause chronic GIB and is seen in patients who undergone radiation therapy to the pelvic organs in the treatment of gynaecological or urological malignancies. Up to 25% of patients receiving pelvic radiotherapy develop acute anorectal symptoms and up to 20% will develop persistent radiation proctopathy that is more common in case of radiotherapy for prostate cancer. Five years after radiotherapy about 25% of patients reported rectal bleeding. A variety of therapeutic option has been described including pharmacotherapy (oral and rectal 5-ASA products, steroids) topical formalin application, rectal instillation therapy (eg, hydrocortisone, sucralfate, 5-aminosalicylates, short-chain fatty acids, metronidazole) and hyperbaric oxygen therapy^[91]. Endoscopic treatments include argon plasma coagulation^[92]. APC has been proved efficacious in reducing rectal bleeding in 80-90% of cases. In most cases, 1-3 treatment sessions are required. Power settings of 25-60 W and argon plasma flow rates of 0.5-2.5 l min have been reported^[93].

Radiofrequency ablation using an endoscopically directed focal ablation device and endoscopic cryotherapy have also been described in small case series^[94].

A recent study published by Rustagi has shown that RFA therapy led to complete resolution of rectal bleeding in all treated patients with chronic radiation proctopathy, with improvement in clinical and endoscopic indices without any major adverse events. Anyway further controlled studies are needed to establish RFA as the endoscopic therapy of choice for treatment of Chronic radiation proctopathy^[95].

There are no large randomized, controlled studies of the management of chronic radiation proctopathy, and most of the data are based on case series and small trials^[96].

Dilute (eg, 2%-10%) formalin topical treatment of radiation proctopathy has been applied either through a rigid proctoscope or instilled into the rectum. Complete clinical responses range from 63% to 100% but high risk of complications, including anal stenosis, mucosal ulceration, and mild fecal incontinence have been reported^[97].

Relative contraindications for endoscopic therapy include evidence of malignant recurrence, stenosis, and fistulae.

All thermal treatment methods may create symptomatic ulceration (including pain and bleeding), which can require months to resolve. Laser therapy and dilute formalin (2-10%) application are associated with higher complications and are not commonly utilized.

Table 11 Goligher classification of Hemorrhoid.

Degree	
1	Hemorrhoid bleed but not prolapse
2	Internal haemorrhoid prolapse but spontaneously reduce
3	Hemorrhoids prolapse and require manual reduction
4	Hemorrhoid prolapse but cannot be reduced

Hemorrhoids: Hemorrhoid are classified as external or internal depending on their presence below or above the dentate line. The Goligher classification of internal haemorrhoid has 4 degree^[98] as shown in table 11.

Although haemorrhoids may be present in up to 75% of patients with LGIB, the majority are considered incidental findings. Hemorrhoidal bleeding has been reported to account for only 2% to 10% of acute LGIB^[2,9]. However two recent studies found that hemorrhoids were the underlying etiology from 24% to 64% of patients presenting with hematochezia^[99,100]. Patients typically presenting with painless, intermittent, hematochezia characterized by bright red blood on the toilet paper, coating the stool or dripping in the bidet bowl.

Endoscopic treatment include sclerotherapy^[101], rubber band ligation^[102], infrared photocoagulation, electrocoagulation^[103,104] and cryotherapy^[105]. Surgical treatments are the treatments of choice for all fourth grade of hemorrhoids, strangulated hemorrhoids and those that have not been successfully treated by other forms of therapy^[106]. RBL is the treatment of choice for first and second degree of hemorrhoid when medical treatment fails. Surgical treatment should be reserved for patients with very large third or fourth degree haemorrhoid or in patients in whom endoscopic treatment failed^[107].

Management in case of endoscopic failure or for massive bleeding: It is paramount keep in mind that in case of endoscopic failure or in case of massive and persistent bleeding angiographic approach should be made in order to localize the source of bleeding and to perform a definitive treatment.

Angiography: Localization and characterization of the bleeding source are important in determining the appropriate intervention, as treatment options range from minimally invasive catheter-directed therapy to extensive surgical resection. Although there has generally been a decline in the number of patients who present with acute gastrointestinal hemorrhage re-quiring angiography and/or transcatheter intervention, there are still patients who are unresponsive to either medical or endoscopic management and thus require emergency angiographic evaluation and possible transcatheter treatment. Angiography can identify and localize bleeding accurately but it requires a bleeding rate of at least 0.5 to 1.0 mL/min to be positive. When the bleeding is identified, it may be treated with intra-arterial infusion of vasopressin or superselective embolization with various agents (eg, coil micrombolization). Vasopressin infusion was the first modality used and it controlled bleeding in up to 90% of case^{s[108,109]}. Unfortunately the complications rate was 10% to 20% and included arrhythmias, pulmonary edema, hypertension and ischemia. Therefore it is no longer used. Selective embolization initially controls hemorrhage in up to 100% of patients, but rebleeding rates are 15% to 40%^[110,111]. Angiography can be used in the setting of active, persistent bleeding or in cases of non diagnostic endoscopic evaluation^[112]. Superselective catheterization using a coaxial system that allows for microcoil embolization is an effective

and safe alternative to emergency surgery. Other embolic agents such as gelatin sponge, spherical particles and liquids also have a role in transcatheter management of gastrointestinal bleeding as do transcatheter therapies such as covered stent placement^[113].

A meta-analysis of angiography in LGIB included 7 case series, each with more than 10 patients with major LGIB ad with attempt to embolization, the result showed that median rebleeding rate was 14%, 75% of rebleeding occurred within 3-5 days. Superselective mesenteric angiography remains at the cornerstone of management of patients with acute LGIB but it is invasive and time-consuming procedure. In addition due to low sensitivity for slower and intermittent bleeding most of radiologists suggest the use of a CT angiography to demonstrate a contrast extravasation and localize the source of bleeding in attempt to improve the diagnostic yield of mesenteric angiography. The recent studies analyzing the outcome after mesenteric angiography are shown in table 12.

Surgery: Several surgical option are available. These include emergency limited segmental resection for an identified bleeding source (“directed segmental resection”), emergent segmental resection for an unknown source (“blind segmental resection”), and emergency total colectomy for an unknown colonic bleeding source with or without ileo-rectal anastomosis. Emergent surgery should be considered only as a last resort and is rarely needed to prevent death from exanguination. However the morbidity and mortality associated with “blind” subtotal colectomy is higher than segmental resection of a preoperatively identified bleeding site. In one study, the rebleeding rate over 1 year follow up period was 14% after segmental colectomy directed by angiography, but 42% after blind segmental colectomy^[114].

Current evidence about the role of surgery in the management of LGIB are based on small cohort studies and case-control studies and any actual recommendations are opinion based rather than evidence based.

The golden standard for surgical treatment of acute severe LGIB should be directed segmental resection based on aggressive preoperative identification of the bleeding site.

CONCLUSION

LGIB represents a serious and increasingly important problem for patients and gastroenterologists because of lower GI complications had a higher mortality, longer hospitalization, and higher resource utilization than did upper GI complications. LGIB encompasses a wide clinical spectrum ranging from occult bleeding to overt hematochezia until massive hemorrhage with shock requiring emergency hospitalization. The use of risk model stratified patients in high, moderate and low risk group permitting to select the appropriate level of care and therapeutic approach.

Endoscopy is the diagnostic procedure of choice in most patients with Lower GI bleeding and the role of colonoscopy in the treatment of lower GI bleeding has been shown to be an efficacious and

Table 12 Outcomes after mesenteric angiography.

Author	No. of patients	No. of patient embolized	Hemostasis %	Rebleed (early) %	Surgery %	Ischemic complications needing surgery	Mortality %
Koh	68	68	100	9	15	1.5	0
Tan	265	32	97	22	28	3	9
Ahmed	20	20	80	27	30	0	25
Maleux	122	39	100	6	18	10	5
De Barros	27	27	100	22	5/27	7.2%	-

safe method even if a therapeutic endoscopy occurred in about 30% of patients underwent to colonoscopy. The optimal timing of colonoscopy in LGIB remains to be determined. Data suggest that colonoscopy performed early is more likely to identify and treat stigmata, but to demonstrate this would require a much larger trial. In case of massive and persistent bleeding without identification of source of bleeding it is reasonable a more aggressive approach with angiographic treatment.

Surgery is considered the last resort in the treatment of lower GI bleeding because of high morbidity and mortality in case of blind subcolectomy therefore every effort should be made to search the bleeding source.

CONFLICT OF INTERESTS

The authors declare that they have no conflict of interests.

REFERENCES

- Zuccaro GJ. Management of the Adult patients with Acute Lower Gastrointestinal bleeding. *Am J Gastroenterol* 1998; **93(8)**: 1202-1208
- Longstreth GF. Epidemiology and outcome of patients hospitalized with acute lower gastrointestinal hemorrhage: a population-based study. *Am J Gastroenterol* 1997; **92(3)**: 419-24
- Strate LL. Lower GI bleeding: epidemiology and diagnosis. *Gastroenterol Clin North Am* 2005;Dec; **34(4)**: 643-664
- Lanas A, Garcia-Rodriguez LA, Polo-Tomas M, Ponce M, Alonso-Abreu L, Perez-Aisa MA. Time trends and impact of upper and lower gastrointestinal bleeding and perforation in clinical practice. *Am J Gastroenterol* 2009; **104(7)**: 1633-1641
- Peura DA, Lanza FL, Gostout CJ, Foutch PG. The American College of Gastroenterology Bleeding Registry: preliminary findings. *Am. J. Gastroenterol* 1997; **92(6)**: 924-928
- Bounds BC, Kelsey PB. Lower gastrointestinal bleeding. *Gastrointest. Endosc. Clin N Am* 2007; **17(2)**: 273-288
- Imdahal A. Genesis and pathophysiology of lower gastrointestinal bleeding. *Langenbecks Arch Surg* 2001; **386**: 1-7
- Zuckerman GR, Prakash C. Acute lower intestinal bleeding Part II: Etiology, therapy, and outcomes. *Gastrointest Endosc* 1999; **49(2)**: 228-238
- Strate LL, Orav J, Syngal S. Early predictors of severity in acute lower intestinal tract bleeding. *Arch Intern med* 2003; **163(7)**: 838-843
- Velayos FS, Williamson A, Sousa KH, Lung E, Bostrom A, Weber EJ, Ostroff JW, Terdiman JP. Early predictors of severe lower gastrointestinal bleeding and adverse outcome: A prospective study. *Clin Gastroenterol Hepatol* 2004; **2(6)**: 485-490
- Strate LL, Saltzman JR, Ookubo R, Mutinga ML, Syngal S. Validation of a clinical prediction rule for severe acute lower intestinal bleeding. *Am J Gastroenterol* 2005; **100(8)**: 1821-1827
- Strate LL, Ayanian JZ, Kotler G, Syngal S. Risk factors for mortality in lower intestinal bleeding. *Clin Gastroenterol Hepatol* 2008 Sep; **6(9)**: 1004-1010
- Kollef MH, O'Brien JD, Zuckerman Gr, Shannon W. BLEED: a classification tool to predict outcomes in patients with acute upper and lower GI haemorrhage. *Crit Care Med* 1997; **25(7)**: 1125-1132
- Das A, Ben-Menachem T, Cooper GS, Chark A, Sivak MV, Gonet JA, Wong RC. Prediction of outcome in acute lower GI hemorrhage based on an artificial neural network: internal and external validation of a predictive model. *Lancet* 2003; **362(9392)**: 1261-1266
- Green BT, Rockey DC, Portwood G, Tarnasky PR, Guarisco S, Branch MS, Leung J, Jowell P. Urgent colonoscopy for evaluation and management of acute lower gastrointestinal hemorrhage: a randomized controlled trial. *Am J Gastroenterol* 2005; **100(11)**: 2395-2402
- Jensen DM; Machicado GA. Diagnosis and treatment of severe hematochezia: the role of urgent colonoscopy after purge. *Gastroenterology* 1988; **95(6)**: 1569-1574; 13
- Jensen DM; Machicado GA, Jutabha R, Kovacs TO. Urgent colonoscopy for the diagnosis and treatment of severe diverticular bleeding. *N Eng. J Med* 2000; **342(2)**: 78-82
- Schmulewitz N, Fischer 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; **58(6)**: 841-846
- Kok K, Kum CK, Goh PM. Colonoscopic evaluation of severe hematochezia in an Oriental population. *Endoscopy* 1998; **30(8)**: 675-680
- Chaudhry AM, Hyser MJ, Gracias VH, Gau FC. Colonoscopy: initial test for acute lower gastrointestinal bleeding. *Am Surg* 1998; **64(8)**: 723-728
- Ohyama T Sakurai Y, Ito M, Daito K, Sezai S, Sato Y. Analysis of urgent colonoscopy for lower gastrointestinal tract bleeding. *Digestion* 2000; **61(3)**: 189-92
- Angtuaco TL, Reddy SK, Drapkin S, Harrell LE, Howden CW. The utility of urgent colonoscopy in the evaluation of acute lower gastrointestinal tract bleeding: a 2-year experience of a single center. *Am J Gastroenterol* 2001; **96**: 1782-1785
- Green BT, Rockey DC. Lower Gastrointestinal bleeding-management. *Gastrointest Clin North Am* 2005; **34(4)**: 665-678
- Laine L, Shah A. Randomized trial of urgent vs elective colonoscopy in patients hospitalized with lower GI bleeding. *Am J Gastroenterol* 2010; **105(12)**: 2636-2641
- Jensen DM. What to choose for diagnosis of bleeding colonic angiomias: colonoscopy, angiography or helical computed tomography angiography? *Gastroenterology* 2000; **119(2)**: 581-584
- Ohyama T, Sakurai Y, Ito M, Daito K, Sezai S, Sato Y. Analysis of urgent colonoscopy for lower gastrointestinal tract bleeding. *Digestion* 2000; **61(3)**: 189-192
- Al Qahtani, Satin R, Stern J, Gordon PH. Investigative modalities for massive lower Gastrointestinal bleeding. *World J Surg* 2002 May; **26(5)**: 620-625
- Davila RE, Rajan E, Adler DJ, Egan J, Hirota WK, Leighton JA, Qureshi W, Zuckerman MJ, Fanelli R, Wheeler-Harbaugh J, Baron TH, Faigel DO. ASGE Guideline. The role of endoscopy in the patients with lower GI bleeding. *Gastrointest Endosc* 2005; **62(5)**: 656-660
- Frossard JL, Gervaz P, Huber O. Water immersion sigmoidoscopy to treat acute GI bleeding in the perioperative period after surgical colorectal anastomosis. *Gastrointest Endosc* 2010; **71(1)**: 167-170
- NG DA, Opelka FG, Beck DE, Milburn JM, Whitterspoon LR, Hicks TH, Timmecke AE, Gathright JB Jr. Predictive value of technetium TC 99m-labeled red blood cell scintigraphy for positive angiogram in massive lower gastrointestinal haemorrhage. *Dis colon Rectum* 1997; **40(4)**: 471-477
- Scheffel H, Pfammatter T, Wildi S, Bauerfeind P, Marincek B, Alkadhi H. Acute gastrointestinal bleeding: detection of source and etiology with multi-detector-row CT. *Eur Radiol* 2007; **17(6)**: 1555-1565
- Ernst O, Bulois P, Saint-Drenant S, Leroy C, Paris JC, Sergeant G. Helical CT in acute lower gastrointestinal bleeding. *Eur Radiol* 2003; **13(1)**: 114-117
- Milagros Martí, Artigas JM, Garzón G, Alvarez-Sala R, Soto JA. Acute Lower Intestinal Bleeding: Feasibility and Diag-

- nostic Performance of CT Angiography. *Radiology* 2012; **262**(1): 109-116
- 34 Holster L, Brullet E, Kuipers E, Campo R, Fernández-Atutxa A, Tjwa ET. Hemospray treatment is effective in lower GI bleeding. *Endoscopy* 2014; **46**(1): 75-78
- 35 Schuetz A, Jauch KW. Lower Gastrointestinal bleeding: therapeutic strategies, surgical techniques and results. *Langebechs Arch Surg* 2001; **386**(1): 17-25
- 36 Wong KS, Baron TH. Endoscopic management of lower gastrointestinal bleeding. *Am J Gastroenterology* 2008; **103**(8): 1881-1887
- 37 Jensen DM, Machicado GA. Colonoscopy for diagnosis and treatment of severe lower GI bleeding. Routine outcomes and cost analysis. *Gastrointest Endosc Clin N Am* 1997; **7**(3): 477-498
- 38 McGuire HH. Bleeding colon diverticula. A reappraisal of natural history and management. *Ann Surg* 1994; **220**(5): 653-656
- 39 Prakash C, Chokshi H, Walden DT, Aliperti G. Endoscopic hemostasis of in acute diverticular bleeding. *Endoscopy* 1999; **31**(6): 460-463
- 40 Foutch PG, Zimmermann K. Diverticular bleeding protuberance (sentinel clot); clinical implication, histopathological correlation, and result of endoscopic interventions. *Am J Gastroenterol* 1996; **91**(12): 2589-2593
- 41 Bloomfield RS, Rockey DC, Shetzline MA. Endoscopy therapy of acute diverticular haemorrhage. *Am J Gastroenterol* 2001; **96**(8): 2367-2372
- 42 Ramirez FC Johnson DA, Zierer ST, Walker GJ, Sanowski RA. Successful endoscopic hemostasis of bleeding colonic diverticula with epinephrine injection. *Gastrointest Endosc* 1996; **43**(2): 167-170
- 43 Simpson PW, Nguyen NH, Lim JK, Soetikno RM. Use of endoclip in the treatment of massive colonic diverticula bleeding. *Gastrointest. Endosc* 2004; **59**(3): 433-437
- 44 Farrell JJ, Graeme-Cook F, Kelsey PB. Treatment of bleeding colonic diverticula by endoscopic band ligation: an in-vivo and ex-vivo pilot study. *Endoscopy* 2003; **35**(10): 823-829
- 45 Witte JT. Band ligation for colonic bleeding: modification of multiband ligating device for use with a colonoscope. *Gastrointest Endosc* 2000; **52**(8): 762-765
- 46 Setoyama T, Ishii N, Fujita Y. Endoscopic band ligation (EBL) is superior to endoscopic clipping for the treatment of colonic diverticular bleeding. *Surg Endosc* 2011; **25**(11): 3574-3578
- 47 Boley S, Sammartano R, Adams A, DiBiase A, Kleinhaus S, Sprayregen S. On the nature of vascular ectasia of the colon. Degenerative lesions of aging. *Gastroenterology* 1977; **72**(5): 650-660
- 48 Moore JD, Thompson NW, Appelman HD, Foley D. Arteriovenous malformation of the gastrointestinal tract. *Arch Surg* 1976; **111**(4): 381
- 49 Helmrich GA, Stalworth JR, Brown JJ. Angiodysplasia: characterization, diagnosis and advances in treatment *South Med. J* 1990; **83**(12): 1450-1453
- 50 Farrell JJ Friedman LS. Gastrointestinal bleeding in the elderly. *Gastroenterol Clin North. Am* 2001; **30**(2): 377-407
- 51 Brandt LJ, Spinell MK. Ability of naloxone to enhance the colonoscopy appearance of normal colon vasculature and colon vascular ectasia. *Gastrointest Endosc* 1999; **49**(1): 79-83
- 52 Sawhney MS, Salfiti N, Nelson DB, Lederle FA, Bond JH. Risk factors for severe delayed postpolypectomy bleeding. *Endoscopy* 2008; **40**(2): 115-119
- 53 Watabe H, Yamaji Y, Okamoto M, Kondo S, Ohta M, Ikenoue T, Kato J, Togo G, Matsumura M, Yoshida H, Kawabe T, Omata M. Risk assessment for delayed hemorrhagic complication of colonic polypectomy: polyp-related factors and patient-related factors. *Gastrointest Endosc* 2006; **64**(1): 73-78
- 54 Shioji K, Suzuki Y, Kobayashi M, Nakamura A, Azumya M, Tacheuchi M, Baba Y, Honma T, Narisawa R. Prophylactic clip application does not decrease delayed bleeding after colonoscopic polypectomy. *Gastrointest Endosc* 2003; **57**(6): 691-694
- 55 Rey JF, Marek TA. Endoloop in the prevention of the post-polypectomy bleeding: preliminary results. *Gastrointest Endosc* 1997; **46**(4): 387-389
- 56 Katsinelos P, Kountouras J, Paroutoglu G, Beltsis A, Chatzimavroudis G, Zavos C, Vasiliadis I, Katsinelos T, Papaziogas B. Endoloop-assisted polypectomy for large pedunculated colorectal polyps. *Surg Endosc* 2006; **20**(8): 1257-1261
- 57 Iishi H, Tatsuta M, Narahara H, Iseki K, Sakai N. Endoscopic resection of large pedunculated colorectal polyps using a detachable snare. *Gastrointest Endosc* 1996; **44**(5): 594-597
- 58 Matsushita M, Hajiro K, Takakuwa H, Kusumi F, Maruo T, Ohana M, Tominaga M, Okano A, Yunoki Y. Ineffective use of a detachable snare for colonoscopic polypectomy of large polyps. *Gastrointest Endosc* 1998; **47**(6): 496-499
- 59 Brandt LJ, Feuerstadt P, Blaszcza MC. Anatomic patterns, patient characteristics, and clinical outcomes in ischemic colitis: a study of 313 cases supported by histology. *Am J Gastroenterol* 2010; **105**(10): 2245-2252
- 60 Hourmand-Ollivier I, Bouin M, Saloux E, Morello R, Rouselot P, Piquet MA, Dao T, Verwaerde JC. Cardiac sources of embolism should be routinely screened in ischemic colitis. *Am J Gastroenterol* 2003; **98**(7): 1573-1577.4
- 61 Greenwald DA, Brandt LJ, Reinus JF. Ischemic bowel disease in the elderly. *Gastroenterol Clin North Am* 2001; **30**(2): 445-473
- 62 Chait MM. Lower gastrointestinal bleeding in the elderly. *World J Gastrointest Endosc* 2010; **16**; **2**(5): 147-154
- 63 Brandt LJ, Feuerstadt P, Blaszcza MC. Anatomic patterns, patient characteristics, and clinical outcomes in ischemic colitis: a study of 313 cases supported by histology. *Am J Gastroenterol* 2010; **105**: 2245-2245
- 64 Zuckerman GR, Prakash C, Merriman RB, Sawhney MS, DeSchryver-Keckemeti K, Clouse RE. The colon single-stripe sign and its relationship to ischemic colitis. *Am J Gastroenterol* 2003; **98**(9): 2018-2022
- 65 Robert JR, Sachar DB, Greenstein AJ. Severe gastrointestinal hemorrhage in Crohn's disease. *Ann Surg* 1991; **213**(3): 207-211
- 66 Tsujikawa T, Nezu R, Andoh A, Saotome T, Araki Y, Ishizuka Y, Sasaki M, Koyama S, Fujiyama Y. Infliximab as a possible treatment for the hemorrhagic type of Crohn's disease. *J Gastroenterol* 2004; **39**(3): 284-287
- 67 Papi C, Gili L, Tarquini M, Antonelli G, Capurso L. Infliximab for severe recurrent Crohn's disease presenting with massive gastrointestinal hemorrhage. *J Clin Gastroenterol* 2003; **36**(3): 238-241
- 68 Hebuterne X, Dreyfus G, Fratini G, Rampal P. Non-steroidal anti-inflammatory drug-induced colitis and misoprostol. *Dig Dis Sci* 1996; **41**(3): 520-521
- 69 Crosby JC, Ross GJ, Auchenbach RC, McCafferty MH. Non-steroidal anti-inflammatory drug-induced cecal ulceration diagnosed on barium enema. *J Clin Gastroenterol* 1998; **26**(2): 154-156
- 70 Halter F, Weber B, Huber T, Eigenmann F, Frey MP, Ruchti C. Diaphragm disease of the ascending colon. Association with sustained-release diclofenac. *J Clin Gastroenterol* 1993; **16**(1): 74-80
- 71 Aloysius MM, Kaye PV, Lobo DN. Non-steroidal anti-inflammatory drug (NSAID)-induced colonic strictures and perforation: a case report. *Dig Liver Dis* 2006; **38**(4): 276-8

- 72 Robinson MH, Wheatley T, Leach IH. Non-steroidal anti-inflammatory drug-induced colonic stricture. An unusual cause of large bowel obstruction and perforation. *Dig Dis Sci* 1995; **40**(2): 315-9
- 73 Penner RM, Williams CN. Resolution of multiple nonsteroidal anti-inflammatory drug-induced colonic strictures with prednisone therapy: a case report and review of the literature. *Can J Gastroenterol* 2003; **17**(8): 497-500
- 74 Chalasani N, Wilcox CM. Etiology and outcome of lower gastrointestinal bleeding in patients with AIDS. *American Journal of Gastroenterology* 1998; **93**(2): 175-178. [PubMed: 9468236]
- 75 Nagahashi M, Aoyagi T, Yamada A, Rashid OM, Adams BJ, Takabe K. Intestinal Co-infection of Tuberculosis and CMV can Cause Massive Lower GI Bleeding in a Patient with HIV. *J Surg Sci* 2013; **1**(1): 12-15
- 76 Vimla NS, Khanna SK, Broor SL, Sharma BK, Bhusnurmath SR. Massive melaena: a rare indication for surgery in intestinal tuberculosis. *Tubercle* 1982; **63**(2): 133-135
- 77 Goudarzi HA, Mason LB. Fatal rectal bleeding due to tuberculosis of the cecum. *Journal of the American Medical Association*. 1982; **247**(5): 667-668
- 78 Goudarzi HA, Mason LB. Fatal rectal bleeding due to tuberculosis of the cecum. *Journal of the American Medical Association* 1982; **247**(5): 667-668
- 79 Watanabe T, Kudo M, Kayaba M, Shirane H, Tomita S, Ori-no A, Todo A, Chiba T. Massive rectal bleeding due to ileal tuberculosis. *J Gastroenterology* 1999; **34**(4): 525-529
- 80 ASGE guideline Emerging technology for endoscopic hemostasis. *Gastrointest Endosc* 2012; **75**(5): 933-937
- 81 Beyazit Y, Kekilli M, Kurt M, Sayilir A, Haznedaroglu IC. Ankaferd hemostat for the management of tumoral GI bleeding. *Gastrointest Endosc* 2011; **73**(5): 1072-1073
- 82 Kanwal F, Dulai G, Jensen DM, Gralnek IM, Kovacs TO, Machicado GA, Jutabha R. Major stigmata of recent hemorrhage on rectal ulcers in patients with severe hematochezia: endoscopic diagnosis, treatment, and outcomes. *Gastrointest Endosc* 2003; **57**(4): 462-468
- 83 Lin CK, Liang CC, Chang Ht, Hung FM, Lee TH. Acute hemorrhagic rectal ulcer: an important cause of lower gastrointestinal bleeding in the critically ill patients. *Dig Dis Sci* 2011; **56**(12): 3631-3637
- 84 Oku T, Maeda M, Ihara H, Umeda I, Kitaoka K, Waga E, Wada Y, Katsuki S, Nagamachi Y, Niitsu Y. Clinical and endoscopic features of acute hemorrhagic rectal ulcer. *J Gastroenterol* 2006; **41**(10): 962-970
- 85 Atabek U, Pello MJ, Spence RK, Atabek U, Pello MJ, Spence RK. Arterial vasopressin for control of bleeding from a stapled intestinal anastomosis. Report of two cases. *Dis Colon Rectum* 1992; **35**(12): 1180-1182
- 86 Cirocco WC, Golub RW. Endoscopic treatment of postoperative haemorrhage from a stapled colorectal anastomosis. *Am Surg* 1995; **61**(5): 460-463
- 87 Lustosa SA, Matos D; Atalah AN, Castro AA. Stapled versus handsewn methods for colorectal anastomosis surgery. *Cochrane Database of Systematic reviews* 2001; **3**: D 003144
- 88 Farrell JJ, Friedman LS. The management of lower gastrointestinal bleeding. *Aliment Pharmacol Ther* 2005; **21**(11): 1281-1288
- 89 Rios A, Mariano MJ, Rodriguez MJ, Serrano A, Molina J, Ramirez P, Parrilla P. Acute lower gastrointestinal haemorrhages in geriatric patients. *Dig Dis Sci* 2005; **50**(5): 898-904
- 90 Karamanolis G, Triantafyllou K, Tsiomoulos Z, Polymeros D, Kalli T, Misailidis N, Ladas SD. Argon plasma coagulation has a long-lasting therapeutic effect in patients with chronic radiation proctitis. *Endoscopy* 2009; **41**86: 529-531
- 91 Leiper K, Morris AL. Treatment of radiation proctitis. *Clin Oncol* 2007; **19**(9): 724-729
- 92 Postagata A, Saunders B, Tjandra J, Vargo J. Argon plasma coagulation in chronic radiation proctitis. *Endoscopy* 2007; **39**(4): 361-365
- 93 Sato Y, Takayama, T, Sagawa, T, Hirakawa M, Ohnuma H, Miyanishi K, Sato T, Takimoto R, Kobune M, Okamoto K, Takeuchi H, Kato J. Argon Plasma Coagulation Treatment of Hemorrhagic Radiation Proctopathy: The Optimal Settings for Application Argon Plasma Coagulation Treatment of Hemorrhagic Radiation Proctopathy: The Optimal Settings for Application and Long-Term Outcome. *Gastrointest Endosc* 2011; **73**(3): 543-549
- 94 Zhou C, Adler DC, Becker L, Chen Y, Tsai TH, Figueiredo M, Schmitt JM, Fujimoto JG, Mashimo H. Effective treatment of chronic radiation proctitis using radiofrequency ablation. *Ther Adv Gastroenterol* 2009; **2**(3): 149-56.41,42
- 95 Rustagi T, Corbett S, Mashimo H. Treatment of chronic radiation proctopathy with radiofrequency ablation. *Gastrointest Endosc* 2014; **81**(2): 428-436
- 96 Denton A, Forbes A, Andreyev J, Maher EJ. Nonsurgical interventions for late radiation proctitis in patients who have received radical radiotherapy to the pelvis. *Cochrane Database Syst Rev* 2002; **1**: CD003455
- 97 Saclarides TJ, King DG, Franklin JL, Doolas A. Formalin instillation for refractory radiation-induced hemorrhagic proctitis. Report of 16 patients. *Dis Colon Rectum* 1996; **39**(2): 196-199
- 98 Goligher JC. Hemorrhoids or piles. In: Goligher JC editor. *Surgery of the anus, rectum and colon*. 4th ed. London. *Bailliere Tindal* 1980 p 96
- 99 Gayer C, Chino A, Lucas C, Tokioka S, Yamasaki T, Edelman DA, Sugawa C. Acute lower gastrointestinal bleeding in 1,112 patients admitted to an urban emergency medical center. *Surgery* 2009; **146**(4): 600-606
- 100 Gralnek IM, Ron-Tal Fisher O, Holub JL, Eisen GM. The role of colonoscopy in evaluating hematochezia: a population based-study in a large consortium of endoscopy practices. *Gastrointest Endosc* 2013; **77**(3): 410-418
- 101 Graham Stuart CW. Injection treatment of haemorrhoids. *Br J Surg* 1962; **1**: 213-215
- 102 Steinberg DM, Liegois H, Alexander-Williams J. Long-term review of the results of rubber band ligation of haemorrhoids. *Br J Surg* 1975; **62**(2): 144-146
- 103 Neiger A. Hemorrhoid in every day practice. *Proctology* 1979; **2**: 22-28
- 104 Zingberg SS, Stern DH, Furman DS, Wittles JM. A personal experience in comparing three non-operative techniques for treating internal haemorrhoid. *Am J Gastroenterol* 1989; **84**(5): 488-492
- 105 Tanaka S. Cryosurgical treatment of haemorrhoids in Japan. *Int Surg* 1989; **74**(3): 146-147
- 106 ASGE guideline. The role of endoscopy in patients with anorectal disorders. *Gastrointest Endosc* 2010; **72**(6): 117
- 107 Shanmugam V, Thaha MA, Rabidranath KS, Campbell KL, Steele RJ, Loudon MA. Rubber band ligation versus excisional haemorrhoidectomy for haemorrhoids. *Cochrane Database Syst. Review* 2005; **3**: CD005034
- 108 Browder W, Cerise EJ, Litwin MS. Impact of emergency angiography in massive lower gastrointestinal bleeding. *Ann Surg* 1986; **204**(5): 530-536
- 109 Clark RA, Colley DP, Eggers FN. Acute arterial gastrointestinal hemorrhage: efficacy of transcatheter control. *Am J Roentgenol* 1981; **136**(6): 1185-1189
- 110 Ledermann HP, Schoch E, Jost R, Decurtins M, Zollkofer CL. Superselective coil embolization in acute gastrointestinal hemorrhage: personal experience in 10 patients and review of the literature. *J Vasc Interv Radiol* 1998; **9**: 753-760

Tringali A. A Tringali Endoscopic management of LGIB

- 111 Peck DJ, Mcloughing RF, Hugson RM, Rankin RN. Percutaneous embolotherapy of lower gastrointestinal haemorrhage. *J Vasc Interv Radiol* 1998; **9(5)**: 753-760
- 112 ASGE Guideline. The role of endoscopy in the patient with lower GI bleeding. *Gastrointest Endosc* 2005; **62 (5)**: 656
- 113 Ahmed Mahmoud El-Tawil. Angiographic evaluation and management of acute gastrointestinal hemorrhage. *World J Gastroenterol* 2012 March 21; **18(11)**: 1191-1201
- 114 Parkes BM, Obeid FN, Sorensen VJ, Horst HM, Fath JJ. The management of massive lower gastrointestinal bleeding. *Am Surg* 1993; **59(10)**: 676-678

Peer reviewer: Navneet Kumar AHLUWALIA, Section of Gastroenterology, Department of Medicine, Stepping Hill Hospital, Stockport NHS Foundation Trust, Poplar Grove, Stockport, Cheshire, SK2 7JE, United Kingdom.