

Outcome of Different Diagnostic and Therapeutic Modalities of Acute Lower Gastrointestinal Bleeding; a University Hospital Experience

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Abstract: Background: Acute lower GI bleeding (LGIB) is a common clinical situation that needs work up for proper diagnosis and individualized management. **Aim of the work:** Enlisting causes of acute LGIB in our locality, detecting the relation between causes and severity of the bleeding and compare different available therapeutic options for each case. **Patients and Methods:** One hundred patients with acute LGIB were included in this study. Patients attended our hospital during period from *September 2011 to February 2012* were classified according to the risk assessment score into two groups; group I (high risk group) included 39 patients and group II (moderate risk group) included 61 patients. Initial resuscitation and routine laboratory studies were done for all patients, and then procedures for localization of the bleeding site were done utilizing colonoscopy, enteroscopy, mesenteric angiography and radionuclide scintigraphy that were individualized for every patient. Medical, endoscopic, angiographic or surgical treatments were initiated according to the bleeding type, site and availability of the treatment modality. **Results:** Our study included 100 patients, 63 males and 37 females. Group I included 39 patients (24 males and 15 females) with age range (24-78 y) and mean \pm SD (56.2 \pm 14.1 y) and group II included 61 patients (39 males and 22 females) with age range (30-64 years) and mean \pm SD (44 \pm 15.3 y). Majority of patients (84.7%) in group I were presented with bright red hematochezia (84.6%), while, majority of patients (70.5%) in group II were presented with a maroon stool. Abdominal tenderness was the most frequent associated symptoms in both groups (74.4 % in group I and 36 % in group II). Requirement of blood units in group I was twice as that of group II. Significant differences were found between the two groups regarding hemoglobin level, hematocrit value, PT, and INR concentration. Rectosigmoid junction and descending colon followed by anorectal area and sigmoid colon (20.5 % vs. 37.7 % , 15.4 % vs. 14.7 % and 12.8 % vs. 11.5% respectively) were the commonest sites of the bleeding in both groups, moreover, bleeding from ascending colon was found to be more severe than other sites followed by bleeding from small intestine and descending colon. Diverticular disease, angiodysplasia and portal hypertensive colopathy (28.2 % , 12.9 % and 12.9 % respectively) were the main causes of acute LGIB among patients in group I, while, colitis, malignant neoplasm and benign polyps (14.7 % , 13.3 % and 11.5 % respectively) were the main causes of acute LGIB in group II. Colonoscopy was the best and beneficial diagnostic modality in our study; it was able to detect causes of the bleeding in 95% in group I and 91 % of causes in group II. Conservative medical management was successful in stopping bleeding in 18 patients (46.2%) in group I and in 28 patients (49.9%) in group II, while, endoscopic therapy was effective in stopping the bleeding in 15 patients (38.5%) in group I and in 27 patients (44.3%) in group II. Complete recovery was achieved in 71.8% of cases in group I and in 86% in group II. **Conclusion:** Acute LGIB is a common medical and surgical challenge. Diverticular disease is by far the most common cause of acute LGIB in our study. Medical conservation and endoscopic treatment are successful tools in stopping most cases of acute LGIB.

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1. Introduction

Lower gastrointestinal bleeding (LGIB) is a broad topic, which can include passage of a small amount of red blood on tissue paper associated with formed brown stool to life-threatening severe hemorrhage.⁽¹⁾

LGIB is one-fifth to one-third as common as upper gastrointestinal bleeding (UGIB) and generally has a less severe course. The annual incidence rate of LGIB in United States ranges from 20.5 to 27 cases per 100,000 adults populations, while, the annual incidence rate for UGIB is reported to range from 100 to 200 cases per the same adult population.⁽²⁾ LGIB accounts for approximately 30% of all GI bleeding and it is the reason for hospitalization in up to 0.02 % of hospitalized patients each year and carries a mortality rate of 3.6 %.⁽³⁾

Bleeding from lower GI tract may be acute or chronic, acute LGIB is rather arbitrarily defined as a bleeding situation in which blood loss has been occurring for less than 3 days causing hemodynamic instability, anemia, and/or need for blood transfusion. The source of acute LGIB is not always apparent from initial history and physical examination. Hematochezia is the most common clinical presentation of acute LGIB that necessitating hospitalization and immediate evaluation for proper diagnosis and management.⁽⁴⁾

Causes of acute LGIB include diverticular disease, vascular ectasia, ischemic, inflammatory or infectious colitis, colonic neoplasia (including post-polypectomy bleeding), anorectal diseases (including haemorrhoids, anal fissures and rectal varices) and small bowel lesions (Crohn's, vascular ectasia, Meckel's diverticula, and small bowel tumours).⁽⁵⁾

Acute LGIB is less frequent and less dramatic than UGIB, in most cases, bleeding from colon and rectum is self-limiting and usually requires no specific therapy on contrary to UGIB. ⁽⁶⁾ The incidence of LGIB increases with age, with a more than 200-fold increase from age of 20 to age 80 years. This rise in incidence with age is most likely explained by age-related increase of prevalence of colonic diverticulosis and angiodysplasia. ⁽⁶⁾ Moreover, mortality rate among hospitalized patients with acute LGIB is about 2.4%, but if bleeding occurs during hospital stay in patients hospitalized for causes other than LGIB, the rate increases dramatically to 23.1%. ⁽⁷⁾

The aims of this study were to enlist the causes of acute LGIB in our hospital, to detect the relationship between causes and severity of the bleeding, and lastly to evaluate the outcome of different available therapeutic options.

2. Patients and Methods

Study design and setting:

This study was carried out in our GI unit, internal medicine department, in collaboration with of interventional radiology and general surgery departments, Zagazig university hospitals, Egypt.

Target population and sampling:

All patients with acute LGIB who were admitted to our GI unit during the study period (*from September 2011 to February 2012*) fulfilling inclusion and exclusion criteria. Sampling included cases, which resolve with conservative medical measures as well as those who need radiological, endoscopic or surgical intervention to control the bleeding.

Inclusion criteria:

- Bloody bowel motions within the previous three days.
- More than three bloody bowel motions within less than 8 hours.
- Clinical or laboratory evidence of significant blood loss manifested by any the followings:
 - Decrease of more than 5% hematocrit value in the first 12 hours of hospital admission.
 - Transfusion of more than 3 units of packed RBC within 24 hours.
 - Hemodynamic instability in the previous 6 hours manifested either anginal pain, syncopal attack, orthostatic changes, mean arterial blood pressure <80 mmHg, or resting pulse >110.

Exclusion criteria:

- LGIB case, which was proven and documented by any diagnostic modality to be originated from a lesion above the ligament of Treitz.
- Patients with minimal clinically non-significant bleeding.
- Abdominal surgery within the previous 10 days.

- Known or suspected small or large bowel's ischemia, perforation or peritonitis.
- Patients with acute LGIB as a result of acute infectious diarrhea.
- Documented pregnancy.

Patient's classification:

After fulfillment of inclusion and exclusion criteria, one hundred adult patients were included in this study. Risk score assessment was used to classify patients and to assess patients presented with acute LGIB (*Strate and Saltzman, 2005*). ⁽⁹⁾

- Heart rate >100 BPM.
- Systolic blood pressure <115mmHg.
- Syncope.
- Non -tender abdominal examination.
- Rectal bleeding within the first 4 hours of the clinical evaluation.
- Aspirin use one week prior to evaluation.
- More than two active comorbid conditions.

Using this scoring system, patients were classified into two groups; **group I** (High risk group) that included 39 patients who have acute LGIB with more than 3 risk factors and **group II** (Moderate risk group), which included 61 patients who have acute LGIB with 1-3 risk factors.

Methods and study tools:

All included patients were subjected to the followings work up:

- 1) Initial standard resuscitation and management measures needed for patients stabilization. The number of packed RBC units needed to stabilize patient's hemodynamic status was listed and counted.
- 2) History taking with special stress on:
 - Bleeding per rectum (duration, color of the blood, relation to stool and frequency of bleeding).
 - Associated GI symptoms (abdominal pain, vomiting, hematemesis and disturbed bowel habits).
 - History of prior episodes of GI bleeding.
 - Comorbid diseases (DM, hypertension, IHD, COPD, CRF, cancer, coagulopathy and chronic liver disease).
 - Current/recent medications (NSAIDs, aspirin, antiplatelets and anticoagulant).
 - History of previous abdominal surgery or irradiation.
- 3) Physical examination including general examination, vital data recording, careful abdominal examination, perineal, perianal, and digital rectal examinations.
- 4) Nasogastric tube lavage to exclude possible upper GI bleeding.

5) Routine laboratory investigations that included complete blood picture, ESR, blood grouping and cross match, stool analysis, serum bilirubin, serum albumin, ALT and AST, serum creatinine and BUN and PT/PTT and INR.

6) Real time pelviabdominal U/S.

7) Diagnostic procedures for localization of the bleeding site:

A) *Endoscopic Assessment:*

1) **Colonoscopy:**

- **Timing:** Elective colonoscopy was done within 96 hour of hospital admission in unstable patients while hemodynamic stable patients underwent colonoscopy after rapid colonic preparation within 48 hour.
- **Bowel preparation for colonoscopy:** Bowel cleansing began as fluid resuscitation was carried out. Colonic preparation was done using 500 cc of Mannitol 20 % taken orally one day before the procedure and a cleaning enema at the procedure's day.
- **Equipments for colonoscopy:** Colonoscopy series used was (*GF- Q160AL- Olympous, Japan*). Diathermy equipment (*Berchtold-Electron 610*) included heater probes, electrocautery device and argon plasma coagulation. Snare and biopsy forceps (*CBF2.5 Wilson-cook 230*)
- **Preendoscopic sedation:** *Midazolam* in weight-based regimen (0.1ug/kg) and *Meperidine* (50-100 mg) were used for achievement of conscious endoscopic sedation.
- **Procedure:** procedure was done according to the standard recommendations (*Bejay et al., 2002*).

2) **Upper GI endoscopy:** Was done, if nasogastric lavage was bloody, the patient was hemodynamically unstable and nasogastric lavage was not bile stained and lastly, if negative colonoscopic findings.

3) **Enteroscopy:** Push enteroscopy was done for hemodynamically stable patients, if colonoscopy and upper GI endoscopic findings were negative.

B) **Mesenteric angiography:** (*Darcy, 2006*)

- **Timing:** In hemodynamic stable patients, angiography was done (within 24-48 hours from bleeding onset). For hemodynamically unstable patients, elective angiography was done (within 7 days from the clinical presentation).
- **Procedure:** Transfemoral arterial catheter was inserted and achieved cephalically. Visualization of superior mesenteric, inferior mesenteric and celiac arteries was achieved after injection of a contrast

material. A positive test result was defined as extravasations of contrast into bowel's lumen.

C) **Radionuclide scintigraphy:**

Was performed using Tc^{99} labeled RBCs. Criteria for identifying the site of LGIB included; intraluminal accumulation of radiotracer activity, increasing intensity of intraluminal activity over time and movement of the radiotracer on successive images (*Weldon et al., 2008*).

Treatment of acute LGIB:

Once bleeding site was identified, different therapeutic tools were used separately or in combination according to patient's clinical condition. Treatment options included one/or more of the followings:

1) **Medical treatment:** including I.V., fluid therapy, blood transfusion, vasoactive agents, antibiotics, and other conservative drugs.

2) **Endoscopic treatment:**

- Polypectomy for bleeding colorectal polyps.
- Argon plasma photocoagulation (APC) for angiodysplastic lesions.
- Endoscopic injection with noradrenalin, ethanolamine Oleate or foam gel for bleeding ectatic vessels.

3) **Super-selective angiography:** Transcatheter embolization of the aberrant bleeding vessels if present.

4) **Surgical management:** Exploratory laparotomy and segmental bowel resection or subtotal colectomy were done according to patient's clinical condition, site and pathology of the bleeding.

Outcomes and follow up:

Patients were followed during their hospital stay. Discharged cases were followed in our outpatient clinic and traced by telephone for any new event. Recorded data included; number of transfused RBCs units, rebleeding, length of hospital stay, occurrence of complications and mortality rate.

Statistical analysis:

Microsoft office 2007 (Excel) and Statistical Package for Social Science (SPSS) version 15 (*SPSS, INC Chicago, IL*) were used for analysis of patient's data. Prevalence rates were calculated, tests of significance used were chi-square tests, one-sample t test, and Fisher exact tests via cross-tabulation. *P* value was considered significant at value < 0.05.

3. Results

Our study included 100 patients, 63 males and 37 females, they were classified into 2 groups (according to risk assessment score); *group I* (high risk group) included 39 patients (24 males and 15 females) with

mean age (24-78 y) and mean \pm SD (56.2 ± 14.1 y) and *group II* (Moderate risk group) included 61 patients (39 males and 22 females) with mean age (30-64 y) and mean \pm SD (44 ± 15.3 y). A great significant difference was found as regard patient's age in both patients groups while, six-wise distribution showed no significant difference in both groups (Table 1).

On looking to the bleeding's site in both groups, we found that rectosegmoid junction and descending colon followed by anorectal area were the commonest sites for acute LGIB in both groups, moreover, unidentified site was reported in 4% in *group I* and in 5% in *group II* (Table 3). Moreover, we noticed that bleeding from ascending colon was found to be more severe than other sites followed by bleeding from small intestine and descending colon.

Majority of patients in *group I* were presented clinically with bright red hemochezia while, majority of patients in *group II* were presented with maroon stool. In the same direction, there were great significant differences between the two groups as regard history of previous GI bleeding, drug use and the number of comorbid diseases. Moreover, the duration of bleeding was significantly longer in *group I* than in *group II* (Table 4).

On looking to the bleeding's associated symptoms, no significant difference was found between the two groups. Abdominal pain followed by weight loss was the most frequent associated symptoms in both groups. Interestingly, majority of patients of *group I* (74.6%) had non-tender abdomen on clinical examination (63, 9%) while, more than half of patients of *group II* (63.9%) had non-tender abdomen on clinical examination. Other clinical data are listed in (Table 2 & 4).

A great significant difference was found between both groups as regard number of RBC units needed for patient's stabilization; requirement of blood units in *group I* was twice as that of *group II*. Laboratory findings great significant differences between the two groups as regard hemoglobin level, hematocrit value, PT, and INR concentration; Patients of *group I* tended to have lower hemoglobin and hematocrit level, prolonged PT and higher INR than those of patients in *group II*, however, no significant difference was found as regard platelet count between both groups (Table 5).

As regards the etiology of LGIB in both groups, diverticular disease, angiodysplasia and portal hypertensive colopathy were found to be the main causes of acute LGIB among patients in *group I* while,

colitis, malignant neoplasm and benign polyps were the main causes of bleeding in *group II*. Moreover, there was a high significant difference regarding bleeding duration; patients of *group I* had longer bleeding duration more than those of *group II*, Added to, about half of patients with colopathy and ischemic colitis tend to have severe bleeding (Table 6).

The diagnostic modalities used for diagnosis of acute LGIB were as follow (Table 7):

- Colonoscopy was found to have a good diagnostic yield in *group II* more than in *group I* while, push enteroscopy had a low diagnostic yield in both groups.
- Mesenteric angiography was done for only 3 patients of *group I*; all of them had positive angiographic findings while, 5 patients of *group II* underwent angiography and all of them showed negative angiographic findings.
- Nuclear scan was done only for 2 patients but both of them had negative findings.

Considering therapeutic procedures that were done for patients according to etiology of the bleeding (Table 7 & 8):

- With conservative medical management, bleeding stopped in 18 patients (46.2%) in *group I* and in 28 patients (49.9%) in *group II*.
- Endoscopic therapy was effective in stopping the bleeding in 15 patients (38.5%) in *group I* and in 27 patients (44.3%) in *group II*. The main endoscopic modalities used for bleeding cessation were endoscopic injection with vasoactive or sclerosant materials, APC and endoscopic polypectomy.
- Angiographic intervention using transarterial embolization was successful in bleeding cessation in 2 patients in *group I*.
- Surgical interference was indicated for 4 patients in *group I* and for 10 patients in *group II*. Rectosegmoid junction and descending colon lesions were found to be the commonest sites for surgical intervention. Surgical intervention included four surgical resections with end-to-end anastomosis, 5 proctosigmoidectomies, 4 right hemicolectomies, and one left hemicolectomy.

Duration of hospital stay was found to be significantly longer in *group I* than in *group II*, but, complete recovery was achieved in the majority of patients in both groups (71.8% & 86.9% respectively). Seven deaths were recorded in *group I* with nil deaths in *group II* (Table 8)

Table (1): Demographic data of the patients in both groups.

Variable	Group I (N=39)		Group II (N=61)		<i>t</i>	<i>p</i>
	Mean ± SD		Mean ± SD			
Age (years)	56.2 ± 14.1		44.1±15.3		3.9	0.001
Sex	No.	%	No	%	X ²	<i>p</i>
Male	24	61.9	39	63.93		
Female	15	38.5	22	36.07		

Table (2) Selected clinical signs among studied patients with in both groups.

Variable	Group I (N=39)		Group II (N=61)		X ²	<i>p</i>
	No.	%	No.	%		
Non tender abdomen	29	74.36	39	63.9	4.56	0.032
Tender abdomen	10	25.64	22	36.1		
Pulse (beat/min)	X ± SD		X ± SD		<i>t</i>	<i>p</i>
	106±11.3		96.2±8.6		5.02	0.001
Systolic BP	79.9± 13.87		110.8 ± 13.5		4.63	0.001
Diastolic BP	61± 8.7		70.1 ± 8.3		4.7	0.001

Table (3) Sites of acute LGIB among patients of both groups.

Site of bleeding	Group I (N=39)		Group II (N=61)		X ²
	N.	%	N.	%	
Anorectal	6	15.38	9	14.75	0.04
Sigmoid colon	5	12.82	7	11.46	0.18
Descending colon	4	10.26	2	3.28	1.96
Transverse colon	1	2.56	8	13.11	5.56
Ascending colon and Caecum	3	7.69	1	1.46	3.97
Recto sigmoid and Descending colon	7	20.52	23	37.70	3.91
Transverse and Ascending colon	1	2.56	5	8.20	2.64
All the colon	4	7.69	1	1.46	4.79
Small intestine	4	10.26	0	0.00	4.12
Unidentified	4	10.26	5	8.20	0.09

Table (4): Selected clinical variables among the studied patients with acute LGIB.

Variable	Group I (N=39)		Group II (N=61)		X ²	p
	No.	%	No.	%		
Clinical presentation:						
Maroon stool	6	15.34	43	70.49	28.91	0.001
Bright red hematochezia.	33	84.66	18	29.51		
Associated symptoms:						
Negative symptoms	18	46.15	17	27.87	3.5	0.06
Positive symptoms	21	53.85	44	72.13		
Abdominal pain	12	30.77	31	50.82	5.56	0.351
Anal pain	00	00	9	14.75		
Constipation	5	12.82	16	26.23		
Diarrhea	2	5.13	9	14.75		
Wight loss	8	20.51	17	27.87		
Fever	00	00	4	6.56		
Previous GI bleeding:						
Upper	4	15.4	00	00	7.44	0.006
Lower	2					
Drug history:						
Aspirin	18	46.15	10	16.39	8.80	0.032
NSAIDs	5	12.82	8	13.11		
Steroid	1	2.56	9	14.75		
Anticoagulant	4	10.26	2	3.28		
Number of comorbid disease:						
No comorbidity	3	7.69	28	45.9	22.17	0.0001
1-2 comorbid Disease.	16	41.02	24	14.8		
> 2 comorbid Diseases.	20	51.28	9	39.3		
	X ± SD		X ± SD		t	p
Duration of bleeding (hours):	42.9 ± 22		28.6 ± 18		3.4	0.001

Table (5) Hemostatic findings and need for packed RBCs among the studied patients of both groups.

Variable	Group I (N=39)	Group II (N=61)	t	p
	X ± SD	X ± SD		
Hemoglobin(g/dl)	7.7 ± 1.5	9.65 ± 1.3	7.6	0.001
Hematocrit (%)	25.5 ± 5	29.3 ± 3.6	4.37	0.001
Prothrombin time (sec)	13.2 ± 3.4	11.5 ± 2.1	3.01	0.003
INR	1.34 ± 0.49	1.1 ± 0.25	3.3	0.001
Platelet count (per cc)	193.2 ± 89.9	216 ± 62.5	1.51	0.13
Transfused packed RBCs per Pt.	4.3 ± 1.9	1.9 ± 2.1	5.7	0.0001

Table (6): Causes of acute LGIB in both groups.

Causes of bleeding	Group I (N=39)		Group II (N=61)		X ²
	N.	%	N.	%	
Diverticulosis	11	28.21	8	13.11	3.52
Angiodysplasia	5	12.82	4	6.56	0.5
Anorectal causes:					
Rectal varix	2	5.13	00	00	1.11
Solitary rectal ulcer	00	00	1	1.64	0.05
Internal hemorrhoids	4	10.26	4	6.56	0.08
Colitis:					
Ischemic colitis	1	2.26	9	14.75	3.89
IBD	2	5.13	10	16.39	1.38
Belharzial colitis	0	00	5	8.2	1.23
Malignant neoplasm	1	2.26	8	13.12	2.07
Polyps and postpolypectomy bleeding	1	2.26	7	11.48	4.26
Portal hypertensive colopathy	5	12.82	2	3.28	4.95
Others: (DL, vasculitis, haemangioma)	3	7.68	0	00	0.05
Unknown	4	10.26	3	4.92	0.07

Table (7): Diagnostic modalities used for diagnosis of acute LGIB in the studied groups.

Diagnostic tool	Group I (N=39)		Group II (N=61)		X ²
	N.	%	N.	%	
Colonoscopy:					
Total cases	38	95.44	61	100	0.05
Positive cases	30	78.95	56	91.80	4.38
Negative cases	8	21.05	5	8.20	3.19
Enteroscopy:					
Total	7	17.95	5	8.19	3.12
Positive cases	2	28.57	00	00	3.16
Negative cases	5	71.43	5	100	0.5
Nuclear scan:					
Total	1	2.56	1	1.64	0.17
Positive cases	0	00	0	00	00
Negative cases	1	100	1	100	0.17
Angiography:					
Total	3	7.67	5	8.20	0.08
Positive cases	3	100	0	00	4.79
Negative cases	0	00	5	100	3.33

Table (8): The outcomes of patients of the studied groups.

Variable	Group I (N=39)		Group II (N=61)		X ²	
	N.	%	N.	%		
Complete recovery:						
Yes	28	71.79	53	86.89	3.52	
No	11	28.21	8	13.11		
Rebleeding	4	10.26	8	13.12	0.01	
Need for surgery	4	10.26	10	16.39	0.74	
Death	7	17.95	0	00	9.18	
Duration of hospital stay(days)	X ± SD		X ± SD		t	P
	7.78 ± 3.6		4.96 ± 3.8		2.31	

4. Discussion

Lower gastrointestinal bleeding (LGIB) accounts for approximately 20 to 33% of GI bleeding with an annual incidence of about 20-27 cases per 100,000 populations in Western countries. Although LGIB is statistically less common than UGIB, it has been suggested that LGIB is underreported because, a higher percentage of affected patients do not usually seek medical attention; indeed, acute LGIB continues to be a frequent cause of hospital admission and is a factor in hospital morbidity and mortality. (20) LGIB is distinct from UGIB in epidemiology, management, and prognosis. It encompasses a wide spectrum of symptoms, ranging from trivial hematochezia to massive bleeding with shock. (5)

Acute LGIB is defined as bleeding that is of recent duration, originates beyond ligament of Treitz, and results in instability of vital signs and is associated with clinical signs of anemia with or without need for blood transfusion. (4) Despite advanced age and significant comorbid diseases, most patients with acute LGIB have favorable outcome; indeed, bleeding in majority of patients (at least 75%) with acute LGIB will stop spontaneously without any interference. (5) The hospital mortality rate of LGIB is less than 5%, and often caused by a comorbid illness or nosocomial complication rather than uncontrolled bleeding. (8)

Our study was aimed to detect the common causes of acute LGIB, to assess bleeding severity, and lastly, to discuss the available diagnostic and therapeutic procedures in our unit for management of acute LGIB.

Most published studies reported no sex predilection regarding acute LGIB, however, in our study, acute LGIB was predominant in men than in women. *Fearhead* (9) also recorded the same finding, however, *Das et al.*, (13) reported slight predominance of acute LGIB in females.

The chief presenting complaint in our study was hematochezia (84.6%) in *group I* followed by maroon stool (15.4%) in *group II*. Our finding is slight similar to that of *Gayer et al.*, (14) who reported that the chief presenting complaint in 1112 patient with acute LGIB admitted to an emergency medical center was bright red hematochezia (62%) followed by maroon stool (14.3%).

Negative symptom (absent of visible bleeding) was found in 46% of our patients in *group I* and in 28% of patients in *group II*. Moreover, abdominal pain was found to be the main associated symptom in 31% and 51% in *group I* and *group II* respectively. These results are slightly consistent with findings recorded by *Rios et al.*, (15) who reported negative symptom in 54% and 24% of patients with severe and non-severe acute LGIB respectively. Moreover, in *Rios's* study, abdominal pain was the common associated symptom in 21% and 47% of patients with severe and non-severe acute LGIB respectively.

About 46% of *group I* and 16% of *group II* were found to be chronic aspirin users; moreover, about 15% of the patients had history of previous episodes of GI

bleeding. *Wilcox and Clark* (16) reported slightly similar percentages regarding aspirin intake (52%) and previous GI bleeding (20%), but *Rios et al.*, (15) reported concomitance aspirin use in only 12% of patients with severe bleeding which required urgent surgery. Moreover, *Rios* reported that about 29% of the patients with acute LGIB have history of previous episodes of GI bleeding. Association of aspirin use may reflect its common use in elderly patients who usually have diabetes and hypertension together with diverticular disease which is common in this age category.

In our study, the mean duration of bleeding was (43±22 hours) and (29±18 hours) in *group I* and in *group II* patients respectively. *Riose et al.*, (15) reported that more than 24 hours passed before medical consultation in about 46% of patients with severe LGIB. Delay in requesting medical consultation in our study, may be related to delayed referral from primary centers. Moreover, *Zink et al.*, (17) reported mean duration of bleeding (72±16.8 hours). This much delay in patient's presentation in *Zink's* study may be related to the fact that he and his colleagues studied all cases presented with acute LGIB regardless the severity while we studied only cases presented with moderate and severe bleeding.

We reported the colon as the most common site for acute LGIB (more than 75% of cases), small intestine was a cause in ~10% of cases, and about 8~10% of cases were of unidentified origin. The rectosigmoid and descending colon followed by the anorectal area were the most frequent sites of acute colonic LGIB. Regarding the site of colonic bleeding, our conclusion was identical to that reported by *Ohyama et al.*, (18) *Ohyama* reported that small intestine as a cause of acute LGIB in only 1.7% of cases and about 11% of acute LGIB cases were of unidentified origin. Also, in *Ohyama's* study, bleeding from right side colon tend to be graver than that from left sided colonic lesion. This finding may be due to predominance of right-sided diverticular bleeding which is more common and more severe than the left colonic diverticular bleeding. (22)

Causes of acute LGIB are numerous and uncountable ranging from small bleeding piles to small intestinal lymphoma. Colonic causes represent about 85% of all causes of LGIB. (23) Diverticular disease, angiodysplasia and portal hypertensive colopathy were the most frequent causes of acute LGIB in our study, while ischemic and Bilharzial colitis and IBD followed by malignant neoplasm and benign polyps were the most frequent causes of bleeding in patients with moderate bleeding. *Wilcox and Clark* (16), *Ohyama et al.*, (18) *Rios et al.*, (15) *Gayer et al.*, (14) and *Strate and Neumann* (2) reported slightly similar findings. With more advancement in digital medical visualization that including CT virtual colonoscopy and wireless capsule endoscopy for diagnostic work up of acute and chronic LGIB, more small and large intestinal causes and unidentified etiologies may be easily diagnosed and managed. (19)

Rios *et al.*,⁽¹⁶⁾ found that diverticular disease was the main colonic cause for acute LGIB in 51 % of cases, followed by angiodysplasia and anorectal causes (28%, 16% and 15% respectively), also *Strate and Nauman* reported the diverticular disease as the main causes of acute LGIB (30-33%) followed by angiodysplasia (9-15%), colitis (8-15%) and anorectal causes (6-12%), however, slight low percents were recorded by *Cyzmek et al.*,⁽²¹⁾ regarding colonic diverticular bleeding (22.7%), angiodysplasia (22.3%), colitis (13.6%) and small bowel bleeding (9.4%). In our study, colonic diverticulosis, angiodysplasia and portal hypertensive colopathy were found to produce severe LGIB than other causes. This finding is in agreement with that of *Riose et al.*,⁽¹⁵⁾ who reported a similar finding.

About 95% of *group I* and 100% of *group II* patients in our study underwent colonoscopic examination. The diagnostic yield of colonoscopy was ranged from 79% in *group I* to 91% in *group II*. Our findings regarding the value and diagnostic yield of colonoscopic examination for patients with acute LGIB were slightly near to that of *Strate and Neumann*⁽²⁴⁾, *Gayer, et al.*,⁽¹⁴⁾, *Strate and Syngal*⁽²⁵⁾, *Smoot et al.*,⁽²⁶⁾ and *Schmulewitz et al.*,⁽²⁷⁾; They reported that the diagnostic yield of colonoscopy for detection of the site of lesions is ranging from 74% to 96% of patients presented with acute LGIB. These slight similar results regarding colonoscopic benefits were obtained regardless the site of bleeding, patient selection criteria, operator's experience and timing of colonoscopic examination. In the other hand, push enteroscopy had a low diagnostic yield; only 18% of patients with *group I* and less than 5% of patients with *group II* had positive enteroscopy findings, moreover, *Gayer et al.*,⁽¹⁴⁾ reported that the sources of bleeding were identified in only half of patients who underwent push enteroscopy.

Considerable controversy exists about the reliability of nuclear scans for localizing of the site of acute LGIB, its non-invasive nature makes it attractive, but its usefulness is questionable. *Al Qahtani et al.*,⁽²⁸⁾ used nuclear scans in 39% of patients with acute LGIB, only 24% of the scans were positive and the site of bleeding was localized only in 17% of the patients whom underwent nuclear scan. Only 2% of patients in our study underwent nuclear scan, but the scans could not specify the location of the bleeding. Moreover, *Gayer et al.*,⁽²⁴⁾ reported a positive scan in 41% of patients underwent nuclear scan, but the scans could not specify the location of bleeding, also *Zink et al.*,⁽¹⁷⁾ *Czimeck et al.*,⁽²¹⁾ and *Lee et al.*,⁽²⁹⁾ reported a low diagnostic yield of nuclear scans for the localization of the site of acute LGIB ranging from (39% to 46%).

As regard the beneficial effects of the mesenteric angiography in our study, it was found that angiography had a slight low diagnostic yield (positive data in only 3 patients with *group I* but, nil positive data in patients with *group II*). *Strate and Nauman* reported positive angiographic data in approximately 85% of patients with

severe LGIB who are hemodynamically unstable compared to 15% of hemodynamically stable patients. Moreover, *Weldon et al.*,⁽¹¹⁾ and *Fearnhead*⁽¹²⁾ reported that positive angiographic examinations were found in 25% to 70% of cases presented with acute LGIB depending on the nature and the rate of bleeding. Positive angiographic findings in patients with continuous bleeding may be due to the fact that active arterial bleeding is generally present at the time of examination, while, positive angiographic data are usually absent in cases with intermittent or slow bleeding as in moderate bleeders.⁽¹²⁾

Majority of our patients with acute LGIB were managed conservatively. Endoscopic hemostasis was effective in stoppage of LGIB in about 38% of *group I* patients and 43% of *group II* patients, while, transcatheter embolization was effective in stoppage of bleeding in only 5% of patients with severe bleeding. Surgical intervention was indicated only in about 10% of *group I* patients and in 16% of *group II* patients. Our obtained results and percentages are slightly near to that of *Rios et al.*, who reported that acute LGIB can be managed conservatively in majority of patients even with severe bleeding, and if not controlled medically, interventional endoscopy and/or radiology can currently be used and usually effective for bleeding cessation. Moreover, in *Rios's* series, surgery was indicated in approximately 10 to 25% of all patients presented with acute LGIB. Also, *Fearnhead* concluded that most cases of acute LGIB are self-limited with adequate resuscitation and only about 10% of cases require surgical intervention. The need for endoscopic, radiological and surgical interventions is actually depend upon type and clinical presentations of the patients, availability of expertise endoscopist, radiologist and surgical teams.

Complete recovery of our patients with acute LGIB was the role; 7 deaths were reported in *group I* (18%) and the overall mortality was 7%. Hospital rebleeding was recorded in 13% of patient and 10% patients in *group I* and 16% of patients in *group II* needed surgical intervention with hospital stay approximately 5-8 days. Results obtained by *Wilcox et al.*⁽¹⁶⁾ are slight near to our findings; *Wilcox et al.*, reported an overall rebleeding in 20% of patients, overall mortality was 3.6%, need for surgery was 9.7% of cases and the mean hospital stay was (6± 2.3 days). Hospital outcomes and mortality are depending upon many factors that include patient's clinical condition, patient's age and comorbidities together with availability of necessary diagnostic procedures and expertise operators. Early diagnosis and proper management after lesion localization will improve the outcomes of acute LGIB especially after the vast advancement in diagnostic and interventional radiological tools.

In conclusion, acute LGIB is a common medical and surgical challenge. Diverticular disease is by far the most common cause of acute LGIB. Medical conservation and endoscopic treatments are successful tools in stopping most cases of acute LGIB.

References

- (1) Zuccaro G. (2008): **Epidemiology of lower gastrointestinal bleeding.** Best Practice & Research Clinical Gastroenterology, Vol. 22, No. (2): p. 225–32.
- (2) Strate LL. and Nauman CR. (2010): **The Role of Colonoscopy and Radiological Procedures in the Management of Acute Lower Intestinal Bleeding.** ClinGastroenterolHepatol; 8: 333–43.
- (3) Laing CJ, Tobias T, Rosenblum DI. et al. (2007): **Acute gastrointestinal bleeding: Emerging role of multidetector CT angiography and review of current imaging techniques.** Radiographics; 27:1055-70.
- (4) Zuccaro G. (1998): **Management of the adult patient with acute lower gastrointestinal bleeding.** AJG, Vol. 93, No. 8, PP.1202-08.
- (5) Barnett J. and Messmann H. (2008): **Management of lower gastrointestinal tract bleeding.** Best Practice & Research Clinical Gastroenterology Vol. 22, No. 2, pp. 295–312.
- (6) Bramley PN, Masson JW, McKnight G. et al. (1996): **The role of an open-access bleeding unit in the management of colonic hemorrhage. A 2-year prospective study.** Scand J Gastroenterol ; 31: 764–69.
- (7) Longstreth GF. (1997): **Epidemiology and outcome of patients hospitalized with acute lower gastrointestinal hemorrhage: a population-based study.** Am J Gastroenterol; 92: 419–24.
- (8) Strate LL, Ayanian J, Kotler G. and Syngal S. (2008): **Risk Factors for Mortality in Lower Intestinal Bleeding.** Clinical Gastroenterology and Hepatology; 6, Issue 9: 1004-10.
- (9) Beejay U. and Marcon NE. (2002): **Endoscopic treatment of lower gastrointestinal bleeding.** CurrOpinGastroenterol; 18:87-93.
- (10) Darcy M. (2006): **Embolization for lower GI bleeding.** In: Golzarian J, Sun S, Sharafuddin MJ (eds) Vascular embolotherapy. Springer, Berlin Heidelberg, New York, pp 73–84
- (11) Weldon DT, Burke SJ, Sun S, et al. (2008): **Interventional management of lower GI bleeding.** EurRadiol 18: 857–67.
- (12) Fearnhead NS. (2011): **Acute lower gastrointestinal bleeding.** Medicine; 39, 2:100-04.
- (13) Das A, Ben-Menachem T, Cooper GS. et al. (2003): **Prediction of outcome in acute lower gastrointestinal hemorrhage based on an artificial neural network: internal and external validation of a predictive model.** Lancet; 362: 1261–6.
- (14) Gayer C, Chino A., Lucas C, et al. (2009): **Acute lower gastrointestinal bleeding in 1112 patients admitted to an urban emergency medical center.** Surgery; 14:2364-9.
- (15) Rios A, Mariano J, Jose M. et al., (2007): **Severe acute lower gastrointestinal bleeding: risk factors for morbidity and mortality.** Langenbecks Arch Surg; 392: 165-171.
- (16) Wilcox CM. and Clark WS. (1999): **Causes and outcomes of upper and lower gastrointestinal bleeding: The Gray hospital Experience.** Southern Medical J; 92:44-49.
- (17) Zink SI, Ohki SK, Stein, et al. (2008): **Noninvasive evaluation of active lower gastrointestinal bleeding:** AJR Am J Roentgenol; 191:1107-14.
- (18) Ohyama T, Sakurai Y, Ito M. et al. (2000): **Analysis of urgent colonoscopy for lower gastrointestinal tract bleeding.** Digestion; 61: 189–92.
- (19) Cave DR, Fleischer DE, Leighton JA, et al. (2008): **A multicenter randomized comparison of the EndoCapsule and the Pillcam SB.** GastrointestEndosc 68:487-94.
- (20) Talley NJ, Jones M.(1998):**Self-reported rectal bleeding in a United States community: prevalence, risk factors, and health care seeking.**Am J Gastroenterol. Nov; 93(11): 2179-83.
- (21) Czymek R, Kempf A, Roblick U. et al. (2008): **Surgical Treatment Concepts for Acute Lower Gastrointestinal Bleeding.** GastrointestSurg; 12:2212–20.
- (22) Barnett J. and Messmann H. (2009): **Diagnosis and Management of lower gastrointestinal tract bleeding.** Nat. Rev. Gastroenterol. Hepatol. 6, 637–46.
- (23) Hoemeda RE. and Luchtefeld MA. (2005): **The Management of acute Lower Gastrointestinal Hemorrhage.** Dis Colon Rectum; 48: 2010–24.
- (24) Neumann S, Schoppmeyer K, Lang T. et al. (2007): **Wireless capsule endoscopy for diagnosis of acute LGIB.** Gastro IntestEndosc; 65:403-40.
- (25) Strate LL. and Syngal S. (2003): **Timing of colonoscopy: impact on length of hospital stay in patients with acute lower intestinal bleeding.** Am J Gastroenterol; 98: 317–22.
- (26) Smoot R, Gostout C, Rajan R. et al., (2003): **Is early colonoscopy after admission for acute diverticular bleeding needed.** Am J Gastroenterol; 98:1996-99.
- (27) Schmulewitz N, Fisher DA. and Rockey DC. (2003): **Early colonoscopy for acute lower GI bleeding predicts shorter hospital stay: a retrospective study of experience in a single center.** GastrointestEndosc; 58: 841–46.
- (28) Al Qahtani AR, Satin R, Stern J. et al. (2002): **Investigative modalities for massive lower gastrointestinal bleeding.** World J Surg; 26:620-25.
- (29) Lee j., Costantini TW. and Coimbra R. (2009): **Management of lower GI bleeding.** Scandinavian Journal of Surgery 98: 135–14