

CLINICAL STUDY

Significant associations of acute gastrointestinal haemorrhage and the estimation of effectiveness of the newly proposed acute abdominal bleeding score (AABS) in emergency patients

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Abstract: This study was carried out in different units of surgical wards of Khulna Medical College Hospital, Bangladesh. Among the total number of 284 patients about 88.7 % (252) was admitted for the upper GIT pathology and in relation to this, lower GIT bleeding was less common 11.2 % (32). In the question of emergency surgical management, 156 (61.9 %) patients out of 252 upper GIT bleeding patients needed an emergency surgery, whereas it was only 12 (37.5 %) out of the rest of lower GIT bleeding patients. Fortunately, in this study, it was clearly found that, in case of bleeding control, 234 (82.3 %) patients had it without intervention. This study also suggests that only 29.6 % (84) patients had specific clinical manifestation out of 284 patients, followed by 43 % (122) who had vague signs-symptoms and the rest 27.5 % (78) had latent symptoms of acute haemorrhage. In case of diagnosis of acute hemorrhagic patients, only 34.5 % (98) was diagnosed clinically. A 36 % (102) patient out of 284 patients was diagnosed by ultrasound of abdomen and 14.8 % (42) was diagnosed by endoscopy. It was a very remarkable finding of this study that 33.8 % (96) patients were remained undiagnosed till admission to first 24 hours and among 284 hemorrhagic patients, only 7.7 % (22) required the emergency surgery on admission. In the type of acute episodic bleeding as well as in associated family history, some significant positive associations were found.

The most important contribution of this clinical study was a very new but effective score (Acute Abdominal Bleeding Score=AABS) for the diagnosis. Also the clinical status of patients had been proposed which could be a very good tool for measuring the severity of acute abdominal hemorrhage in patients on admission (Fig. 6, Tab. 7, Ref. 46). Full Text (Free, PDF) www.bmj.sk.

Key words: acute gastrointestinal haemorrhage, estimation of effectiveness, newly proposed acute abdominal bleeding score, emergency patients.

Acute Gastrointestinal Hemorrhage

Acute gastrointestinal (GI) hemorrhage is a frequent cause of admission to the surgical intensive care unit. Upper gastrointestinal (UGI) causes are more common (85 %) than lower gastrointestinal (LGI) causes (15 %). The likelihood that emergency surgery will be necessary is estimated at 40 % for UGI sources and at 30 % for LGI sources (1). Clinically, the distinction between UGI and LGI sources is important.

Practically, LGI bleeding is hemorrhage that occurs beyond the range of the UGI endoscope. Fortunately, bleeding ceases in

85 % of patients without intervention. The remaining 15 % of patients require early, accurate diagnosis and therapy (2).

UGI hemorrhage

In the United States, the estimated rate of hospitalization for UGI bleeding is ~100/100,000 patients per year. Typically, patients are 70 years of age and have one or more major chronic organ system diseases (3). Patients hospitalized for another reason before significant bleeding begins have a mortality of 70 % (4). In the hospitalized patient, tachycardia, hypotension, or anemia should suggest the possibility of GI hemorrhage. The most common causes of UGI hemorrhage are esophageal varices, gastric ulcer, and duodenal ulcers. In two large series, these entities accounted for 20 % of UGI bleeding sources (4, 5). Other causes included esophageal ulcers, malignant ulcers, hiatal herniae, and diverticula. Each of these entities was associated with a 5 % chance of producing clinically significant hemorrhage. In the series of Chalmers et al (5), esophageal varices were demonstrated in 33 of 44 patients with cirrhosis. In 16 of these 33 patients, no point of rupture of varices was found. Yet in only one of these patients was seen another possible source of bleeding, a

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Acknowledgement: I am highly grateful to my respected teacher, Prof. DR. Saroj Kumar Mazumder, the honorable Head of the Department of Community Medicine, Chittagong Medical College, Bangladesh, for his kind permission, support and help for conduct me this study and also thankful to the respondents who gave their valuable time to enhance me the study.

chronic duodenal ulcer. When faced with the unusual situation of a patient with apparent GI bleeding, known varices, no evidence of bleeding from the varices, and no other identifiable source, the clinician should continue to suspect variceal bleeding until another source can be found.

UGI endoscopy can locate a bleeding lesion, assess the risk of re-bleeding, and, with increasing frequency, control bleeding. With respect to locating the lesion, diagnostic accuracy of 90–95 % is typical (6, 7). With respect to the risk of re-bleeding, several observations have special importance. Active arterial bleeding with streaming of blood, clot adherence to a lesion, an exposed vessel that protrudes from the lesion, and staining of the ulcer base within a lesion are collectively known as stigmata of recent hemorrhage (SRH) (8).

Presence of SRH is associated with increased re-bleeding risk and increased mortality. For ulcers actively bleeding at the time of endoscopy, the re-bleeding rate has been reported to range from 53 % to 100 %. For collected series, the mean rate is 66 % (9). For patients with visible vessels in ulcers, the re-bleeding rate is ~50 %, and the emergency surgery rate has been reported at 52 % (9, 10). Other SRH have a lower incidence of re-bleeding, 10 % (10). In one large series, mortality for patients with SRH was 12 %; whereas, for patients without SRH, the mortality was 0 % (11). Early endoscopy in acutely bleeding patients presumably would have several advantages.

First, surgery, when indicated, would be performed earlier. With more rapid definitive surgery, the patient is likely to receive fewer transfusions. Second, the surgical procedure could be directed at the specific lesion and site. Accurate preoperative diagnosis would reduce operating time and eliminate inappropriate surgical procedures. Accurate diagnosis also should eliminate the need for blind gastrectomy. Third, accurate preoperative diagnosis would eliminate surgical exploration to detect the bleeding source. Fourth, surgical risk could be predicted better, especially for treatments with an expected high morbidity, such as total gastrectomy for hemorrhagic gastritis. The lavage, which precedes endoscopy, may slow or stop bleeding. In addition, endoscopy can suggest long-term therapy, such as abstention from aspirin or alcohol (12). For patients with continued active bleeding, early and specific diagnosis with UGI endoscopy remains vital for selecting appropriate therapy (13).

Common causes

Mallory-Weiss lesion

The Mallory-Weiss lesion is a linear mucosal tear, usually found on the lesser curvature of the stomach, either at or below the gastroesophageal junction. The lesion is diagnosed by UGI endoscopy in 95 % of patients. Seventy percent of cases of Mallory-Weiss tears can be effectively treated with blood transfusion, control of gastric pH, and saline irrigation. If surgery is necessary, a gastrotomy is made high on the stomach to allow oversewing of the bleeding point. Endoscopic coagulation also has been used successfully (14). Although some authors advocate the use of a Sengstaken-Blakemore tube or other balloon tam-

ponade device, the risk of converting a partial thickness lesion into a full thickness perforation is present and should relegate balloon tamponade to a treatment of last resort.

Esophageal varices

The risk of bleeding from esophageal varices is increased with larger size, location at the gastroesophageal junction, presence of red stigmata at endoscopy, advanced liver failure, and advanced ascites. Patients with Child-Pugh class A cirrhosis have a lower incidence of variceal bleeding, 5–7 %, than those with Child-Pugh class C cirrhosis, who have a 70 % risk (15). Although 85–90 % of patients with nonvariceal UGI bleeding will cease bleeding spontaneously, those patients with variceal bleeding have a 50 % chance of spontaneous cessation of bleeding. Early re-bleeding is common, and 50 % of re-bleeding events occur within 10 days of the index bleeding episode. Treatment of esophageal varices is varied. Balloon tamponade is a decades old therapy and can provide temporary control of bleeding. For combined series, a 78 % rate of bleeding control has been achieved, with a re-bleeding rate of 42 %. The combination of the high re-bleeding rate after deflation of the balloon with the potential for serious complications, including esophageal necrosis and aspiration, relegates the use of balloon tamponade to cases not controlled by drug or endoscopic therapy (16).

Should balloon tamponade be selected, the clinician should follow a careful protocol to achieve control of bleeding with minimum complications.

Protocol for Use of Sengstaken-Blakemore Tube

Before insertion

- 1) Consider nasotracheal intubation
- 2) Use new tube and check balloon for leaks
- 3) Attach No. 18 Salem sump tube above esophageal balloon
- 4) Evacuate blood from stomach with a large tube
- 5) Insert tube through nose using ring forceps, if necessary

After insertion

- 1) Apply low, intermittent suction to stomach tube
- 2) Apply constant suction to Salem sump
- 3) Inflate gastric balloon with 25-mL increments of air to 100 mL, observing patient for pain
- 4) Snug gastric balloon to gastroesophageal junction and affix to nose, under slight tension, with soft rubber pad
- 5) Add 150 mL of air to gastric balloon
- 6) Place two clamps (one taped close) on tube to gastric balloon
- 7) Inflate esophageal balloon to 24–45 mm Hg, clamp, and check every hour
- 8) Perform heavily penetrated upper abdomen-lower chest roentgenography (portable) to confirm balloon positions
- 9) Determine serial hematocrit levels every 4–6 h (gastric tube may occlude and fail to detect recurrent hemorrhage)
- 10) Tape scissors to head of bed so tube can be transected and rapidly removed if respiratory distress develops
- 11) Deflate esophageal and gastric balloons after 24 h

12) Remove tube in an additional 24 h if there is no recurrent hemorrhage

Source

From Rikkers LF, ed. Non-operative emergency treatment of variceal hemorrhage. *Surg Clin North Am* 1990; 70:297.

Vasoconstrictor therapy has been useful in controlling acute variceal bleeding. The American College of Gastroenterology recommends the empiric use of vasoactive therapy in the patient with a high likelihood of variceal bleeding before a definitive bleeding site is identified. Commonly used agents are octreotide and the combination of vasopressin and nitroglycerin. Octreotide, a longer acting analog of somatostatin, does not adversely affect cardiac function or blood pressure and can be used with safety in the patient with coexisting cardiac disease. Patients do not require special monitoring. The combination of safety and relative ease of use has made octreotide the initial choice for suspected or confirmed variceal bleeding. The infusion is generally continued for five days. Vasopressin reduces portal tributary inflow and, consequently, decreases portal pressure. In addition, vasopressin infusion may induce cardiac or splanchnic ischemia or cardiac bradydysrhythmias. Concomitant use of nitroglycerin can mitigate some of these side effects and allow higher doses of vasopressin to be used (16, 17). Endoscopic therapy includes endoscopic sclerotherapy and endoscopic variceal ligation (EVL). These two procedures are now commonly available and allow the possibility of definitive therapy at the time of endoscopic diagnosis. Because of the complications associated with sclerotherapy, including esophageal perforation, esophageal stenosis, and ulcer bleeding, EVL has been used with a lower complication rate. Both sclerotherapy and EVL control acute bleeding in 80 % of patients. Recent data favor EVL over sclerotherapy in the long-term prevention of re-bleeding (18).

Sclerotherapy has been compared with surgical shunts in several studies. In controlled trials with follow-up of two to five years, sclerotherapy resulted in a decreased re-bleeding rate or decreased transfusion requirements when compared with medical therapy. Despite improved control of bleeding, studies do not demonstrate a clear increase in survival (19–23). When compared with portacaval shunt, sclerotherapy has a significantly higher re-bleeding rate. Despite better control of bleeding, portacaval shunt does not improve either encephalopathy or mortality (24). Three controlled trials have compared sclerotherapy with distal splenorenal shunt (DSRS). In all three trials, DSRS significantly decreased the re-bleeding rate but did not change survival (25–27). Transhepatic internal jugular portosystemic shunts (TIPS), placed in the radiology suite, are now available.

In four studies that compared TIPS with endoscopic treatment, re-bleeding was reduced, encephalopathy was higher, and survival was comparable (28). With acute bleeding, rapid control of hemorrhage is critical. As neither TIPS nor surgery can be performed as rapidly as endoscopic and pharmacologic therapies, these modalities are not recommended as first line therapy. TIPS has been successfully used as salvage therapy for patients

who have failed endoscopic control of bleeding (29). A comprehensive review can be found in Ref. 30.

Gastritis

Association of ulceration of the gastric mucosa with subsequent stress bleeding and critical illness has a long history. Cushing reported acute duodenal ulceration in patients with major burn injury in 1842. Cushing's ulcer refers to the association of peptic ulcers with traumatic brain injury. With advances in resuscitation, metabolic support, and prophylaxis, the incidence of stress bleeding and surgical intervention for massive stress gastritis hemorrhage has decreased. The mortality for patients who require intervention for stress related bleeding remains high, up to 50 % (31, 32). Prophylaxis against stress bleeding is effective. Antacids, H₂-receptor antagonists, and sucralfate all have demonstrated efficacy. Several studies demonstrate that antacids decrease stress bleeding when compared with placebo or control groups. For collected series, a bleeding rate of 18.9 % in the placebo or control groups was reduced to 7.1 % in the antacid group. H₂-receptor antagonists can provide a similar degree of bleeding prophylaxis. Again, from collected series, bleeding in control groups was 17.1 %. In patients treated with H₂ blockers, the bleeding rates in collected series were 6 % to 7 %. Several studies confirm the efficacy of sucralfate for prophylaxis. From pooled data, the bleeding incidence in the sucralfate group was 3.8 %; in the antacid/H₂-blocker groups, it was 8 % (32). These findings suggest that all patients in the ICU would receive benefit from stress ulcer prophylaxis. One shortcoming of the foregoing studies is that microscopic bleeding was not distinguished from clinically significant bleeding. The incidence of clinically significant bleeding was observed to be 1.5 % in a multicenter trial of more than 2000 patients. Two independent risk factors for clinically significant bleeding were determined: respiratory failure and coagulopathy. The investigators concluded that stress ulcer prophylaxis could be safely withheld from patients in the ICU unless they have a coagulopathy or respiratory failure requiring mechanical ventilation (33). This conclusion arises from the estimation of the numbers needed to treat to prevent a complication. For those patients without coagulopathy and not requiring mechanical ventilation, more than 900 patients would need to receive prophylaxis to prevent a single bleeding episode. In contrast, in mechanically ventilated patients with coagulopathy, only 30 patients would need prophylaxis to prevent a bleeding complication. In a subsequent investigation, Cook et al (34) compared the efficacy of ranitidine and sucralfate. The incidence of clinically important bleeding was 1.7 % in the ranitidine group and 3.8 % in the sucralfate group. The authors observed no significant differences in the incidence of ventilator associated pneumonia, ICU length of stay, or mortality. In both of these studies, the number of patients with head injury was small. Patients with CNS injuries, especially those involving the diencephalon and the brain stem, may have disinhibition of the medullary vagal system. Stress induced stimulation of brain neuropeptide production may directly affect the gut. The combination of these

Tab. 1. Causes of Acute LGI Hemorrhage in a Series of Patients During a 4-Year Period.

Diagnosis	No of patients
Diverticular disease	30
Carcinoma of colon	28
Inflammatory bowel disease	17
Colonic polyps	10
Vascular ectasia	6
Ischemic colitis	3
Rectal ulcer	3
Hemorrhoids	2
Anticoagulant treatment	1
Thrombocytopenia	1
Malignant histiocytosis	1
Anastomotic bleeding	1
Undetermined	1
Total	105

Source: From Ref. (42). Farrands PA, Taylor I. Management of acute lower gastrointestinal haemorrhage in a surgical unit over a 4-year period. *J Royal Soc Med* 1987; 80:79–82.

two effects may result in a higher incidence of stress ulceration and hemorrhage. For patients with head injury, especially those requiring mechanical ventilation, our practice is the use of stress ulcer prophylaxis. Lack of efficacy of H₂-receptor antagonists in controlling pH in patients with severe CNS injury has been shown. Sucralfate or antacids may be preferable to H₂-receptor antagonists in this population of patients (35).

Although effective prophylaxis can be achieved by alkalinizing stomach secretions to achieve a gastric pH 4, an increased incidence of nosocomial pneumonia with increasing pH has been demonstrated. The risk of gastric colonization and consequent nosocomial pneumonia may be comparable to the risk of stress bleeding. Selective decontamination of the digestive tract is effective in reducing nosocomial pneumonia and other infections (36). For critically ill patients who are expected to remain in ICU for at least 5 days, gut decontamination is effective in reducing nosocomial infection. The use of gut decontamination regimen should be considered for patients receiving gastric stress bleeding prophylaxis, especially if these patients are receiving broad-spectrum antibiotic therapy.

Peptic ulcer disease

The mainstay of peptic ulcer disease treatment is medical. Proton pump inhibitors, therapy aimed at *Helicobacter pylori*, and H₂-receptor antagonists are effective. With a re-bleeding rate that is 65 %, further therapy, including emergency surgery, is frequently necessary for ulcers that are acutely bleeding at the time of endoscopy. Other SRH are associated with an increased risk of re-bleeding. In a meta-analysis of endoscopic therapy for acute nonvariceal UGI hemorrhage, Cook et al found that endoscopic therapy results in a reduction in morbidity and mortality rates. In the series reviewed, the fraction of patients with peptic ulcer disease approached 1.0. Monopolar and bipolar electroco-

agulation, injection therapy, and laser coagulation all significantly reduced re-bleeding rates. With respect to mortality, all modalities reduced mortality, but only laser coagulation achieved statistical significance (37). A study of 100 patients with bleeding peptic ulcer diatheses in whom control of bleeding by endoscopic means was achieved demonstrated significantly decreased re-bleeding when they received a proton pump inhibitor rather than an H₂-receptor antagonist (38). These findings were corroborated by Lau et al. Re-bleeding after control of ulcer bleeding was decreased when the patient received intravenous proton pump inhibitor following control of the bleeding and oral proton pump inhibitor for 2 months (39).

Surgical treatment is indicated for severe or unrelenting bleeding. A definitive ulcer procedure can be performed; the choice of procedure depends on the condition of the patient and the severity of the bleeding. In a series of more than 1000 patients with bleeding duodenal ulcers, 250 patients had emergency surgery. Truncal vagotomy and antrectomy were performed in all. In the emergency surgery group, the mortality was 5.5 % (29). A discussion of specific surgical considerations is beyond the scope of this chapter but may be found in Ref. 40.

LGI hemorrhage

The most likely causes of LGI hemorrhage are related to age. In children, Meckel's diverticulum is common; whereas, in adults over 60 years of age, vascular ectasias are frequently associated with severe LGI hemorrhage (41). In one large series, the most common causes of LGI bleeding were diverticular disease, colon cancer, inflammatory bowel disease, and colonic polyps (31) (Tab. 1). Vascular ectasias, ischemic colitis, rectal cancer, and hemorrhoids all had an incidence of 10 %. Although vascular ectasias had a 6 % incidence, they accounted for one-third of patients requiring emergency surgery and 50 % of patients requiring 4 units of blood in the first 24 h. Vascular ectasias are usually multiple and most commonly found in the cecum or proximal ascending colon. Two thirds of affected patients are over 70 years old. The lesions are thought to be acquired (41). Approximately 20 % of patients with LGI bleeding have a transfusion requirement of 3 units. A transfusion requirement of 3–4 units or more of blood in the first 24 h is associated with an increased need for emergency surgery. In one large series, all patients who required emergency surgery needed 4 units of blood in the first 24 h (42).

Patient evaluation

Evaluation of patients with suspected LGI hemorrhage should begin with a thorough physical examination, including a careful rectal examination. Coagulation studies and platelet counts should be obtained. A BUN/creatinine 25 distinguishes UGI bleeding from LGI bleeding in 90 % of patients (32). In 10–15 % of patients, the source of presumed LGI bleeding is actually a UGI source. UGI endoscopy or nasogastric tube aspiration is indicated. Absence of blood and presence of bile in the aspirate vir-

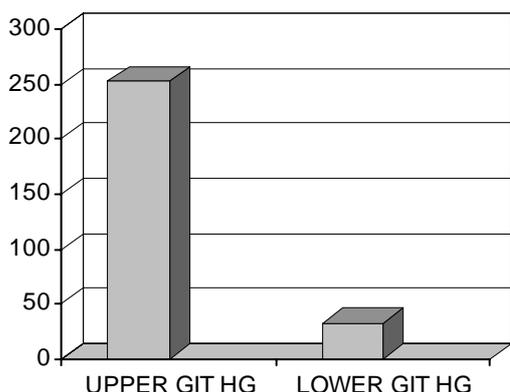


Fig. 1.

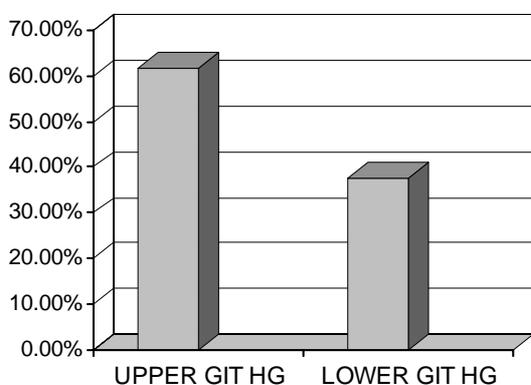


Fig. 2.

tually exclude a bleeding source proximal to the ligament of Treitz (41). In recent years, colonoscopy has become the diagnostic procedure of choice in the stable patient, in whom bleeding has stopped or significantly decreased. Some authors advocate colonoscopy in the patient with severe hematochezia and report 74–82 % diagnostic accuracy (43, 44). If colonoscopy cannot be performed or is unsuccessful, angiography should be performed in the patient with persistent, severe bleeding. If the patient's condition is stable or cessation of bleeding is not certain, colonoscopy should be followed by tagged-RBC study, if no diagnosis is made endoscopically. Although not as definitive as angiography in identifying the site of bleeding, the tagged-RBC study may be more sensitive in detecting bleeding. Increased sensitivity of the nuclear medicine study arises from the potential to accumulate radionuclide at the bleeding site over several hours. It does not depend on active bleeding during the brief period of colonoscopic examination or of injection of radiocontrast agent. Furthermore, several examinations can be obtained for up to 36 h after injection of the radionuclide (45). If the radionuclide study suggests a site of bleeding and the patient's condition is stable, colonoscopy is next performed.

The examination should be abandoned if technical problems are encountered. If the patient continues to bleed actively, an-

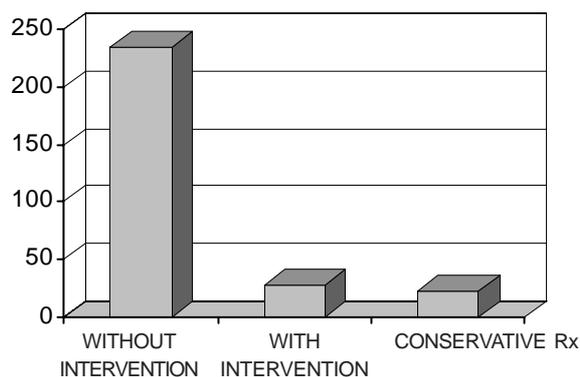


Fig. 3.

giography is the next study. Bleeding rates of 0.5 mL/min can be detected. Angiography is successful in locating the source of bleeding in one-half to two-thirds of patients. Angiographic diagnostic success is increased if angiography is performed within 24 h of the onset of bleeding (41, 46). In most patients, bleeding will spontaneously stop. If a source of bleeding has not been identified, repeat colonoscopy should be performed. Studies to evaluate the small bowel, including UGI with small bowel follow-through or enteroclysis, should be considered. In the small group of patients who continue to lack diagnosis, repeat angiography, including the celiac axis, should be performed. In an occasional patient, hemobilia may be the source of bleeding. Conservative management of LGI hemorrhage is indicated initially. Approximately 90 % of bleeding will stop before transfusion requirements exceed 2 units. If 4 units are required in 24 h, the chance that surgery will be necessary approaches 50 %. An aggressive search for the source should be undertaken. If a bleeding source is clearly identified, segmental resection is recommended. If a bleeding source is not clearly identified, subtotal colectomy with or without ileorectal anastomosis should be done (42).

Methods and materials

- 1) Type of study: Descriptive type of epidemiological cross-sectional study.
- 2) Place of study: The general surgery indoor department of Khulna Medical College Hospital, Bangladesh.
- 3) Period of study: From 10.12.08 to 03.05.09
- 4) Study population: Patients, admitting in the general surgery indoor department of Khulna Medical College Hospital, Bangladesh.
- 5) Sample size: 284
The sample size was selected by using the formula $Z^2pq \cdot D$
Where, Z =given confidence level. ($Z=1.96$ for 95 % confidence level)
 p =Probability =20 %=0.20
 $q=1.0-p=0.8$ ($C1-p$)
Degree of error limit (the accuracy desired)
- 6) Sampling technique: Convenient type of purposive sampling.

Table 2

Age	Male Female	p-value
<10	52	0.05
%	3.1	1.6
10—29	24	17
%	15.2	13.5
30—49	32	26
%	20.3	20.6
50-69	45	32
%	28.5	25.4
≥70	78	49
%	49.4	38.9

Table 3

Sex	Mortality	Morbidity over 2 weeks to 1 month	Morbidity over 1 month	p-value
Male	30	72	38	0.01
%	19	45.6	24.1	
Female	22	58	28	
%	17.5	46	22.2	

Table 4

Primary causes	Number	%	p
Diverticular diseases	44	15.5	0.05
Oesophageal bleeding	42	14.8	
Ectopic pregnancy and other gynecological causes	20	7	
Carcinoma colon	32	11.3	
IBD	24	8.5	
Colonic polyp	13	4.6	
Ischemic colitis	5	1.7	
Rectal ulcer	10	3.5	
Hemorrhoids	28	9.9	
Thrombocytopenia	11	3.9	
Anastomotic bleeding	2	0.7	
Others	36	12.7	
Undetermined	47	16.5	

- 7) Data collection instruments: a) By preparing questionnaires. b) By direct observation. c) By active participation.
- 8) Data collection period: From 10.12.08 to 29.04.09
- 9) Methods of data collection: a) By interviewing through questionnaires. b) By direct observation.
- 10) Data analysis: After collection, data were checked, verified, compared, reviewed and analyzed according to the objectives and purposes of the study.

Using computer based statistical package statistical analysis of the data was done. Data were analyzed with SPSS computer package programme. The survey data was usually analyzed using descriptive statistic. Such as; mean, SD, percentage, co-efficient of variation.

Report was produced by computer based programme- Microsoft Word, Power point, Photoshop, Adobe and other accessories.

Result and interpretations

This study was carried out in different units of surgical wards of Khulna Medical College Hospital, Bangladesh from 10.12.08 to 03.05.09 which brought the following findings related to acute gastrointestinal haemorrhage:

Among the total number of 284 patients about 88.7 % (252) was admitted for upper GIT pathology and in relation to this, lower GIT bleeding was less common 11.2 % (32) (Fig. 1).

In the question of emergency surgical management, 156 (61.9 %) patients out of 252 upper GIT bleeding patients needed emergency surgery, whereas it was only 12 (37.5 %) out of the rest of lower GIT bleeding patients (Fig. 2).

Fortunately, in this research, it was clearly found that, in case of bleeding control, 234 (82.3 %) patients had it without intervention (Fig. 3).

The age and sex distribution those were found in this study are mentioned in the Table 2.

The mortality and the related morbidity in different scale has been demonstrated in Table 3.

In the question of relative associated specific etiology, the answers are listed in the Table 4.

Figure 4 suggest that only 29.6 % (84) patients had specific clinical manifestation out of 284 patients, followed by 43 % (122) had vague signs-symptoms and the rest 27.5 % (78) had latent symptoms of acute haemorrhage.

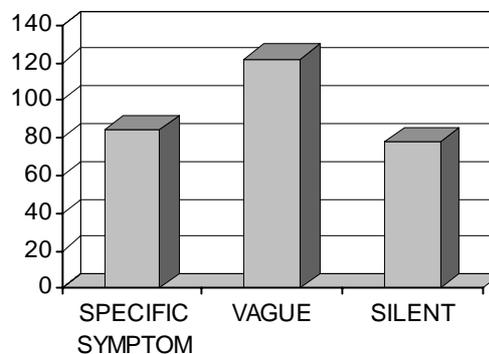


Fig. 4.

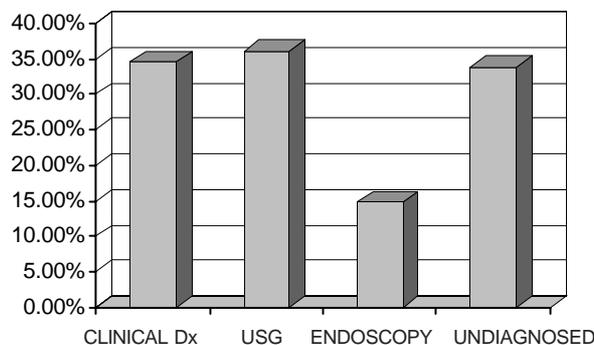


Fig. 5.

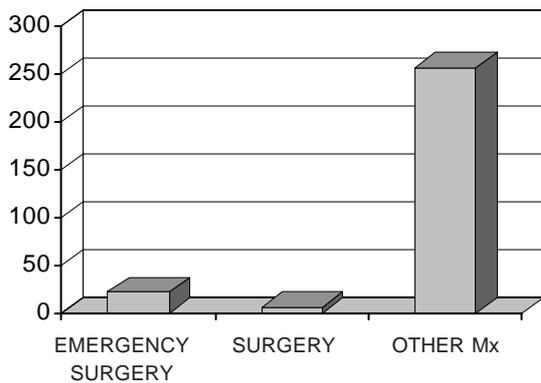


Fig. 6.

In case of diagnosis of acute hemorrhagic patients, only 34.5 % (98) was diagnosed clinically. A 36 % (102) patient out of 284 patients was diagnosed by ultrasound of abdomen and 14.8 % (42) was diagnosed by endoscopy. It was a very remarkable finding of this study that 33.8 % (96) patients were remained undiagnosed till admission to first 24 hours (Fig. 5).

Among the 284 hemorrhagic patients, only 7.7 % (22) required emergency surgery on admission (Fig. 6).

In the question of same type of acute episodic bleeding as well as associated family history, the relative observations are given in Table 5.

In case of some other relative associations, the findings are listed in Table 6.

Conclusion and recommendation

Honestly I believe that this is nothing but an assumption and time is the vital deadline here and it will take more years of debate and study before an original fruitful final conclusion is

Table 5

Sex	Total pts	Past episode	Positive family history	p
Male	158	48	52	0.01
%	30.4	32.9		
Female	126	39	41	0.01
%	31	32.5		

Table 6

Sex	Aspirin and other relevant drugs	Cigarette smoking	Alcohol	Any of 2	All of them	p
Male	6	92	58	43	2	0.01
%	1.3	58.2	36.7	27.2	1.3	
Female	2	12	23	8	-	
%	1.6	9.5	18.3	6.3	-	

Table 7

1) Non specific etiology (Total Score 10)				
	Highest score	Lowest score		
Smoking	1	0		
Alcohol	1	0		
Aspirin and other anti-platelet and relevant drugs	1	0		
Family history of AAB	1	0		
Family history of specific abdominal pathology and/or neoplasm	1	0		
Age over 50-70 or <70	1 or 2	0		
Previous episodes of AAB for single or more than 1time	1 or 3	0		
	10			
2) Clinical presentation (Total Score 4)				
Signs and symptoms				
Specific	4	0		
Vage	1	0		
3) Specific primary pathology (Total Score 4)				
Present	4			
Absent	0			
4) Other consideration by the judgment of the physicians (Total Score 2)				
Highest score	2			
Lowest score	0			
Interpretation				
	Highly significant	Significant	Consider significant	Suspicious
Non specific etiology	≥6	≥4	≥3	≥3
Clinical presentation	4	4	4	1
Specific presentation	4	4	4	1
Physician consideration	2	1	0	0
Total	≥16	≥13	≥11	≥5
So, the approximately range of interpretation for diagnosis and evaluation of AAB patients:				
Total highest score	= 20			
Highly significant	≥16			
Significant	13 to 15			
Consider significant	11 to 12			
Suspicious	6 to 10			
Not significant	≤5			

reached. I am quite certain about the fact and it is a reality without any doubt. So, it is very clear that though the study was held on a very small sample of population in the general surgery indoor department of Khulna Medical College Hospital, Bangladesh, it may be unable to depict the more realistic picture as a whole- in fact the actual situation may be more severe than it is

depicted here. Moreover, no satisfactory number as well as level of study in this relation is available in our developing country now. So the most important pearl is that indeed a study in large scale should be required just now having a more realistic and a more accurate image of this alarming problem.

A very new score (Acute Abdominal Bleeding Score=AABS) for the diagnosis as well as realizing the clinical status of patients can be proposed and to my mind, it can be a very effective tools for measuring the severity of the acute abdominal hemorrhagic patients on admission.

Proposed Acute Abdominal Bleeding Score (AABS) in suspected patients (Tab. 7)

References

1. Longstreth GF. Epidemiology of hospitalization for acute gastrointestinal hemorrhage: A population based study. *Am J Gastroenterol* 1995; 90: 206—210.
2. Gostout CS. Acute gastrointestinal bleeding-A common problem revisited. *Mayo Clin Proc* 1988; 63: 596—604.
3. Cutler JA, Mendeloff AI. Upper gastrointestinal bleeding: Nature and magnitude of the problem in the US *Dig Dis Sci* 1981; 26 (Suppl 7): 90S—96S.
4. Chojkier M, Laine L, Conn HO, Lerner E. Predictors of outcome in massive upper gastrointestinal hemorrhage. *J Clin Gastroenterol* 1986; 8: 16—22.
5. Chalmers TC, Zamcheck N, Curtins GW. Fatal gastrointestinal hemorrhage: Clinicopathologic correlations in 101 patients. *Am J Clin Pathol* 1952; 22: 633—645.
6. Dagradi AE, Arguello JF, Weingarten ZG. Failure of endoscopy to establish a source for upper gastrointestinal bleeding. *Am J Gastroenterol* 1979; 72: 395—402.
7. Lieberman D. Gastrointestinal bleeding: Initial management. *Gastroenterol Clin North Am* 1993; 22: 723—736.
8. Foster DN, Miloszewski KJA, Losowsky MS. Stigmata of recent haemorrhage in diagnosis and prognosis of upper gastrointestinal bleeding. *Br Med J* 1978; 1: 1173—1177.
9. Pescovitz MD, Satterberg TL, Shearen JG. Endoscopic control of bleeding ulcers: The Minnesota experience with several methods. In: Najarian JS, Delaney JP, eds. *Progress in Gastrointestinal Surgery*. Chicago, IL: Year Book, 1989: 247—254.
10. Swain CP, Storey DW, Bown SG, Heath J, Mills TN, Salmon PR, Northfield TC, Kirkham JS, O'Sullivan JP. Nature of the bleeding vessel in recurrently bleeding gastric ulcers. *Gastroenterology* 1986; 90: 595—608.
11. Brearley S, Morris DL, Hawker PC, Dykes PW, Keighley MR. Prediction of mortality at endoscopy in bleeding peptic ulcer disease. *Endoscopy* 1985; 17: 173—174.
12. Dagradi AE, Ruiz RA, Weingarten ZG. Influence of emergency endoscopy on the management and outcome of patients with upper gastrointestinal hemorrhage. *Am J Gastroenterol* 1979; 72: 403—415.
13. Domschke W, Lederer P, Lux G. The value of emergency endoscopy in upper gastrointestinal bleeding: Review and analysis of 2014 cases. *Endoscopy* 1983; 15: 126—131.
14. Todd GJ, Zikira BA. Mallory-Weiss Syndrome. *Ann Surg* 1977; 186: 146—148. 566 Abrams
15. Prediction of the first variceal hemorrhage in patients with cirrhosis of the liver and esophageal varices. A prospective multicenter trial. The North Italian Endoscopic Club for the study and Treatment of Esophageal Varices. *New Engl J Med* 1988; 319: 983—989.
16. Carey WD, Grace ND, Reddy KR, Shiffman ML. Managing variceal hemorrhage in the cirrhotic: A primer. *Am Coll Gastroenterol* 1998; 1—12.
17. Burnett DA, Rikkers LF. Nonoperative emergency treatment of variceal hemorrhage. *Surg Clin North Am* 1990; 70: 291—306.
18. Laine L, Cook D. Endoscopic ligation compared with sclerotherapy for treatment of esophageal variceal bleeding. A meta-analysis. *Ann Intern Med* 1995; 15: 280—287.
19. Terblanche J, Bornman PC, Kahn D, Jonker MA, Campbell JA, Wright J, Kirsch R. Failure of repeated injection sclerotherapy to improve long-term survival after oesophageal variceal bleeding. A five-year prospective controlled trial. *Lancet* 1983; 2: 1328—1332.
20. Copenhagen Esophageal Varices and Sclerotherapy Project. Sclerotherapy after first variceal hemorrhage in cirrhosis: A randomized multicenter trial. *New Engl J Med* 1984; 311: 1594—1600.
21. Westaby D, MacDargall BRD, Williams R. Improved survival following injection sclerotherapy for esophageal varices: Final analysis of a controlled trial. *Hepatology* 1985; 5: 827—830.
22. Korula J, Balart LA, Radvan G, Zweiban BE, Larson AW, Kao HW, Yamada S. A prospective, randomized controlled trial of chronic esophageal variceal sclerotherapy. *Hepatology* 1985; 5: 584—589.
23. Soderlund C, Ihre T. Endoscopic sclerotherapy v. conservative management of bleeding oesophageal varices. A 5-year prospective controlled trial of emergency and long-term treatment. *Acta Chir Scand* 1985; 151: 449—456.
24. Cello JP, Grendell JH, Crass RA, Weber TE, Trunkey DD. Endoscopic sclerotherapy versus portacaval shunt in patients with severe cirrhosis and acute variceal hemorrhage: Long-term follow-up. *New Engl J Med* 1987; 316: 11—15.
25. Warren WD, Henderson JM, Millikan WJ, Galambos JT, Brooks WS, Riepe SP, Salam AA, Kutner MH. Distal splenorenal shunt versus endoscopic sclerotherapy for long-term management of variceal bleeding. Preliminary report of a prospective, randomized trial. *Ann Surg* 1986; 203: 454—462.
26. Rikkers LF, Burnett DA, Volentine GD, Buchi KN, Cormier RA. Shunt surgery versus endoscopic sclerotherapy for long-term treatment of variceal bleeding. Early results of a randomized trial. *Ann Surg* 1987; 206: 261—271.
27. Teres J, Bordas JM, Bravo D, Visa J, Grande L, Garcia-Valdecasas JC, Pera C, Rodes J. Sclerotherapy vs. distal splenorenal shunt in the elective treatment of variceal hemorrhage: A randomized controlled trial. *Hepatology* 1987; 7: 430—436.
28. Luketic BA, Sanyal AJ. Esophageal varices II. TIPS (Transjugular Intrahepatic Portosystemic Shunt) and surgical therapy. *Gastroenterol Clin North Am*. 2000; 29: 387—421.
29. Sanyal AJ, Freedman AM, Luketic VA, Purdum PP, Shiffman ML, Tisando J, Cole PE. Transjugular portosystemic shunts for patients with active variceal hemorrhage unresponsive to sclerotherapy. *Gastroenterol*. 1996; 111: 138—146.

30. **Cappell MS.** High risk gastrointestinal bleeding, part II. Gastroenterol. Clin North Am. 2000; 29: 275–557.
31. **Greenburg AG, Saik RP, Bell RH, Collins GM.** Changing patterns of gastrointestinal bleeding. Arch Surg 1985; 120: 341–344.
32. **Gourdin TG, Smith BF, Craven DE.** Prevention of stress bleeding in critical care patients: Current concepts on risk and benefit. Perspect Crit Care 1989; 2: 44–73.
33. **Cook DJ, Fuller HD, Guyatt GH, Marshall JC, Leasa D, Hall R, Winton TL, Rutledge F, Todd TJ, Roy P.** Risk factors for gastrointestinal bleeding in critically ill patients. Canadian Critical Care Trials Group. New Engl J Med 1994; 330: 337–381.
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34. **Cook D, Guyatt G, Marshall J, Leasa D, Fuller H, Hall R, Peters S, Rutledge R, Griffith L, McLellan A, Wood G, Kirby A.** A comparison of sucralfate and ranitidine for the prevention of upper gastrointestinal bleeding in patients requiring mechanical ventilation. Canadian Critical Care Trials Group. New Engl J Med 1998; 338: 791–797.
35. **Lu WY, Rhoney DH, Boling WB, Johnson JD, Smith TC.** A review of stress ulcer prophylaxis in the neurosurgical intensive care unit. Neurosurgery 1997; 41: 416–426.
36. **Ramsey G, van Saene RHKF.** Selective gut decontamination in intensive care and surgical practice: Where are we? World J Surg 1998; 22: 164–170.
37. **Cook DJ, Guyatt GH, Salena BJ, Laine LA.** Endoscopic therapy for acute nonvariceal upper gastrointestinal hemorrhage: A meta-analysis. Gastroenterology 1992; 102: 139–148.
38. **Lin HJ, Lo WC, Lee FY, Perng CL, Tseng GY.** A prospective randomized comparative trial showing that omeprazole prevents rebleeding in patients with bleeding peptic ulcer after successful endoscopic therapy. Arch Intern Med 1998; 158: 54–58.
39. **Lau JY, Sung JJ, Lee KK, Yung MY, Wong SK, Wu JC, Chan FK, Ng EK, You JH, Lee CW, Chan AC, Chung SC.** Effect of intravenous omeprazole on recurrent bleeding after endoscopic treatment of bleeding peptic ulcers. New Engl J Med 2000; 343: 310–316.
40. **Herrington JL, Davidson J III.** Bleeding gastroduodenal ulcers: Choice of operations. World J Surg 1987; 11: 304–314.
41. **Dickstein G, Boley SJ.** Severe lower intestinal bleeding in the elderly. In: Najarian JS, Delaney JP, eds. Progress in Gastrointestinal Surgery. Chicago: Year Book, 1989: 525–542.
42. **Farrands PA, Taylor I.** Management of acute lower gastrointestinal haemorrhage in a surgical unit over a 4-year period. J Royal Soc Med 1987; 80: 79–82.
43. **Vernava AM III, Moore BA, Longo WE, Johnson FE.** Lower gastrointestinal bleeding. Dis Colon Rectum 1997; 40: 846–858.
44. **Jensen DM, Machicado GA, Jutabha R, Kovacs TOG.** Urgent colonoscopy for the diagnosis and treatment of severe diverticular hemorrhage. New Engl J Med 2000; 342: 78–82.
45. **Winzelberg GG, Froelich JW, McKusick KA, Strauss HW.** Scintigraphic detection of gastrointestinal bleeding: A review of current methods. Am J Gastroenterol 1983; 78: 324–327.
46. **Nusbaum M, Baum S.** Radiographic demonstration of unknown sites of gastrointestinal bleeding. Surg Forum 1963; 14: 374–375.

Received May 27, 2009.

Accepted January 17, 2010.