

Upper Gastrointestinal Bleeding in Kuala Lumpur Hospital, Malaysia

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Summary

Despite advancements in endoscopy and pharmacology in the treatment of peptic ulcer disease the overall mortality has remained constant at 10% for the past four decades. The aim of this study was to determine the age, gender, racial distribution, incidence and causes of endoscopically diagnosed cases of upper gastrointestinal (UGI) bleeding to summarise treatments undertaken and to report their outcome. A prospective study of UGI bleeding in 128 patients was performed in two surgical wards of Kuala Lumpur Hospital, involving both elective and emergency admissions. The study group comprised of 113 (88.2%) males and 15 (11.7%) females. The mean age was 51.9 years (range 14 to 85 years) and 37.5% (48 of 128 patients) were older than 60 years. The Indian race was over-represented in all disease categories. Smoking (50.1%), alcohol consumption (37.5%), non-steroidal anti-inflammatory drugs (NSAIDs) (17.2%), traditional remedies (5.5%), anti-coagulants (2.3%) and steroids (0.8%) were among the risk factors reported. Common presenting symptoms and signs included malaena (68.8%), haematemesis (59.4%) and fresh per rectal bleeding (33.6%). The commonest causes of UGI bleeding were duodenal ulcer (32%), gastric ulcer (29.7%), erosions (duodenal and gastric) (21.9%), oesophageal varices (10.9%) and malignancy (3.9%). UGI bleeding was treated non-surgically in 90.6% of cases. Blood transfusions were required in 62.6% (67/107) of peptic ulcer disease patients. Surgical intervention for bleeding peptic ulcer occurred in around 10% of cases and involved under-running of the bleeding vessel in most high risk duodenal and gastric ulcer patients. The overall mortality from bleeding peptic ulcer disease was 4.7%. Six patients died from torrential UGI haemorrhage soon after presentation, without the establishment of a cause. Active resuscitative protocols, early endoscopy, more aggressive interventional therapy, early surgery by more senior surgeons, increasing intensive care unit beds and more active participation of multidisciplinary teams in co-ordinating management are among remedial measures advocated. Broader educational preventive strategies should target the causes of UGI bleeding.

Key Words: Bleeding, Ulcers, Varices, NSAIDS, Alcohol, Smoking

Introduction

Upper gastrointestinal (UGI) bleeding is a common cause of morbidity and mortality among urban dwellers in Malaysia. Despite advancements in endoscopy and pharmacology in the treatment of peptic ulcer disease, the most frequent cause of such bleeding, the overall

mortality has remained constant at 10% for the past four decades¹⁻⁵. This is due to an increase in the proportion of elderly patients developing complications of peptic ulcer disease⁶. As such, early diagnosis, systematic management and regular follow-up via endoscopy are vital.

The objectives of this study were to determine the age, gender, racial distribution, incidence and causes of endoscopically diagnosed cases of upper gastrointestinal (UGI) bleeding, to summarise treatments undertaken and to report the outcome. From this information, remedial measures to reduce morbidity and mortality have been formulated.

Materials and Methods

A prospective study of UGI bleeding involving 128 patients was performed in two surgical wards of the government unit, Kuala Lumpur Hospital between January 1995 to December 1995. All cases aged 12 years and above admitted for haematemesis or malaena secondary to an upper gastrointestinal cause, that is, proximal to the duodeno-jejunal flexure, were included in the study. All patients underwent oesophagogastroduodenoscopy (OGDS). Elective and emergency cases were included. A further six patients who died from torrential UGI bleeding soon after presentation, without an endoscopically established cause, were considered separately.

Peptic ulcer disease was defined as inflammation, erosions and/or ulcerations of the oesophageal, gastric and/or duodenal mucosa. Endoscopic biopsy was necessary in some cases to confirm diagnosis.

The amount of blood transfused was calculated from the total amount received by each patient during his or her entire hospital stay. This included blood provided as

part of resuscitative measures, and that provided to patients who, already in hospital for other reasons, developed an UGI bleed.

The indications for emergency surgery for bleeding peptic ulcers were failure to control bleeding by conservative management, presence of major endoscopic stigmata of recent haemorrhage and rebleeding. Endoscopic stigmata of recent haemorrhage include an actively spurting vessel, oozing haemorrhage, overlying fresh clot, black clot and a visible vessel in the ulcer base.

Results

1) Patient Profile

This study of 128 consecutive admissions for UGI bleeding comprised of 113 (88.3%) males and 15 (11.7%) females. Six further patients died from severe haemorrhage soon after presentation without the establishment of a diagnosis; the following results relate to the 128 UGI bleeds of known aetiology. The mean age was 51.9 years (range 14 to 85 years) and 48 patients (37.5%) were more than 60 years of age. Of all admissions, 81 (63%) were primary presentations of UGI bleeding, the remaining 47 (36.7%) having been previously admitted or followed-up for peptic ulcer disease.

Sixty-one patients (49.2%) were Chinese, while Malays and Indians comprised 33 (25.8%) and 30 (23.4%)

Table I
Ethnic Distribution of UGI Bleeding in KLH

Race	Male (n)	Female (n)	Total (n)	Proportion (%)	Population of KL 1987 (%) ⁺	D/P Index*
Chinese	56	5	61	47.7	52.4	0.9
Malay	27	6	33	25.8	35.5	0.7
Indian	26	4	30	23.4	7.3	3.2
Others	4	0	4	3.1	4.8	0.6
Total	113	15	128	100	100	

+ Dept of Statistics, Kuala Lumpur

* Disease Proportion/Population Proportion

Table II
Causes of UGI Bleeding and Admission

Cause	Total Number	Percentage (%)
Duodenal Ulcer	41	32.0
Gastric Ulcer	38	29.7
Erosions	28	21.9
Peptic Ulcer Disease Sub-		
Total	107	83.6
Oesophageal		
Varices	14	10.9
Malignancy	5	3.9
Miscellaneous	2	1.6
Total	128	100

Table III
UGI Bleeding and Race

Cause	Total	Chinese	Malay	Indians	Others
		n (%)	n (%)	n (%)	n (%)
Duodenal Ulcer	41	23 (56.1)	10 (24.4)	7 (17.1)	1 (2.4)
Gastric Ulcer	38	20 (52.6)	11 (28.9)	5 (13.2)	2 (5.3)
Erosions	28	14 (50)	6 (21.4)	7 (25.0)	1 (3.6)
Oesophageal					
Varices	14	2 (14.3)	3 (21.4)	9 (64.2)	-
Others	7	2 (28.6)	3 (42.9)	2 (28.6)	-

patients respectively, the latter group being over represented relative to their contribution to the total population of Kuala Lumpur. See Table I.

Presenting symptoms included malaena in 88 patients (68.8%), haematemesis in 76 patients (59.4%) and fresh rectal bleeding in 43 (33.6%).

A history of alcohol consumption was present in 48 cases (37.5%). Sixty-five patients (50.1%) were smokers. Thirty-one (47.7%) of the smokers were Chinese in comparison to 20 (30.8%) and 14 (21.5%) Malay and Indian patients, respectively. It is of note that none of the 15 female patients in this study admitted to smoking or the regular consumption of alcohol. Apart from smoking, the breakdown of risk factors according

to racial group was not recorded.

Thirty-nine patients (30.5%) had been on various drugs prior to admission, either modern drugs or traditional remedies. Specifically, non-steroidal anti-inflammatory drugs had been used by 22 of the 128 admissions (17.2%). Seven patients (5.5%) had used traditional remedies while 9 patients (7%) were taking anti-ulcer drugs prescribed by a doctor. Anticoagulants were in use in 3 patients (2.3%) and prednisolone in 1 patient (0.8%).

The results and racial predisposition are summarised in Table II and Table III.

All patients with oesophageal varices, but only 50 - 70% of those with peptic ulcer disease required blood transfusion. (Table IV).

2) Treatment

a) Non-Operative Treatment

Upper gastrointestinal tract bleeding was managed without operation in 116 of the 128 admissions (90.6%). Initial therapy consisted of resuscitation, including restoration of fluid and blood volume and normalisation of vital signs with crystalloids, colloids and blood transfusion, as required. A central venous line and nasogastric tube was inserted if necessary.

All patients admitted underwent endoscopy either as an emergency (18.8%), during the next scheduled list (54.7%) or electively (26.6%). The indications for endoscopic treatment of non-variceal lesions were bleeding duodenal ulcers ($n = 2$) and gastric ulcers ($n = 4$). Endoscopic treatment consisted of either adrenaline injection sclerotherapy or diathermy heat probe treatment. None of these cases required further surgery.

Of those 14 patients who had oesophageal varices, 3 underwent banding (21.4%) and 8 sclerotherapy (57.1%). Seven patients (50%) had a Sengstaken-Blakemore tube inserted before or after their endoscopy to control severe haemorrhage.

Table IV
Transfusions by Diagnostic Group

Cause	No. of Units Transfused			Total requiring transfusion
	< 2	2 - 5	> 5	
n (%)	n (%)	n (%)		
Duodenal Ulcer	13 (31.7)	5 (12.2)	9 (22.0)	27 (65.9)
Gastric Ulcer	9 (23.7)	9 (23.7)	8 (21.1)	26 (68.4)
Erosions	5 (17.9)	5 (17.9)	4 (14.3)	14 (50.0)
Oesophageal Varices	1 (7.1)	6 (42.9)	7 (50.0)	14 (100.0)

Table V
Mortality

Cause of Bleeding	Total Mortality Due To UGI Bleeding	
		N (%)
Duodenal Ulcer	2	(4.9)
Gastric Ulcer	3	(7.9)
Oesophageal Varices	6	(42.9)
Total deaths	11	(8.6)

Note that six further patients died from torrential UGI haemorrhage soon after presentation, without the establishment of a cause.

b) Operative Management

Twelve patients underwent surgery. The endoscopically proven causes of bleeding in these patients were duodenal ulcer ($n=4$), gastric ulcer ($n=5$), oesophageal varices ($n=2$) and gastric cancer ($n=1$). Of duodenal ulcer and gastric ulcer patients, 9.8% (4 of 41) and 13.2% (5 of 38) went to operation, respectively. Of the peptic ulcer patients who had surgery, comprising 5 Chinese, 3 Indians and 1 Malay, 5 underwent suture ligation of the bleeding point, 3 had a partial gastrectomy [Billroth I ($n=1$), Billroth II ($n=2$)] and one had an excision of their gastric ulcer. Partial gastrectomy was required for one patient with gastric adenocarcinoma. Oesophageal transection with reanastomosis, splenectomy and devascularisation procedures were performed in the two cases of oesophageal varices operated.

Major post-operative complications included recurrence of ulcer bleeding ($n=1$), myocardial infarction ($n=2$), lobar pneumonia ($n=1$), septicaemia ($n=3$), intestinal obstruction ($n=1$), renal failure ($n=1$) and wound infection ($n=3$), however, none required re-operation. There were three post-operative deaths from peptic ulcer disease surgery. The respective causes were myocardial infarction, septicaemia with burst abdomen and recurrence of bleeding gastric ulcer. The latter patient was already in the ICU, had multi-organ failure with septicaemia.

c) Mortality Rate

The overall mortality rate for UGI bleeding of known cause was 8.6% (11 of 128), and a further six patients died from UGI bleeding before the establishment of a diagnosis (Table V). The operative mortality rate for peptic ulcer disease was 25% (3 of 12). The overall mortality rate in peptic ulcer disease patients was 4.7% (5 of 107). In this group, UGI bleeding primarily caused death and the ultimate mechanisms were myocardial infarction, exsanguination and septicaemia with burst abdomen, multiorgan failure and exsanguination with cerebrovascular accident. The latter patient refused to provide consent for surgery before his condition worsened.

Discussion

Upper gastrointestinal haemorrhage was more common in the older age group of 60 years and above (37.5%) although the mean age was 51.9 years. Since over 80% of all bleeds were due to peptic ulcer disease, it is worthy of note that this age trend may be consistent with the increasing incidence of Helicobacter pylori infection known to occur with age in several countries. It may also be due to increased use of NSAID in this group. This organism is now recognised as the cause of peptic ulcers in at least 90% of patients, few of whom are aware of this association⁷. The routine testing for, and eradication of H. pylori for patients with peptic ulcer disease is now the foundation of medical treatment for this condition⁸. Prevalence data for H. pylori were not collected for this study, as testing was not routinely practiced during the study period, however, this information has been the focus of other studies conducted in Malaysia⁹.

Bleeding was much more common in males than females (ratio 9:1). Among the factors explaining this is the observation of the relative infrequency of smoking and alcohol abuse among Malaysian women. For instance, in a survey of Malaysian secondary school teachers, only six of the 766 regular and occasional smokers were female¹⁰. In other published series from the West the gender difference for UGI bleeding has been much smaller, with male to female ratios in the order of 2.4:1^{4,11}.

Although the total number of patients diagnosed with peptic ulcer disease over the period of this study was not recorded, the fact that primary presentations of UGI bleeding from peptic ulcer disease made such a large contribution to the total number of UGI bleeds stresses the importance of the early diagnosis and management of this condition. Such an observation has been made in a study conducted in Kelantan, where 45% of peptic ulcer disease patients presented primarily with acute UGI bleeding¹².

Both Chinese and Malays who presented with UGI bleeding did so in numbers proportional to their relative contribution to the population of Kuala Lumpur. Indians were disproportionately highly represented in every disease category and in the overall results (Table I). Explanation of this discrepancy may relate to a true susceptibility to upper gastrointestinal pathology in this group, their poor socio-economic and educational status, and their tendency to present at a later stage with more severe disease manifestations. Other contributing factors include the choice of alternative therapies, delaying initial presentation, and the presence of a large number of Indian people in the Sentul area close to the hospital. Admittedly, the numbers involved are not large enough to make any definitive explanations, and the use of the population of Kuala Lumpur as representative of the catchment of the hospital is by no means as accurate as the use of general medical patient data for comparison, as employed by others¹³. Nevertheless, the trend towards disproportionately high numbers of Indian patients with gastrointestinal pathology is not unique to this study^{9,14}.

Even with the effective medical treatment regimes currently available, smoking remains a large obstacle to control of peptic ulcer disease, and many other

conditions. Smoking increases the risk of peptic ulcer disease and death, delays healing with or without specific treatment and increases ulcer recurrence rates¹⁵. Lamentably, as yet, there are no nationwide data on smoking prevalence in Malaysia.

The other major predisposing factor to UGI bleeding apart from alcohol and smoking was drug intake. NSAIDs, traditional drug therapy, anticoagulants and steroids were in use by patients in this study. NSAIDs and aspirin are being increasingly employed in the management of arthritis, cerebrovascular disease and coronary artery disease.

In general, patients are poorly informed of the side effects of these drugs, and strategies available to reduce these. NSAIDs should ideally be taken after food or simultaneously with an H₂-antagonist or proton pump inhibitor so as to protect the stomach mucosa from the complications of hyperacidity. It is recommended that all patients above 60 of age should be prescribed an H₂-antagonist or proton pump inhibitor with any drug known to predispose to peptic ulcer disease. The use of traditional remedies, although associated with only 7% of UGI bleeds in this study, is still of significance since this number represents a small proportion of the many patients who rely solely on alternative care.

Intercurrent medical illness such as ischaemic heart disease (7.8%), diabetes mellitus (8.6%) and hypertension (11.7%) were among the remaining predisposing factors for ulcer formation. The number of associated medical conditions is directly related to the increasing risk of mortality. Mortality of patients with four or more associated illnesses can be as high as 70%. The optimal management of these conditions should be seen as integral to the treatment of peptic ulceration.

Peptic ulcer disease was the most common cause of UGI bleeding, being responsible in 83.6% of patients. Other studies have reported incidences of bleeding peptic ulcer ranging from 35% to 55%^{4,6,8,16,17}.

Nine out of 14 (64.3%) patients in this study with oesophageal variceal bleeding were Indian; the high incidence of alcohol-associated chronic liver disease among Indians has been documented¹⁸. As might be

suspected, many patients with oesophageal varices with liver cirrhosis have associated erosions, gastric or duodenal ulcers. The significance of oesophageal varices cannot be overstated: whilst a less common cause of bleeding, 42.9% of this group of patients did not survive. (See Table V). It was clear that oesophageal varices were a cause of more severe UGI bleeding relative to peptic ulcer disease. All of the oesophageal varices patients required blood transfusion and interventional therapy (Sengstaken-Blakemore tube, banding or sclerotherapy). This was in contrast to patients with peptic ulcer where only about 60% required blood transfusion and 5.6% (6 of 108) interventional therapy (saline or adrenaline injection sclerotherapy and diathermy heat probe).

Conventionally, the bleeding duodenal ulcer is treated by under-running followed by truncal vagotomy with pyloroplasty (or gastro-jejunostomy if a pyloroplasty cannot be performed safely). Bleeding gastric ulcer has for many years been taken as an indication for partial gastrectomy¹⁹.

In the current study, the operative rate for bleeding peptic ulcers was 8.4%. Under-running of the bleeding vessel was the operation of choice for the high-risk patients whereby haemostasis was achieved by under-running of the bleeding vessel without performing any ulcer-healing procedure.

About 80-90% of episodes of UGI bleeding from peptic ulcer disease spontaneous ceased with conservative management as with most other studies^{8,17,20}. For these patients the hospital course is relatively smooth, with a mortality rate of 4% or less²⁵. In patients with severe persistent bleeding, however, the mortality rate can be as high as 30-40% and emergency surgical intervention is usually required²¹. Other groups have reported operative rates of 10% to 40% with most surgery done as an emergency procedure^{2,4,22,23}. In this series, only 12 patients (around 10%) received an operation, principally due to a high threshold for surgical intervention.

The mortality rate in the oesophageal varices group was higher (42.9%) than the peptic ulcer disease group (4.7%). Largely as a result of the inability to perform emergency endoscopy due to torrential haemorrhage at presentation, six deaths occurred without any definitive

diagnosis being made (Table V). One study of bleeding peptic ulcers has described a mortality of 6-10%, with most deaths caused by cardiovascular complications associated with impaired tissue perfusion following haemorrhage, rather than by exsanguination. Mortality is substantially increased in older patients requiring transfusion with more than 4 units of blood, because of poor cardiac reserve and a reduced ability to cope with rapid blood volume changes²⁴.

Of the factors affecting prognosis rebleeding is the most important, and may increase the risk of death up to 12 times. Factors such as age over 60 years, shock on admission, and concurrent disease (especially cardio-respiratory problems and pre-existing anaemia) are each associated with increased risk of rebleeding^{25,26,27}. Hunt reported that shock on admission to the hospital was associated with a significantly greater incidence of rebleeding in 70 cases^{26,27}. Endoscopic stigmata of recent haemorrhage (ESRH) are the most important factors in predicting rebleeding^{28,29,30}. The identification of ESRH triples the risk of mortality²⁵.

Factors which complicated management, and which require amelioration, were poor early resuscitation at the casualty level, lack of intensive care unit beds for post-operative care of critically ill patients, unavailability of OGDS facilities during an emergency, poor assessment criteria for surgery, unavailability of operative time and refusal for consent for surgery from patients or relatives despite detailed explanation of the circumstances.

Conclusion and Recommendations

It is important to realise that gastrointestinal bleeding is a difficult and challenging problem worthy of prompt attention in any hospital. With an increasing population of elderly patients, rapid access to endoscopy, and frequently the operating theatre, is essential. The management of acute gastrointestinal haemorrhage requires a multidisciplinary approach, which involves a team of medical staff including a senior surgeon, anaesthetist, physician, and nurses. The combination of adequate resuscitation, close monitoring, early endoscopy surgery and good communication with and support from the hospital blood bank is essential.

Improvements in management could be achieved by the early initiation of more active resuscitative protocols including consideration of central venous line insertion, at the casualty or ward level. This should be followed by early OGDS (within 12 hours) and more aggressive interventional therapy following stabilisation of the bleeding patient. All patients with arterial bleeding from ulcers and high risk patients with visible vessels should be considered for endoscopic therapy; low risk patients with a visible vessel can probably be managed expectantly and treated if bleeding recurs^{19,31}. Endoscopic therapy should not be attempted if there is torrential haemorrhage, if there is doubt about the exact site of the bleeding point or if limited access, for any reason, prevents optimal treatment³¹. There should be an improvement in the emergency OGDS services provided as only 18.8% of OGDS were performed as an emergency. Easy access to operating theatres should be

provided for bleeding emergencies, and the threshold for operation lowered. More intensive care and high dependency unit beds should be available to provide optimal support for critically ill patients (more than 60 years old, clinical shock, consistent medical illness and/or evidence of active bleeding).

On a broader scale, of great importance is education via the mass media, doctors, nurses, and other paramedical staff, as a prevention tool to combat the causes of UGI bleeding, for example, confronting the scourge of smoking and alcohol abuse, particularly among young people, the correct use of NSAIDs, and the promotion of healthy eating habits and awareness of the role of *H. pylori* in peptic ulcer disease. Early intervention has great potential to reduce the number of patients suffering from UGI bleeding as a complication of their disease.

References

1. Avery-Jones F. Haematemesis and melena with special reference to causation and to the factors influencing the mortality from bleeding peptic ulcers. *Gasteroenterology* 1956; 30: 1661- 190.
2. Johnston SJ, Jones PF, Kyle J. Epidemiology and course of gastrointestinal haemorrhage in northeast Scotland. *Br. Med J* 1973; 3: 655-60.
3. Allan R, Dykes. A study of the factors influencing mortality rates from gastrointestinal haemorrhage. *J Med* 1976; 45: 533-50.
4. Silverstein FE, Feld AD, Golbert DA. Upper gastrointestinal tract bleeding. *Arch Intern Med* 1981; 141: 538-43.
5. Fleischer D. Etiology and prevalence of severe persistent upper gastrointestinal bleeding. *Gasteroenterology* 1983; 84: 538-43.
6. Sugawa L. Endoscopic diagnosis and treatment of upper gastrointestinal bleeding. *Surg Clin of North Am* 1989; 69: 1167-183.
7. Knowledge about causes of peptic ulcer disease - United States, March-April 1997. *MMWR Morb Mort Wkly Rep* 1997; 46(42): 985-87.
8. European Helicobacter Pylori Study Group. Current European concepts in the management of Helicobacter pylori infection. The Maastricht Consensus Report. *Gut* 1997; 41(1): 8-13.
9. Goh KL. Prevalence of and risk factors for Helicobacter pylori infection in a multi-racial dyspeptic Malaysian population undergoing endoscopy. *J Gastroenterol Hepatol* 1997; 12(6): S29-35.
10. Yacoub I, Harun MH. Smoking habits and attitudes among secondary school teachers. *Southeast Asian J Trop Med Public Health* 1994 Mar; 25(1): 74-79.
11. Phillip J, Clausen M, Gunselman. Emergency endoscopy study in abstracts of the IV European congress of gastrointestinal endoscopy. Stuttgart, Georg Thieme Verlag 1980.

12. Raj SM. Peptic ulcer disease in Kelantan: an underdiagnosed condition? *Med J Malaya* 1991; 46(2): 183-6.
13. Kudva MV, Thein-Htut. Profile of Peptic Ulcer Disease in Malaysia. *Sing Med J* 1988; 29: 544-47.
14. Chow TK, Lambert JR, Wahlqvist ML, Hsu-Hage BH. Helicobacter pylori in Melbourne Chinese immigrants: evidence for oral-oral transmission via chopsticks. *J Gastroenterol Hepatol* 1995; 10(5): 562-69.
15. Ashley MJ. Smoking and diseases of the gastrointestinal system: an epidemiological review with special reference to sex differences. *Can J Gastroenterol* 1997; 11(4): 345-52.
16. Holman RAE, Davis M, Gough KR. Value of centralised approach in the management of haematemesis and melena: experience in a district general hospital. *Gut* 1990; 31:504-08.
17. Himal HS, Perrault C, Mzabi R. Upper gastrointestinal haemorrhage: Aggressive management decreases mortality. *Surgery* 1978; 4: 448-51.
18. Kudva MV, Zawawi MM. Chronic liver disease in Kuala Lumpur, Malaysia: a clinical study. *Singapore Med J* 1990; 31(4): 368-73.
19. Steele RJC. Non-variceal gastrointestinal haemorrhage. Recent advances in surgery 1993; 16: 87-103V.
20. Larson DE et al. Upper gastrointestinal haemorrhage. *Mayo Clin Pro* 1983; 58: 371-87.
21. MacLleod IA, Mills PR. Factors identifying the probability of further haemorrhage after acute upper gastrointestinal haemorrhage. *Br J. Surg.* 1982; 69: 256-58.
22. Sugawa C, Steffes CP, Nakamura R. Upper gastrointestinal bleeding in an urban hospital: etiology, recurrence and prognosis. *Ann Surgery* 1990; 212: 521-26.
23. Mayberry JF, Counsel BR, Penny WJ. Mortality in acute upper gastrointestinal haemorrhage: a six-year survey from the University Hospital of Wales. *Postgrad Med J* 1981; 57: 627-32.
24. Morris DL, Hawker PC, Brearley S. Optimal timing of operation for bleeding peptic ulcer: prospective randomised trial. *Br Med J* 1984; 288: 1277-80.
25. Branicki FJ, Coleman, Fox PJ. Bleeding peptic ulcer: A prospective evaluation of risk factors for rebleeding and mortality. *World J Surg* 1990; 14: 262-70.
26. Hunt PS. Bleeding gastroduodenal ulcers: selection of patients for surgery, *World J Surg* 1987; 11: 289.
27. Dronfield MW et al. Outcome of endoscopy and barium radiotherapy for acute upper gastrointestinal bleeding: controlled trial in 1037 patients. *Br Med J* 1982; 284: 545-5489.
28. Fullarton GM, Murray WR. Prediction of rebleeding in peptic ulcers by visual stigmata and endoscopic Doppler ultrasound criteria. *Endoscopy* 1990; 22: 68-71.
29. Storey DW, Bown SG, Swain CP. Endoscopic prediction of recurrent bleeding in peptic ulcers. *N Engl J Med* 1981; 305: 915-16.
30. Griffiths WJ, Neumann DA, Welsh JD. The visible vessel as an indicator of a controlled or recurrent gastrointestinal haemorrhage. *N Engl J Med* 1979; 300: 1411.
31. Lawrence BH, Cotton PB. Bleeding gastroduodenal ulcers: nonoperative treatment. *World J Surg* 1987; 11: 295-303.