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A Comparative study of various causes of mortality in acute upper gastrointestinal bleed patients in the goan population - A five-year retrospective study at a tertiary care centre

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ABSTRACT

Introduction: In Goa, physicians and surgeons frequently deal with a life-threatening emergency known as acute upper gastrointestinal (GI) bleed. It is defined as blood loss from a gastrointestinal source above the ligament of Treitz, which may present as hematemesis, melaena, or blood loss symptoms such exhaustion, weakness, and syncopal attacks.

Aims: To study the various causes of mortality of the patients with acute upper GI bleed and to identify the risk factors and demographic factors of upper GI bleed in patients.

Materials and Methods: This was a retrospective study conducted from January 2018 to January 2023 over a five-year period at Goa Medical college and hospital. Patients admitted with hematemesis and melaena, satisfying the inclusion criteria. The clinical profile, risk factors and demographic profile were noted.

Results: A total of 485 patients were studied during those periods. The most common cause of mortality was bleeding oesophageal varices secondary to alcoholic liver disease (71%). the mean age was 55 years and were predominantly males (94%). Demographically it was noted that mortality was highest in patients from Bardez Taluka.

Conclusion: According to our study, bleeding oesophageal varices were the leading cause of mortality. A common medical emergency, UGI bleeding has a high risk of mortality and morbidity as well as hefty medical expenses. Our research gives us a better understanding of the numerous causes of death, enables us to pinpoint risk factors and demographic factors, and enables us to lower the costs of healthcare associated with this clinical entity by reducing mortality and morbidity.

Keywords: Upper GI Bleeding; Mortality; Oesophageal Varices; Haematemesis

Abbreviation: UGI Bleed: Upper Gastrointestinal Bleed

Introduction

Acute UGI bleed, which is defined as blood loss from a gastrointestinal source above the ligament of Treitz, is a common but potentially fatal medical emergency [1]. Hematemesis, which can be bright red emesis, coffee

grounds, melaena, or even symptoms related to blood loss such syncopal episodes and weakness, might be an indication of it [1,2]. A significant source of mortality and morbidity, UGI tract bleeding is four times more often than lower gastrointestinal tract bleeding. Everywhere in the world, UGI bleed has a different etiology. In contrast to peptic ulcers in western nations, oesophageal varices are the most typical cause of UGI haemorrhage in Asians [1,2]. Peptic ulcers, esophagitis, drug-induced mucosal damage, vascular abnormalities, traumatic and post-operative lesions, and tumours are the main causes of non-variceal
bleeding. Varices of the oesophagus, stomach, duodenum and portal hypertensive gastropathy are examples of sequelae of portal hypertension that result in variceal UGI bleeding [3]. Early risk assessment, resuscitation, identification, and treatment of the bleeding cause are necessary for patients with UGI bleed. To accomplish haemostasis, endoscopy and endotherapy may be necessary or surgery in situations of severe bleeding [1-3]. Even without therapy, 80% of patients with UGI bleed get self-limited bleeding. Variceal bleeding has a death rate of 20% of those who still bleed or continue to haemorrhage with a 6%-10% total occurrence [3].
UGI bleeds are likely to be seen more in the coming years with an ageing population with increasing use NSAIDS, single on multiple anti-traumatic agents and novel anti coagulants [1-4].

A serious emergency is acute UGI haemorrhage. Significant changes in the frequency, aetiology, and prognosis of individuals with acute UGI bleed occurred over the past 20 years [2]. The discovery of proton pump inhibitors in the late 1980s and H2 receptor antagonists in the middle of the 1970s opened new possibilities for preventing ulcer complications, reducing rebleeding, and promoting ulcer healing [1-5].
Eliminating Helicobacter pylori stops ulcer recurrence because it is a known etiological factor in the onset of peptic ulcer disease [1-5]. The performance of endoscopic therapy and determining the source of bleeding are made easier by endoscopy [2-5]. The treatment of a patient who has a UGI bleed should always be approached in a progressive manner. The first step is to evaluate the patient's hemodynamic status and start resuscitative measures, such as administering fluids and blood transfusions, if necessary [6]. Based on the patient's initial presentation, haemodynamic status, comorbidities, age, and preliminary laboratory tests, risk stratification should be done [3-6]. The most widely used scoring systems are Rockall and Blatchford scores, though there are other options as well [4]. Within 24 hours, upper endoscopy is available to help identify the cause of bleeding and, if necessary, further direct therapy [6].
If it is determined that the bleeding ulcer is the responsible lesion, steps should be taken to stop further bleeding. Eradication should be the goal if H. pylori is discovered in the patient [7]. If bleeding was likely caused by NSAIDS, they should be halted and substitute medications like COX-2 selective NSAIDs plus a PPI should be given instead [1-7]. Patients with a history of cardiovascular disease who need to take aspirin or other antiplatelet medications should be receiving PPI therapy. Antiplatelet therapy can usually be resumed after the bleeding has stopped, ideally within 1-3 days and definately within 7 days [1,3,6].
An interprofessional team composed of an internist, gastroenterologist, surgeon, and emergency department physician diagnoses and treats a UGI bleed. The ATLS protocol should be followed throughout the first stages of CPR. In the natural course of things, 80–90% of patients who receive endoscopic therapy will always be able to manage their bleeding [2][8]. Patients who rebleed should attempt a second endoscopic surgery because 10% - 20% of them will do so. Other modalities like angiography or surgery should be taken into consideration if bleeding does not stop despite endoscopic procedures or if the source of the bleeding cannot be found [3-8].

Aims and Objectives

1. To study the various causes of mortality in patients admitted with acute upper GI bleed in the department of General Surgery at Goa Medical College & Hospital
2. To identify the various risk factors and demographic factors causing upper GI bleed in patients.

Methodology

Study Design: Retrospective study

Study setting – The retrospective will be carried out by obtaining data from the medical records department from January 2018 to January 2023 over a five year period at Goa Medical College &Hospital

Study Population – All patients admitted with acute UGI Bleeding with a reddish Ryle's tube aspirate or melaena on per rectal examination or both admitted under the department of General Surgery at Goa Medical College and Hospital and who expired due to UGI bleed.

Sample Size: All patients with UGI bleed who expired between January 2018- January 2023.

Sample selection method: In this study all patients who have been admitted with acute UGI bleeding with

1. Reddish or bloody Ryle's tube aspirate
2. Melaena on per rectal examination
3. Or both

Inclusion Criteria

All patients admitted as acute UGI bleed under department of General Surgery and who expired due to UGI bleed.

Exclusion Criteria

Patients admitted with acute UGI Beed who have expired due to other causes other than UGI bleed.

Ethical Considerations- Nil

Observation and Results:

Month-Wise Distribution Of Yearly Ugi Bleed Cases					
	2022	**2021**	**2020**	**2019**	**2018**
January	13	5	13	10	5
February	11	6	7	7	7
March	10	5	10	8	5
April	8	5	1	4	6
May	11	8	18	9	11
June	7	7	13	6	3
July	10	9	6	7	5
August	10	5	3	9	7
September	12	4	8	5	11
October	12	4	6	13	5
November	11	5	7	10	9
December	9	11	10	10	13
Total	**124**	**74**	**102**	**98**	**87**

Figure 1: Monthly distribution of Yearly UGI bleeds.

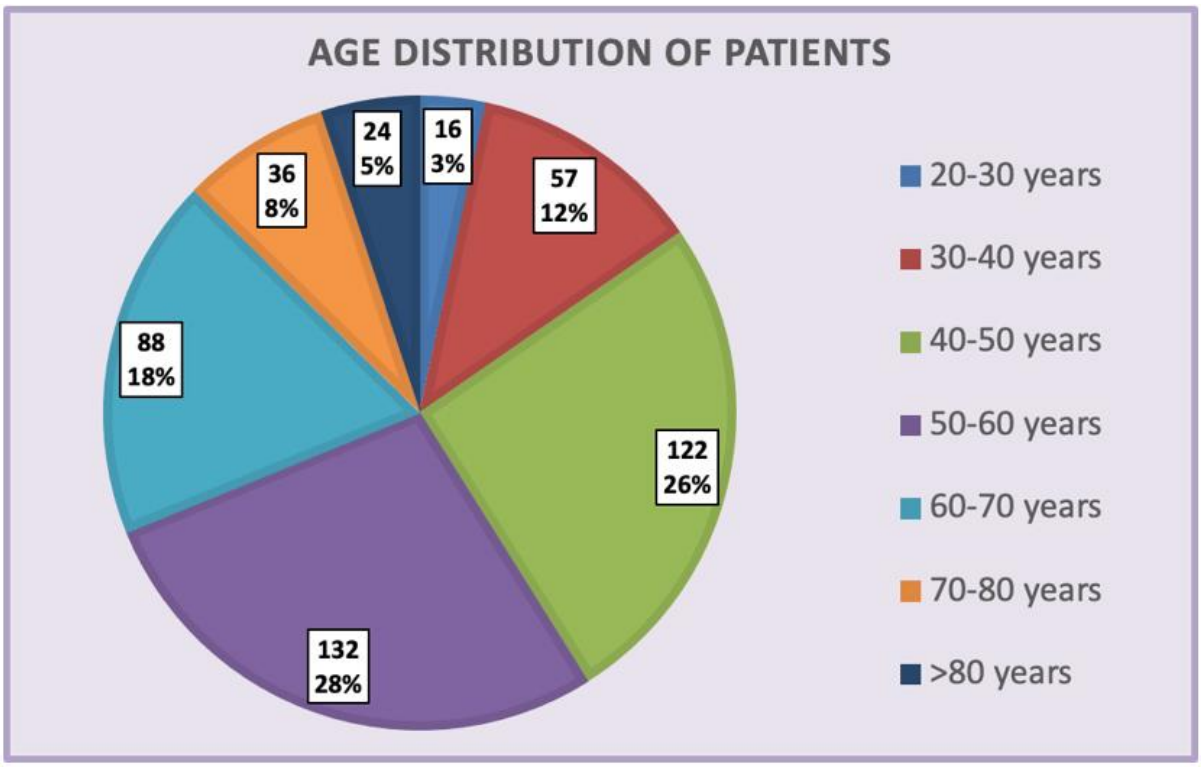

Figure 2: Age wise distribution.

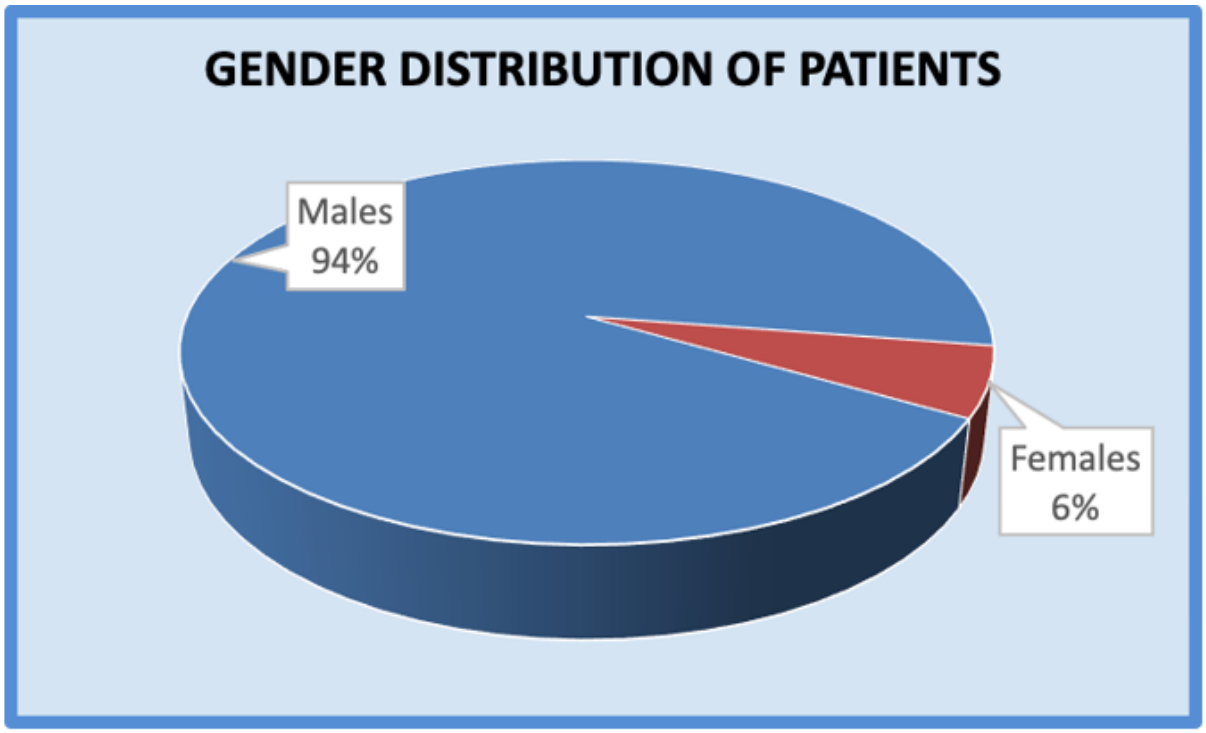

Figure 3: Gender distribution.

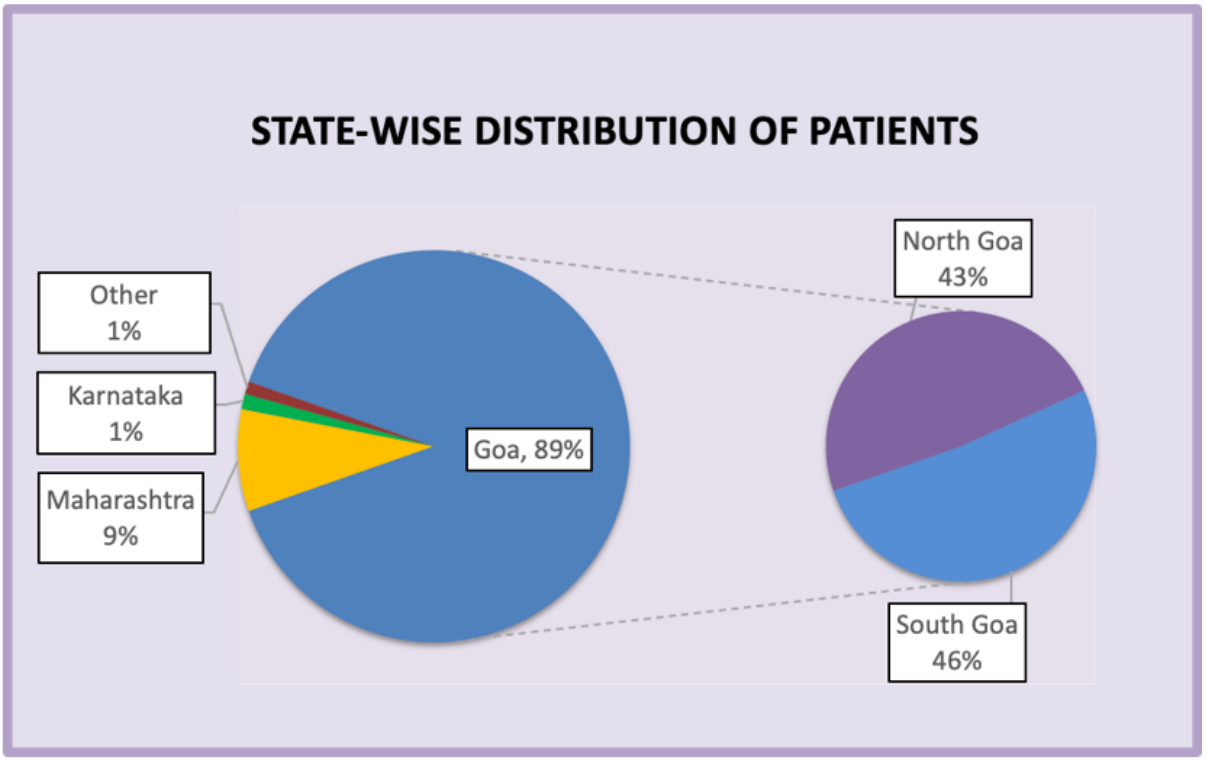

Figure 4: State wise comparison.

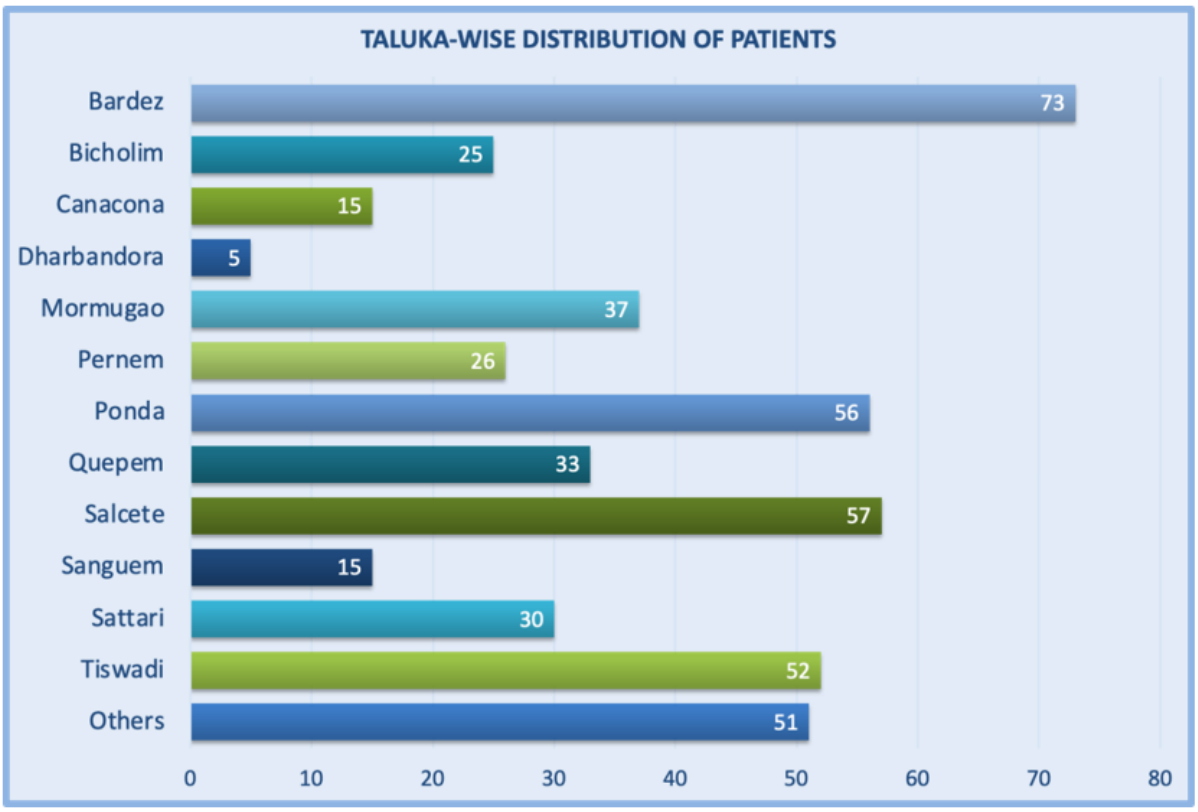

Figure 5: Taluka wise distribution of patients in state of Goa.

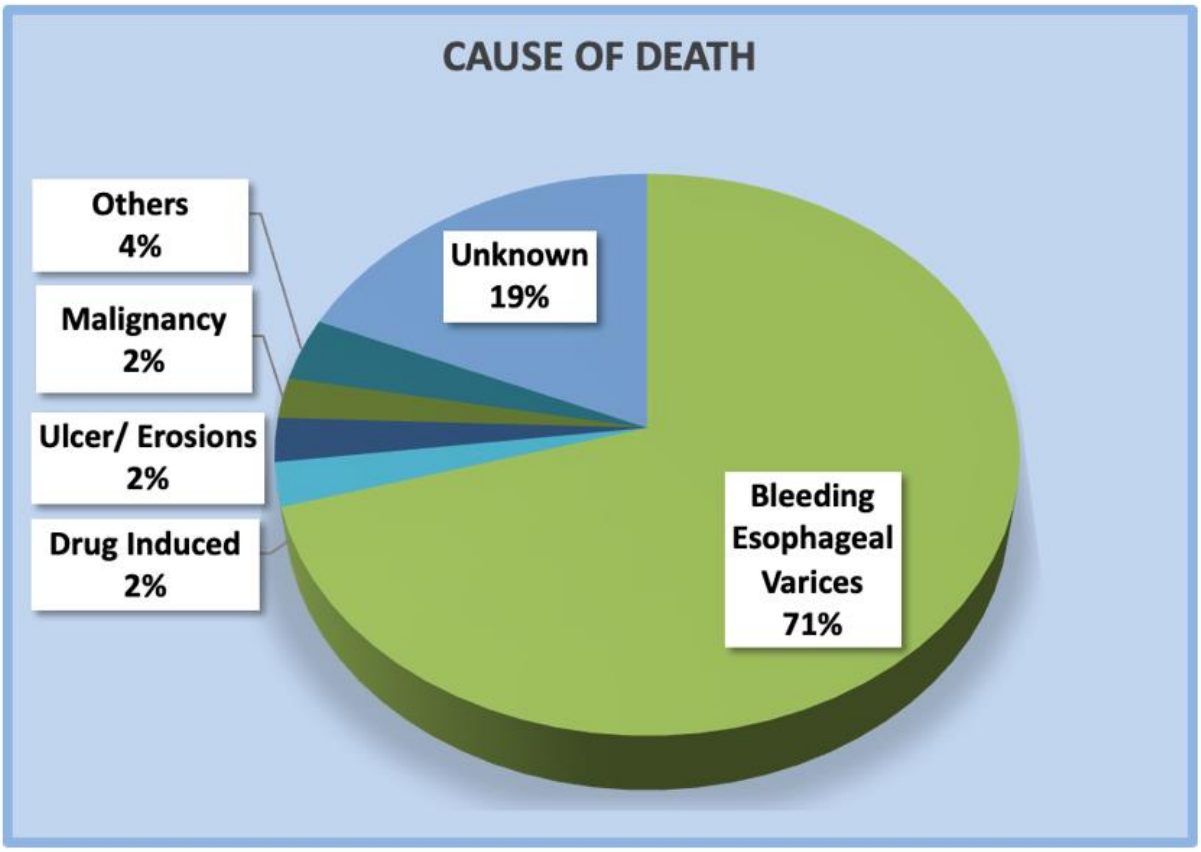

Figure 6: Cause of death in patients with UGI bleed.

Discussion

Upper gastrointestinal bleeding (UGIB) is a common problem that is estimated to occur in 80-150 out of 1,00,000 people each year. Estimated mortality rate are between 2-15 percent [1,2,8]. It is described as blood loss for a gastrointestinal source above the ligament of Treitz. It can manifest as hematemesis, melaena or patients may also present with symptoms secondary to blood loss such as syncopal episodes, fatigue, and weakness [9].

Aetiology

Peptic ulcer disease (PUD) accounts for 40–50% of cases among the potential causes of UGIB. The bulk of these cases (30%) are the result of duodenal ulcers [2-10]. NSAIDS, Helicobacter pylori, and stress-related mucosal illness can all be factors in PUD. In addition to PUD, erosive oesophagitis accounts for 11% of cases, duodenitis for 10%, varices for 5-30%, depending on whether the population under study has chronic liver illness, Mallory–Weiss tears for 5-10%, and vascular malformations for 5% [1,2,11].

Epidemiology

75% of all cases of acute GI bleeding involve UGIB. It affects 80 to 150 people out of every 100,000 people annually. Compared to placebo, patients on long-term low-dose aspirin are more likely to develop overt UGIB [12]. The number of UGIB patients has increased two-fold when compared to P2Y12 inhibitors like clopidogrel. The risk of UGIB is substantially higher when a patient needs triple therapy, which includes aspirin, a P2Y12 inhibitor, and a vitamin K antagonist [13].

History and Physical Examination

Comorbidities should be taken into consideration when taking a patient's history. Patients should be specifically questioned on their usage of NSAIDS, antiplatelets medicines, aspirin, or anticoagulation after a thorough review of their current medications is completed [3,14]. Additionally, it's crucial to obtain a thorough social history of alcohol usage. Although the clinical presentation may differ, it should be well defined [15]. Melaena describes dark, tarry-appearing stools with a distinct scent, whereas haematemesis refers to overt bleeding with vomiting of fresh blood or clots [1,12]. Coffee grounds refers to stomach aspirate or vomitus that has little, black flecks of previous blood in it. The transport of fresh blood through the rectum, or hemotochezia, signifies a lower gastric intestine bleed [2,14]. If the bleeding is significant enough to result in haemodynamic instability, patients may also exhibit syncope or orthostatic hypotension [3]. Additionally, it is important to monitor the patient's vital signs. Additionally, orthostatic vital signs need to be recorded [3]. In a thorough examination, look for signs of chronic liver illness such as ascites, palmar erythema, spider angiomas, gynecomastia, and jaundice as these conditions may provide information about the cause of bleeding, such as varicose veins [3,16].

Evaluation

Initial Laboratory work must include a complete blood count to look for current levels of haemoglobin, haematocrit, and platelets [2,15]. A low MCV can point towards chronic blood loss and iron deficiency anaemia [14]. Biochemistry should also be evaluated elevated BUN or elevated BUN/Creatinine can also be an indicator of UGIB. Coagulation profile should also be checked [1,14].

There are few scoring systems designed to predict which patients, will likely need intervention and to predict rebleeding and mortality [2,16]. The Rockall score was designed to predict re-bleeding and mortality and includes age, comorbidities, presence of shock and endoscopic stigmata [16]. There is also a pre-endoscopic Rockall score that can be used to stratify a patient's risk for mortality and rebleeding even before the endoscopic evaluation [16]. Patients with 2 or fewer points on the Rockall scale are deemed low risk and have a 4.3% chance of rebleeding and a 0.1% fatality rate [2,16]. Patients with a score of 6 or above, in comparison, have a 13% rebleeding rate and a 39% mortality rate.

Another scoring system that is traditionally used in UGIB is the Blatchford score. This scoring system was designed to predict the need for intervention [1,17]. It includes haemoglobin levels, blood pressure, presentation of syncope, melaena, liver diseases and heart failure. A score of 6 or higher is associated with a greater than 50 % risk of needing an intervention [17].

If the patient is suspected of having UGIB, endoscopy must be performed to identify the cause and potentially treat the source of bleeding. Multiple studies have tried to identify the best timing to perform endoscopy [18]. Until now there is no evidence that emergency endoscopy is superior to routine endoscopy (done in 24 to 48 hours). The American College of Gastroenterology continues to recommend that all patients with UGIB should undergo endoscopy within 24 hours of admission, following resuscitative efforts to optimize haemodynamic parameters and other medical problems [2,18]. As per American College of Gastroenterology recommendations endoscopy within 12 hours should be considered for all patients with higher risk clinical features e.g.- tachycardia, hypertension, bloody emesis or Ryle’s tube aspirate in the hospital to potentially improve clinical outcomes [3,16].

Management

Patient must have a minimum of two large-bore peripheral access catheters [1,2]. Intravenous fluid should be administered to maintain adequate blood pressure and haemodynamic stability. Endotracheal intubation is indicated in individuals who are unable to protect their airway or who continue to experience severe haematemesis. Targeting a haematocrit over 20% and a haematocrit above 30% in high-risk patients such the elderly and those with coronary heart disease is recommended for blood transfusions [2,18]. There is no evidence to support the pursuit of greater haematocrit targets, which may potentially be harmful.

Patients with non-variceal UGIB are treated with proton pump inhibitors (PPI). It has been demonstrated that the administration of antacids changes the course of patients with acute UGI bleeding [16]. Patients who are bleeding significantly should be given an 80mg PPI bolus followed by a continuous infusion. For individuals whose endoscopy revealed high-risk lesions, the average time frame is 72 hours [19]. If the endoscopy revealed no abnormalities or only low-risk lesions, the patient may move from the PPI infusion to a twice-daily infusion or even to oral PPIs [2,20]. If variceal bleeding is suspected, vasoactive medications should be started right once, ideally during transport or on admission before endoscopy, and they should be continued for 5 days [2,12]. This is one of the most important procedures to diminish mortality and it achieves haemostasis in 80% of patients [3,13]. Vasoactive medications include terlipressin, octreotide, somatostatin, and vapreotide, with availability varying by country [2,14]. Vasopressin is no longer used for variceal bleeding due to its numerous negative effects. Octreotide is the only medication authorised in the USA for variceal haemorrhage [12,15]. Vasoactive substances reduce portal pressure and reduce or stop variceal bleeding by causing splanchnic vasoconstriction [12]. A synthetic counterpart of vasopressin with a longer half-life and fewer negative effects is terlipressin. In patients with IHD or peripheral vascular disease, it may lead to ischemic consequences and

dysrhythmias similarly to vasopressin [13]. Several studies have shown that terlipressin is effective in bleeding control and it was the only vasoactive drug that diminished mortality in these patients. It is given at a dose of 2mg intravenous every 4 hourly during the first 48 hrs reducing to 1mg every 4 hourly for another 3 days [18]. If the bleeding is controlled somatostatin caused splanchnic vasoconstriction and it inhibits the post prandial increase of portal blood flow and portal pressure. It is given as an initial bolus of 250 mg followed by 250 -500 ug/hr in a continuous infusion [19]. Octreotide is a synthetic analogue of somatostatin with a longer half-life, which is not reflected by longer haemodynamic effects, which may be caused by rapid desensitization or tachyphylaxis. It is administered as an initial bolus of 50 ug followed by a continuous infusion of 50 ug/hr.

Vapreotide is a somatostatin analogue is given as a 50 ug bolus followed by an infusion of 50 ug/hr [3,20]. A Cochrane review of 21 trials involving 2588 patients with active variceal haemorrhage found no difference in mortality rate or risk of rebleeding with somatostatin and its derivatives and a recent study comparing terlipressin, somatostatin and octreotide in the control of acute oesophageal variceal haemorrhage showed no difference in the haemostatic efficacy between these drugs [4,21]. In addition, the same study shown that when these three medications are used in combination with endoscopic treatment, the mortality rate is not statistically different between them [21,22]. As a result, any of these medications may be administered in conjunction with endoscopic therapy to manage oesophageal varices bleeding [13,24].

Antibiotic prophylaxis-For the post two decades it has been widely known that patients with cirrhosis with variceal haemorrhage have a high risk of bacterial infections, which relates to early rebleeding rates and to a high mortality mainly in more decompensated patients with cirrhosis, Child Pugh B and C - however bacterial infections, early

rebleeding and mortality are reduced when patients are given prophylactic antibiotics which are nowadays a part of the standard of care of these patients [3,23]. The recommended antibiotics is norfloxacin in a dose of 400mg orally twice a day or ciprofloxacin 200mg intravenously twice a day if the oral route is not possible [22,24]. In patient with advanced cirrhosis, child -Pugh B or C, ceftriaxone proved to be more effective than oral norfloxacin [25]. A review of 12 trials involving 1241 patients with variceal haemorrhage found that blood spectrum antibiotics (e.g.-ceftriaxone, norfloxacin, ciprofloxacin) reduced overall mortality [2,24,26]. Endoscopic band ligation is preferred as it provides better bleeding and rebleeding control and has fewer adverse events compared to sclerotherapy [1,3,27]. The most frequent complications of band ligation are superficial ulceration, oesophageal stricture, and delayed bleeding after falling of the rubber bands [1,2,28]. Sclerotherapy is used when band ligation is technically difficult or notavailable. Combined therapy (vasoactive drugs and endoscopic therapy) is more effective than other treatment alone, as has been shown by randomized controlled trials and meta-analysis of these trials [29].

Endoscopic intervention might be warranted depending on the findings during the endoscopy. If a patient has an ulcer with a clean base, no intervention is needed [12,30]. However, if a bleeding vessel is visualized or there is a stigma of recent bleeding therapeutic options might include thermal coagulation to achieve haemostasis, local injection of epinephrine or use of clips, a coronation of these methods might be needed based on the seventy of the lesions [2,31].

Conclusion

In Goa, acute UGI bleeding is a frequent life-threatening emergency. Oesophageal varices in patients with alcoholic liver disease-a serious consequence of portal hypertension-were the most frequent cause of mortality in our study. Treatment for these individuals is typically complicated and necessitates a team approach with clearly defined step-by-step management, endoscopic treatments, as well as the use of vasopressors and antibiotics. According to our research, acute UGI bleeding in Goa carries a substantial risk of mortality and morbidity as well as considerable medical costs. Our research gives us a better understanding of the various causes of death and enables us to pinpoint risk factors and demographic factors which helps is to take preventive measures which in turn helps us to lower the costs of healthcare associated with this clinical entity.

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