Prospective Assessment of the Rockall Risk Scoring System in patients with Upper Gastrointestinal haemorrhage

Thesis

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List of Contents

List of Abbreviations

ALT	:	Alanine Transferase
APC	:	Argon Plasma Coagulation
AST	:	Aspartate Transferase
AUROC	:	Area Under ROC Curve
BP	:	Blood Pressure
BUN	:	Blood Urea Nitrogen
CI	:	Confidence Interval
CT	:	Computed Tomography
ERCP	:	Endoscopic Retrograde Cholangio-Pancreatography
EV	:	Esophageal Varices
GAVE	:	Gastric Antral Vascular Ectasia
GBS	:	Glasgow Blatchford Score
GIST	:	Gastro Intestinal Stromal Tumor
GOV	:	Gastro-Oesophegal Varices
GV	:	Gastric Varices
HVPG	:	Hepatic Venous Pressure Gradient
ICU	:	Intensive Care Unit
IGV	:	Isolated Gastric Varices
IHD	:	Ischemic Heart Disease
INR	:	International Normalizing Ratio
MELD	:	Model for End-Stage Liver Disease
MW	:	Mallory –Weiss tear
NSAIDS	:	Non-Steroidal Anti -Inflammatory Diseases
PHG	:	Portal Hypertensive Gastropathy
PT	:	Prothrombin time
PTC	:	Percutaneous Transhepatic Cholangiogram
PTT	:	Partial Thromboplastin Time
PUD	:	Peptic Ulcer Disease.
SRH	:	Stigmata of Recent Bleeding
TIPS	:	Trans Jugular Intrahepatic Portosystemic Shunt
UGIB	:	Upper GastroIntestinal Bleeding
UGIH	:	Upper Gastrointestinal Hemorrhage

List of Tables

List of Figures

Introduction

Upper gastrointestinal (UGI) bleeding is a common disorder affecting over 100 per 100 000 population yearly **(Rollhauser,1997).**

Liver cirrhosis is a major health problem in Egypt, especially that complicating viral hepatitis **(Attia, 1998; El-Zayadi et al., 2005)**. Portal hypertension commonly accompanies the presence of liver cirrhosis. The development of esophageal varices (EV), gastric varices (GV) and portal hypertensive gastropathy (PHG) are the major presentation of portal hypertension **(De Franchis&Primignani, 2001)**.

Bleeding esophageal varices represent one of the most common causes of mortality among patients with chronic liver disease. The incidence of varices in cirrhotic patients is approximately 60-80%. The risk of bleeding may reach 25-35% of all cases within the first year of variceal detection. The mortality from each episode of variceal bleeding is 17-57%**(Jensen, 2002).**

Although endoscopic findings can identify individuals at a high risk of rebleeding, overall mortality is often reflective of other factors such as age and co-morbid conditions. In an effort to risk-stratify subjects with UGI bleeding, numerous scoring systems have been developed to predict bleeding

recurrences, the need for surgical procedures and death**(Blatchfordet al., 2000).**

One scoring system designed for that purpose is the Rockall scoring system **(Rockallet al., 1996 a).** The Rockall system has been shown to represent an accurate and valid predictor of rebleeding and death, performing better in the latter than in the former**(Vreeburget al., 1999).** Rockall scores were designed to combine information such as the subject's age, occurrence of shock assessed from systolic blood pressure readings and pulse rate, presence and severity of co-morbid conditions,diagnosis and endoscopic stigmata of recent bleeding.

Table (1): The Rockall Risk Scoring System

	Score			
Variable	0	1	2	3
Age	<60 yr	60–79 yr	≥80 yr	
Shock	"No shock" Systolic BP >100 pulse <100	"Tachycardia" Systolic BP >100 pulse >100	"Hypotension" Systolic BP <100	
Comorbidity	No major comorbidity		Cardiac failure, IHD, any major comorbidity	Renal failure, liver failure, disseminated malignancy
Diagnosis	Mallory-Weiss tear, no lesion identified, no SRH	All other diagnoses	Malignancy of upper GI tract	
Major SRH	None or dark spot only		Blood in upper GI tract, adherent clot, visible or spurting vessel	

BP = blood pressure; IHD = ischemic heart disease; SRH = stigmata of recent hemorrhage.

(Rockall et al., 1996 a)

Summing up the different levels of a point grading system assigned to each of the components yields a subject's risk score bounded on a scale of 0 to 11, with 11 representing the highest risk. Results of previous investigations and validations of the scoring system have highlighted that those with a score of ≤ 2 were associated with a very low rate of bleeding recurrences and death and, therefore,can be reasonably managed as outpatients. This has the potential to result in amore appropriate management of subjects'conditions based on their assessed risk of complications following the initial UGI bleeding. Further,managing low risk subjects as outpatients would free upscarce hospital resources for treating more serious cases.

Aim of the Work

This Study Aims to Evaluate:-

The validity of the Rockall score for the prediction of rebleeding and death in patients withupper gastrointestinal bleeding.

Chapter (I)

Upper Gastrointestinal Bleeding

Upper gastrointestinal bleeding (UGIB) is a major public health problem, its prevalence being around 150 per 100,000 adults per year **(Palmer, 2002; Hopper and Sanders, 2011)**. This condition is the commonest emergency medical admission for gastroenterology worldwide and has a significant inpatient mortality of 10% **(Hearnshaw et al.,2011)** that has remained unchanged over the past 30 years, in spite of the modern methods of diagnosis and treatment **(Palmer,2002; Hearnshaw et al.,2011; Amitrano et al.,2012).** Upper gastrointestinal (GI) bleeding is usually defined by a bleeding source proximal to the ligament of Treitz although some authors may also include a bleeding source in the proximal jejunum.

Many upper GI bleeding cases (e.g. erosive gastritis and esophagitis, angiodysplasia, gastric antral vascular ectasia or watermelon stomach, Cameron erosions, portal hypertensive gastropathy and small ulcers) cause iron-deficiency anemia but do not usually present as emergencies.

Upper GI bleeding emergencies were characterized by hematemesis, melena, hematochezia (if the bleeding is very

massive and brisk) and evidence of hemodynamic compromise such as dizziness, syncope episodes and shock. They were often caused by major hemorrhage from varices, ulcers, Dieulafoy lesions, Mallory-Weiss tears and neoplasms. Rare causes include hemobilia and hemosuccus pancreaticus as well as enteric fistula connecting with major blood vessels **(Enestvedt et al., 2008).**

These patients should be admitted to ICU and urgent gastroenterology consult should be requested. Surgery should also be notified in cases of massive bleeding (**Kolkman and Meuwissen 1996; Enestvedt et al., 2008).**

Upper endoscopy is the diagnostic modality of choice for acute upper GI bleeding and often the treatment of choice as well (**Adang et al., 1995; Jutabha and Jensen, 1996)**.

A recent study has shown that UGIB bleeding events result in significant mortality, similar to that of an acute myocardial infarction (0.64% versus 0.77%) after adjusting for the initial hospitalization **(Wilcox et al.,2009)**.

Variceal bleeding represents 60–65% of the bleeding episodes in patients with cirrhosis **(Garcia-Tsao et al.,2007)**. The outcome for patients with variceal haemorrhage is closely related to the severity of the underlying liver disease. The 6-

week mortality with each episode of variceal haemorrhage is approximately 15–20%, ranging from 0% among patients with Child-Pugh classA disease to approximately 40% among patients with Child-Pugh class C disease **(Villanueva et al.,2006; Abraldes et al., 2008; Bosch et al., 2008).**

Variceal bleeding in patients with cirrhosis

General considerations:

Variceal haemorrhage is a true medical emergency and a lethal complication of cirrhosis, particularly in patients in whom clinical decompensation (i.e. ascites, encephalopathy, a previous episode of haemorrhage, or jaundice) has already developed and especially in patients with Child–Pugh B or C disease in whom bleeding only stops spontaneously in about 50% of cases **(D'Amico et al., 1999)**. The risk for variceal hemorrhage increases with the severity of the liver disease **(Fallatah et al., 2012).** For these reasons, management of those patients has to be rapid and efficient to lower both morbidity and mortality.

The overall mortality of variceal bleeding in patients with cirrhosis is between 10% and 20% **(Carbonell et al.,2004)**. This mortality has decreased steadily since the 1980s, when the overall mortality was about 40%, due to aggressive resuscitation in the intensive care setting, increasing use of vasoactive drugs, therapeutic endoscopy, and antibiotic prophylaxis **(Carbonell et al.,2004)**. However, early (first 6

weeks) mortality is still high (around 40%) in Child–Pugh C patients. Risk factors for early mortality include Child–Pugh and MELD score **(D'Amico and De Franchis, 2003)**, active bleeding on admission **(Goulis et al.,1998)**, the presence of infection **(Bernard et al.,1995)**, portal vein thrombosis **(D'Amico and De Franchis, 2003)** and an initial hepatic-venous pressure gradient (HVPG) higher than 20 mmHg **(Abraldes et al.,2008)**. Although HPVG is a powerful indicator of the severity of the bleeding, it is not possible to use in everyday practice.

When addressing the management of variceal bleeding in patients with cirrhosis, we must always bear in mind that there were two essential steps for success: the management of acute bleeding and the prevention of rebleeding. After stopping the acute bleeding, if left untreated, 60% of these patients will rebleed, with a mortality of 33% **(Bosch and Garcia-Pagán, 2003**).

Pathophysiology and Pathophysiological Bases of Therapy

Gastro esophageal varices are a direct consequence of portal hypertension that, results from both increased resistance to portal flow and increased portal venous blood inflow. Increased resistances are both structural (distortion of liver vascular architecture by fibrosis and regenerative nodules) and

dynamic (increased hepatic vascular tone due to endothelial dysfunction and decreased nitric oxide bioavailability) (**Imperiale and Birgisson, 1997).**

When the portal-pressure gradient (the difference between portal-vein pressure and hepatic-vein pressure) increases above a certain threshold, collaterals develop at sites of communication between the portal and systemic circulations (**Kolkman and Meuwissen, 1996)**. This process is modulated by angiogenic factors **(Hunt,1995; Boonpongmaneeetal.,2004).**

Concomitantly with the formation of portosystemic collaterals, portal venous blood inflow increases as a result of splanchnic vasodilatation and increased cardiac output **(Levitzky and Wassefetal., 2010)** Increased portal flow maintains and exacerbates portal hypertension. Gastroesophageal varices are the most important collaterals, because as pressure and flow increase through them, they grow and eventually rupture.

Management of acute bleeding:

Available therapies for varices and varicealhemorrhage can be classified according to whether they act on the physiological mechanisms of portalhypertension.The optimal management of acute bleeding (Figure 1) requires a