Frequency of Bacteremia in Chronic Liver Diseased Egyptian Patients after Oesophageal Variceal Band Ligation

Thesis

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List of Abbreviations

A-II ..... Angiotensin II

A-V block .... Atrio ventricular block

ALB ..... Albumin

ALT ..... Alanine amino transferase

ALK..... Alkaline phosphatase

AST..... Aspartate amino transferase

ASGE..... American society for Gastrointestinal endoscopy

BSG..... British society of gastroenterology

Ca..... Calcium

CB1 ..... Cannabinoid 1

CBC ..... Complete blood picture

CDC..... Centers for disease control

Child..... Child-turcotte- Pugh score

CNS..... Central nervous system

CO..... Carbon monoxide

COX..... Cyclooxygenase

EBL..... Endoscopic band ligation

EGD ..... Oesophagogastroduedenoscopy

EIS ..... Endoscopic injection sclerotherapy

eNOS..... Endothelial nitric oxide synthase

EST ..... Endoscopic sclerotherapy

Ets ..... Endothelins

EVBL..... Endoscopic variceal band ligation

EVL ..... Endoscopic variceal ligation

FHVP..... Free hepatic vein pressure

FDA ..... Food and drug administration

GI ..... Gasterointestinal

HBV ..... Hepatitis B virus

HbsAg..... Hepatitis B surface antigen

HCV ..... Hepatitis C virus

HIV ..... Human immunodeficiency virus

HOS ..... Heme oxygenase

HREV ..... High risk oesophageal varices

HVPG ..... Hepatic venous pressure gradient

INR ..... International normalizing ratio

IQR ..... Inter-quartile range (non parametric)

ISMN ..... Isosorbide mono nitrate

IV ..... Intravenous

NO..... Nitric oxide

MELD……..MODEL FOR END- STAGE LIVER DISEASE

NSBB ..... Non selective B blocker

PGH2 ..... Prostaglandin H2

PTFE ..... Polytetrafluroethylene

PT..... Prothrombine time

PROTH.CONC Prothrombine concentration

SAGES……society of American gastrointestinal and endoscopic surgeons

SAJS ……South African journal of surgery

SGNA..... The Society of Gastroenterology Nurses and Associates

SIRS..... Systemic inflammatory response syndrome

SBP ..... Spontaneous bacterial peritonitis

TIPS..... Transjagular intrahepatic portosystemic shunts

T.BIL ..... Total bilirubin

TXA2 ..... Thromboxane A2

US..... United states

VEGF..... Vascular endothelial cell growth factor

WHVP ..... Wedged hepatic vein pressure

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INTRODUCTION

Oesophageal varices (OV) affect about 50% of patients with liver cirrhosis and formation rate is about 5% per year ***(Escorsell and Bosch, 2004)***. In patients with cirrhosis, the incidence of OV ranges from 35% to 80% and approximately a third of patients with OV experience variceal bleeding, and up to70% of the survivors have one or more additional episodes of bleeding ***(Tsokos et al., 2002)***. The identification of patients at high risk for OV bleeding enables prophylactic management ***(Kayacetin et al., 2004)***. Endoscopic variceal band ligation has become the method of choice for secondary prophylaxis of variceal bleeding. As a result of this favorable experience, there has been interest in extending the use of esophogeal band ligation to primary prophylaxis of oesophageal variceal bleeding ***(De le Pena et al., 2005)***.

Complications are rare, but no procedure is completely free of risk. If you are planning to have an endoscopic band ligation, your doctor will review a list of possible complications, which may include: Painful swallowing, bleeding, oesophageal damage, infection, fever ***(McCoy, 2009)***.

The risk of infection after upper gastrointestinal tract endoscopy has been found to be infrequent ***(Low et al., 1980)***; However, invasive endoscopic procedures can lead to various complications including remote bacterial infection ***(Vergis et***

al., 2007). The most common systemic complication of the upper endoscopic procedure is fever which is usually transitory and results from mucosal inflammation not related to infection, however, there are considerable reports of serious bacterial complications such as; brain, perinepheric abscesses, bacterial peritonitis and bacterial endocarditis ***(Allison et al., 2009)***.

Gastrointestinal bleeding is associated with bacterial infection up to 66% of patients with cirrhosis ***(Pauwels et al., 1996)***. These patients are vulnerable to infection because of disruption of the intestinal mucosal barrier and frequent invasive manipulation during hemorrhage ***(Deitch et al., 1990***. Approximately one third of cirrhotic patients with EV develop an episode of oesophageal hemorrhage, with subsequent high morbidity and mortality ***(Hong et al., 2009)***.

Endoscopic variceal ligation, which has largely supplanted sclerotherapy ***(Da Silvreira Rohr et al., 1997)***. Six studies with EVL have reported bacteremia rates ranging from 1-25% with a mean frequency of 8.8% ***(Lin et al., 2000)***. Most reported organisms were, a streptococcus of the viridans group ***(Maulaz et al., 2003)***, staphylococcus aureus ***(Rohr et al., 1997)***, coagulase-negative Staphylococcus epidermidis ***(Tseng et al., 1993)***.

Although antibiotic prophylaxis is indicated for all patients with variceal bleeding, some experts suggest that the

decision to use antibiotic prophylaxis in high-risk patients solely to prevent infectious complications should be individualized ***(Hirota et al., 2003)***. According to the guidelines antibiotic prophylaxis is not indicated for patients undergoing EBL for non-bleeding varices ***(Qureshi et al., 2005)***.

AIM OF THE WORK

The purpose of this study is to determine:

1- The incidence of bacteremia in chronic liver disease patients submitted to elastic band ligation for oesophageal varices.

2- To determine the commonest type of bacteria.

PATHOPHYSIOLOGY OF PORTAL HYPERTENSION AND OESOPHAGEAL VARICES

Introduction

Portal hypertension is a clinical syndrome defined by a portal venous pressure gradient exceeding 5 mmHg, Cirrhosis is the most common cause of portal hypertension ***(Garcia-Pagan and Bosch , 2005).*** In cirrhosis, the principal site of increased resistance to outflow of portal venous blood is within the liver itself. This results from 2 factors:

1. Mechanical obstruction to flow because of fibrotic disruption of architecture.

2. A dynamic component produced by active contraction of vascular smooth muscle cells and activated stellate cells ***(Sancho-Bru et al., 2003).***

The hallmark of portal hypertension is a pathologic increase in the pressure gradient between the portal vein and the inferior vena cava, which is measured by the hepatic venous pressure gradient (HVPG). Briefly, the wedged hepatic vein pressure (WHVP), a marker of sinusoidal pressure, and the free hepatic vein pressure (FHVP) are measured with radiologic assistance. HVPG is calculated by the following formula: