

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

Thesis

Submitted For Partial Fulfillment of Master Degree

*In Tropical Medicine
Ain Shams University*

Doaa Mohamed shaker

M.B.B.CH

Supervised by

Prof. Dr. Mohga Ali Reda

Professor of Tropical Medicine
Faculty of Medicine-Ain Shams University

Dr. General. Medhat Hassan El Sahhar

Consultant of GIT, Liver and Endoscopy
Police Hospital (El Agouza)

Dr. Hany Mansour Khalil Dabbous

Lecturer of Tropical Medicine
Faculty of Medicine-Ain Shams University

**Faculty of Medicine
Ain Shams University**

2014



Acknowledgement

First and foremost I feel always indebted to **GOD**, the most kind and the most merciful, who gives me the power to complete this work.

It is a great honour to express my sincere gratitude to **Prof. Dr. Mohga Ali Reda**, Professor of Tropical Medicine Department, Faculty of medicine, Ain-Shams University, for her kind and constant supervision, continuous encouragement and great help, which have enabled me to accomplish this work.

My deepest thanks to **Dr. Hany Mansour Khalil Dabbous**, Lecturer of Tropical Medicine Department, Faculty of Medicine, Ain-Shams University, who I am indebted to and who cared about every detail written down in this work.

I am most grateful to **Dr. General. Medhat Hassan El Sahhar**, Consultant of GIT, Liver and Endoscopy, Police Hospital (El Agouza), for his supervision and participation in this work.

Lastly I wish to express my deep thanks to **Dr. Zeinab Elqenawy, Dr. of Medical Microbiology and Immunology**, for her kind help.

List of Contents

	<i>Page</i>
• Introduction	1
• Aim of the Work.....	4
• Review of Literature:	
• Chapter (1): Oesophageal varices	5
• Chapter (2): Instrumental treatment of oesophageal varices	22
• Chapter (3): infectious disease following endoscopy	53
• Patients and Methods	70
• Results	76
• Discussion	94
• Summary	101
• Recommendations	103
• References	104
• Arabic summary	—

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	Oesophagogastrroduodenoscopy
EIS	Endoscopic injection sclerotherapy
eNOS	Endothelial nitric oxide synthase

ESTEndoscopic sclerotherapy
EtsEndothelins
EVBL.....Endoscopic variceal band ligation
EVLEndoscopic variceal ligation
FHVPFree hepatic vein pressure
FDAFood and drug administration
GIGastrointestinal
HBVHepatitis B virus
HbsAg.....Hepatitis B surface antigen
HCVHepatitis C virus
HIVHuman immunodeficiency virus
HOSHeme oxygenase
HREVHigh risk oesophageal varices
HVPGHepatic venous pressure gradient
INRInternational normalizing ratio
IQRInter-quartile range (non parametric)
ISMNIsosorbide mono nitrate
IVIntravenous
NO.....Nitric oxide
MELD.....MODEL FOR END- STAGE LIVER DISEASE
NSBBNon selective B blocker
PGH2Prostaglandin H2

PTFEPolytetrafluoroethylene
PT.....Prothrombine time
PROTH.CONC Prothrombine concentration
SAGES.....society of American gastrointestinal and
endoscopic surgeons
SAJSSouth African journal of surgery
SGNA.....The Society of Gastroenterology Nurses and
Associates
SIRS.....Systemic inflammatory response syndrome
SBPSpontaneous bacterial peritonitis
TIPS.....Transjagular intrahepatic portosystemic shunts
T.BILTotal bilirubin
TXA2Thromboxane A2
US.....United states
VEGF.....Vascular endothelial cell growth factor
WHVPWedged hepatic vein pressure

List of Figures

<i>Figure No.</i>	<i>Page</i>
Figure (1): Pathophysiology of portal HTN	9
Figure (2): Banded varix viewed through amultiband suction cap.....	31

List of Tables

Table No.

Page

Tables of Review:

Table (1): Classification of oesophageal varices hemorrhage	21
Table (2): Baveno scoring system for portal hypertensive gastropathy.....	21
Table (3): Primary prophylaxis and secondary prophylaxis of variceal hemorrhage	46
Table (4): Vasoactive agents used in the management of acute hemorrhage	50
Table (5): Approximate incidence of bacteraemia in immuno-competent individuals undergoing gastrointestinal endoscopy	67
Table (6): Classification of oesophageal varices	72

Tables of Results:

Table (1): Distribution of gender, smoking, child stage and grading of oesophageal varices among studied patients	77
Table (2): Distribution of symptoms among studied patients	78

Table (3):	Distribution of signs of liver disease among studied patients	79
Table (4):	Descriptive statistics of the age, child score and MELD score among studied patients	79
Table (5):	Comparison between gender and occurrence of infection after band ligation	80
Table (6):	Comparison between history of hematemesis and occurrence of infection after band ligation	80
Table (7):	Comparison between history of hepatic coma and occurrence of infection after band ligation	81
Table (8):	Comparison between history of melena and occurrence of infection after band ligation	82
Table (9):	Comparison between presence of ascites and occurrence of infection after band ligation	83
Table (10):	Comparison between presence of lower limb oedema and occurrence of infection after band ligation	84
Table (11):	Comparison between age of patients, mean blood count parameters and occurrence of infection after band ligation	85
Table (12):	Comparison between cases with and without infection as regards the mean liver function tests	86

Table (13): Comparison between cases with and without infection as regards the mean body temperature before and after band ligation and also comparison between cases with and without infection after band ligation as regard the mean albumin	87
Table (14): Distribution of Hepatomegaly and Splenomagly by abdominal ultra sonography	88
Table (15): Comparison between presence of hepatomegaly by ultra sonography and occurrence of infection after band ligation	89
Table (16): Comparison between Child classification of patients and the occurrence of infection after band ligation	90
Table (17): Comparison between mean Child score, MELD score and occurrence of infection after band ligation	91
Table (18): Comparison between grading OV and the occurrence of infection after band ligation	92
Table (19): Distribution of the blood stream infection after band ligation as detected by blood culture (bacteremia) and type of organisms (incidence of infection)	93

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 to 70% 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 esophageal 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, perinephric 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 Silveira 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

Introduction

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: