Endoscopic and Pathological Study for Portal Hypertensive Gastro-Duodenopathy before and After Esophageal Variceal Eradication Using Endoscopic Band Ligation

Thesis

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Mohamed Abd Elbaset

Contents

List of Abbreviations

ADH	**:**	Antidiuretic hormone
ALT	**:**	Alanine aminotransferase
Ao	**:**	Aorta
AST	**:**	Aspartate aminotransferase
CBC	**:**	Complete blood count
CE	**:**	Capsule Endoscopy
CT	**:**	Computed tomography
DAB	**:**	Diaminbenzidine tetrachloride
EBL	**:**	Endoscopic band ligation
EGD	**:**	Esophagogastroduodenoscopy
EGF	**:**	Epidermal growth factor
ET-1	**:**	Endothelin-1
EVS	**:**	Eosophageal variceal sclerotherapy
FGF	**:**	Fibroblast growth factor
FHVP	**:**	Free hepatic venous pressure
GAVE	**:**	Gastric antral vascular ectesia
GI	**:**	Gastrointestinal
GIT	**:**	Gastrointestinal tract
GOV	**:**	Gastro-oesophageal varices
HE	**:**	Hepatic encephalopathy
HS	**:**	Highly significant
HSC	**:**	Hepatic stellate cells

HVPG	**:**	Hepatic venous pressure gradient
IGV	**:**	Isolated gastric varices
INR	**:**	International Normalized Ratio
IVC	**:**	Inferior vena cava
LVP	**:**	Large volume paracentesis
MHE	**:**	Minimum Hepatic encephalopathy
MLP	**:**	Mosaic like pattern
MRA	**:**	Magnetic resonance angiography
NO	**:**	Nitric oxide
NS	**:**	Non significant
PBC	**:**	Primary biliary cirrhosis
PHC	**:**	Portal hypertensive colopathy
PHD	**:**	Portal hypertensive duodenopathy
PHE	**:**	portal hypertensive enteropathy
PHG	**:**	Portal hypertensive gastropathy
PHT	**:**	Portal hypertension
PT	**:**	Prothrombin Time
PV	**:**	portal vein
PVS	**:**	Porto venous shunt
RAAS	**:**	Renin-angiotensin-aldosterone system
S	**:**	Significant
SAAG	**:**	Serum albumin ascites gradient
SB	**:**	Small bowel
SD	**:**	Standard deviation

SEC	**:**	Sinusoidal endothelial cells
SNS	**:**	Sympathetic nervous system
TIPS	**:**	Transjugular intrahepatic portosystemic shunt
TNF	**:**	Tumor necrosis factor
US	**:**	Ultrasound
WBCs	**:**	White blood cells
WHVP	**:**	Wedged hepatic venous pressure

List of Tables

List of Figures

Introduction

Portal hypertension is the increase in hepatic pressure gradient in any portion of the portal venous system. The portosystemic gradient is assessed by measuring the wedged hepatic venous pressure (a measure of sinusoidal hepatic pressure) and subtracting the free hepatic venous pressure (systemic pressure) thus obtaining the hepatic venous pressure gradient (HVPG). A normal HVPG is 3-5 mmHg. An HVPG above 5 mmHg defines portal hypertension ***(Garcia-Pagan et al., 2005).***

Although portal hypertension could result from pre-hepatic abnormalities (e.g., portal or splenic vein thrombosis), post-hepatic abnormalities (e.g., Budd-Chiari syndrome) or intrahepatic non-cirrhotic causes (e.g., schistosomiasis, sinusoidal obstruction syndrome), cirrhosis is by far the most common cause of portal hypertension and, as such, has been the most widely investigated ***(Bosch et al., 2010 and Sanyal et al., 2008).***

An HVPG of 10 mmHg or greater defines clinically significant portal hypertension as this pressure gradient predicts clinical course in patients with cirrhosis including development of varices ***(Groszmann et al., 2005)***, clinical

decompensation (i.e., development of ascites, variceal hemorrhage and encephalopathy) ***(Ripoll et al., 2007)***, decompensation or death after liver resection ***(Bruix et al., 1996)***, and hepatocellular carcinoma ***(Ripoll et al., 2009)***

The complications that most directly result from portal hypertension are the development of varices and variceal hemorrhage. Apart from gastro-esophageal varices, these patients can also bleed form portal hypertensive gastropathy. The mechanisms involved in the pathogenesis of portal hypertensive gastropathy have not been fully elucidated. However, regulation of gastric nitric oxide, prostaglandins, tumor necrosis factor (TNF-), and epidermal growth factor (EGF) production may be involved ***(Burak et al., 2001).***

Endoscopic therapeutic interventions like sclerotherapy and band ligation have changed the outlook for patients with variceal bleeding. Sclerotherapy was the initial available modality that led to marked reduction in immediate mortality of cirrhosis due to variceal bleeding. It is now gradually being replaced by esophageal varices band ligation which has shown better results in terms of variceal obliteration and fewer side effects like ulceration, perforation and strictures formation than sclerotherapy

(Sarin et al., 1997). But few studies have shown that degree of portal hypertensive gastropathy has also been worsening after introduction of therapeutic endoscopic interventions ***(De la Pena et al., 1999).***

In view of excellent results of band ligation as far as obliteration and eradication of esophageal varices is concerned, its effect on development or worsening of portal hypertensive gastropathy has raised concern among endoscopists.

Aim of the Work

The aim of this study is to determine the endoscopic and pathologic effect of esophageal variceal eradication by endoscopic rubber band ligation on portal hypertensive gastro-duodenopathy.

Patients and Methods

Study design: prospective observational study.

Place of study: this study will be performed at Ain shams university hospitals

Patients: The patients in this study will be recruited from Tropical Medicine department, Tropical outpatient clinic and endoscopy unit at Ain shams university hospitals.

- 50 patients will be included in this study with the following inclusion and exclusion criteria

Inclusion criteria:

All patients with esophageal varices in whom band ligation is indicated according to Baveno V consensus will be included ***(de Franchis, 2010).***

Exclusion criteria:

- Presence of gastric varices
- Patients who underwent previous band ligation or variceal sclerotherapy.
- Patients who underwent Splenectomy
- Presence of portal vein thrombosis
- Presence of Hepatocellular carcinoma
- Patients with eradicated varices

Method

1-Clinical study: including full medical history and complete **clinical** examination for all systems with stress on hepatobiliary system especially ascites, jaundice, previous attacks of hepatic encephalopathy, hematmsis or melena, previous endoscopy.

2- Laboratory study

a- Complete blood count.

b- Liver function tests (ALT, AST, Albumin, PT, alkaline phosphates, total bilirubin, INR)

c- Serum creatinine)

3- Imaging

Abdominal Ultrasound will be performed

4- Upper gastro intestinal endoscopy

This will be done with esophageal variceal ligation until complete eradication of the varices, The degree of severity of PHG will be determined before and after eradication and will be recorded. The degree of severity of PHG will be recorded every endoscopic session as follows according to guidelines ***(de Franchis, 2005).***