Esophageal varices as additional site for platelet sequestration:

Effect of variceal eradication

On platelet count

THESIS

Submitted for Fulfillment of The
Master degree in Internal medicine

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2011

ACKNOWLEDGEMENT

Before all and above all, thanks to ***GOD*** *for all things*

I *am greatly honored to express my sincere gratitude, deepest appreciation to Prof. Dr.* ***Taher Elzanaty****, Professor of Internal Medicine, Faculty of Medicine, Cairo University, for his outstanding guidance and kind support throughout the work.*

I *would like to express my deepest gratitude and appreciation to Prof. Dr.* ***Mona Hegazy****, Associate Professor of Internal Medicine, Faculty of Medicine, Cairo University, for her generous help, guidance and faithful support.*

I *would like to thank all my fellow residents and dear house officers for the great help they offered throughout this work.*

I *would like to thank all patients who participated in this study and wish them a speedy recovery.*

I *dedicate this work to my family who supported me all through this work.*

May ***ALLAH*** *accept the work of all those and reward them for it.*

Ahmed Abd El-Ghany

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Abstract

Chronic liver diseases and cirrhosis are now being recognized as an important cause of morbidity and mortality world-wide Our goal was to assess the effect of variceal eradication either by endoscopic band ligation or injection sclerotherapy on platelet count.

We found that Platelet count showed significant elevation in the third month compared to the initial reading in control group on B-blocker, while Platelet count showed no significance all over the 3 months period of follow up in group I & II.

Also follow up after one and Three months showed significant increase in portal vein diameter in group I (patients undergone band ligation), but in group II (patients undergone injection sclerotherapy) there was significant decrease in portal vein diameter. While in Control groups no significant decrease. Follow up after one and Three months showed increase in splenic size in all patients, we compared within each group, there were significant increase in spleen size within patients of group II (patients undergone injection sclerotherapy).

Key words: Livsr cislhosir, orvephageol variad – plalelt count

LIST OF ABBREVIATIONS

AAAs	Aromatic amino acids
AAT	Alpha 1-antitrypsin
AFP	Alpha-fetoprotein
ALD	Alcoholic liver disease
ALT	Alanine aminotransferase
AST	Aspartate aminotransferase
ATN	Acute tubular necrosis
AVT	Antiviral therapy
CLD	Chronic liver disease
Cr	Creatine
CT	Computed tomographic scan
CTP	Child-Turcotte-Pugh score
DSRS	Distal splenorenal shunts
EGD	Esophagogastroduodenoscopy
eNOS	Endothelial NO synthase
EVL	Endoscopic variceal ligation
FHVP	Free hepatic venous pressure
GABA	Gamma-aminobutyric acid
GGT	Gamma-glutamyl transpeptidase
GOV	Gastroesophageal varices
HCC	Hepatocellular carcinoma
HCV	Hepatitis C virus
HE	Hepatic encephalopathy
HPS	Hepatopulmonary syndrome
HSC	Hepatic stellate cells
HRS	Hepatorenal syndrome
HVPG	Hepatic venous pressure gradient
IGV	Isolated gastric varices
INR	International normalized ratio
IPVDs	Intrapulmonary vascular dilatations
IVC	Inferior vena cava
LFTs	Liver function tests
MELD	Model for End-stage Liver Disease
MRI	Magnetic resonance imaging

NAFLD	Nonalcoholic fatty liver disease
NASH	Non-alcoholic steatohepatitis
NCT	Number connection test
NO	Nitric oxide
OLT	Orthotopic liver transplantation
PVT	Portal vein thrombosis
SBP	Spontaneous bacterial peritonitis
SHE	Subclinical hepatic encephalopathy
TIPS	Transjugular intrahepatic portosystemic shunt
TPO	Thrombopoietin
VEGF	Vascular endothelial growth factor
WHVP	Wedged hepatic venous pressure

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INTRODUCTION

Cirrhosis, which can be the final stage of any chronic liver disease, is a diffuse process characterized by fibrosis and conversion of normal architecture to structurally abnormal nodules. These "regenerative" nodules lack normal lobular organization and are surrounded by fibrous tissue. The process involves the whole liver and is essentially irreversible. Although cirrhosis is histologically an "all or nothing" diagnosis, clinically it can be classified by its status as compensated or decompensated. Decompensated cirrhosis is defined by the presence of ascites, variceal bleeding, encephalopathy, or jaundice, which are complications that result from the main consequences of cirrhosis: portal hypertension, and liver insufficiency. Established cirrhosis has a 10-year mortality of 34-66% **(Burroughs AK, et al., 2009)**.

At least two-thirds of patients with liver cirrhosis develop esophageal varices during the course of their disease, and severe upper gastrointestinal (GI)bleeding is a common complication of portal hypertension, affecting 30-40% of patients with cirrhosis **(Tacke F, et al., 2007)**.

Thrombocytopenia (platelet count <150,000/μL) is a common complication in patients with chronic liver disease (CLD), reported in as many as 76% of cirrhotic patients **(Giannini EG, 2006),** The clinical significance of mild thrombocytopenia (>75,000/μL–<150,000/μL) is minimal and usually does not interfere with treatment or management decisions. Moderate thrombocytopenia (50,000/μL–75,000/μL) is observed in approximately 13% of cirrhotic patients. Severe thrombocytopenia (<50,000/μL) can be associated with significant morbidity. Severe thrombocytopenia requiring platelet transfusions occurs in 1% of patients. Severe thrombocytopenia can significantly increase the risk of bleeding. Cerebral hemorrhage or hemorrhage from gastrointestinal (GI) sources is rare but can be fatal **(Thomopoulos KC., et al., 2003)**, While mild to moderate thrombocytopenia rarely leads to spontaneous bleeding during invasive procedures including liver biopsy **(Madhotra R., et al., 2002**), and liver transplantation.

AIM OF THE WORK

The Aim of the current study is to assess the effect of variceal eradication either by injection sclerotherapy or band ligation on platelet count among a group of Egyptian cirrhotic patients presenting with bleeding oesophageal varices.

Review of Literature

Chapter 1

ANATOMY AND PYSIOLOGY OF THE LIVER

ANATOMY AND PHYSIOLOGY OF THE LIVER

NORMAL PORTAL CIRCULATION

The portal venous system carries capillary blood from the esophagus, stomach, small and large intestine, pancreas, gallbladder, and spleen to the liver. The portal vein is formed by the confluence of the splenic vein and the superior mesenteric vein behind the neck of the pancreas **(Kumar S, et al., 2001),** the inferior mesenteric vein usually drains into the splenic vein. The left gastric vein, also called the left coronary vein, usually drains into the portal vein at the confluence of the splenic vein and superior mesenteric vein (**Fig.1**).

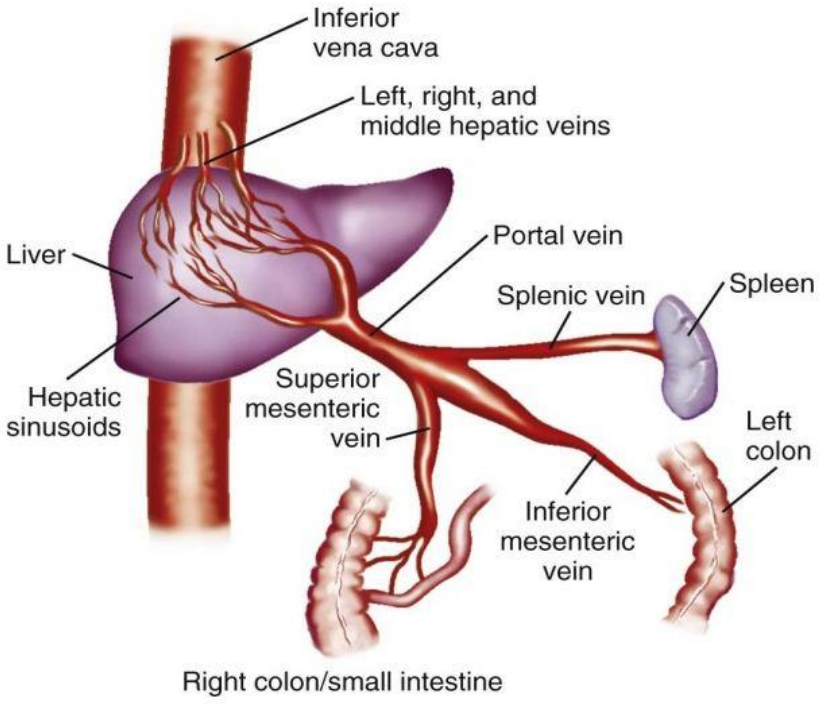

Figure 1: Anatomy of portal circulation

The portal vein is approximately 7.5 cm in length and runs dorsal to the hepatic artery and bile duct into the hilum of the liver. The uppermost 5 cm of the portal vein does not receive any tributaries **(Douglas BE, et al., 1950),** in the hilum of the liver, the portal vein divides into the left and right portal vein branches, which supply the left and right sides of the liver, respectively. The umbilical vein drains into the left portal vein. The cystic vein from the gallbladder drains into the right portal vein, whereas the portal venules drain into hepatic sinusoids that, in turn, are drained by the hepatic veins into the inferior vena cava. The left and middle hepatic veins usually join and drain into the inferior vena cava separately but adjacent to the confluence of the right hepatic vein with the inferior vena cava. The caudate lobe drains separately into the inferior vena cava.

The circulatory system of the normal liver is a high-compliance, low-resistance system that is able to accommodate a large blood volume, as occurs after a meal, without substantially increasing portal pressure. The liver receives a dual blood supply from the portal vein and the hepatic artery that constitutes nearly 30% of total cardiac output. Portal venous blood derived from the mesenteric venous circulation constitutes approximately 75% of total hepatic blood flow, whereas the remainder of blood to the liver is derived from the hepatic artery, which provides highly oxygenated blood directly from the celiac trunk of the aorta. Portal vein–derived and hepatic artery–derived blood flow converge in high-compliance, specialized vascular channels termed *hepatic sinusoids*. A dynamic and compensatory interplay occurs between hepatic blood flow derived from the portal vein and that from the hepatic artery, Specifically, when portal venous blood flow to the liver is diminished, as occurs in portal vein thrombosis, arterial inflow increases in an attempt to maintain total hepatic blood flow at a constant level. Similarly, after hepatic artery occlusion, portal venous inflow increases in a compensatory manner. This autoregulatory mechanism, aimed at maintaining total hepatic blood flow at a constant level, is termed the *hepatic arterial buffer response*.