

**STUDY OF HAEMODYNAMIC CHANGES IN PORTAL
CIRCULATION AFTER INJECTION SCLEROTHERAPY
OF OESOPHAGEAL VARICES**

Thesis

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﴿بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ﴾

«وَعَلَّمَكَ مَا لَمْ تَكُن تَعْلَمُ
وَكَانَ فَضْلُ اللَّهِ عَلَيْكَ عَظِيمًا»

«صدق الله العظيم»

«من الآية ١٣٣ سورة النساء»

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The Candidate

Abbreviations

P H T.	: Portal hypertension .
E I S.	: Endo Scopic injection Sclerotherapy .
E V L.	: Endo Scopic Variceal Ligation .
U S.	: Ultra Sound .
IVC.	: Inferior Vena Cava.
S.	: Schisto Somiasis
RVF.	: Right Ventricular Faliure .
T i.	: Tricuspid incompetence
L i.	: Locus inferior .
L m.	: Locus medialis .
L S.	: Locus Superior .
S V H.	: Splenic Venous Hypertension .
C T.	: Computed Tomography .
P V T.	: Portal Vein Thrombosis .
TIPS.	: Transvenous intrahepatic Porto systemic Shunt .
AST.	: Aspartate Transaminase .
A L T	: Alanin Transferase .

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Review Of Literature

INTRODUCTION AND AIM OF THE WORK

Portal hypertension is a medical problem with a great socioeconomic impact in Egypt. It results from many aetiological conditions that lead to impendence of portal blood flow at prehepatic, hepatic, or post hepatic site, Portal systemic collaterals, in response to portal hypertension, will develop in various sites of the body. The commonest and most dangerous site for these collaterals is the oesophagus, which was present in 80 -90% of portal hypertensive patients (*Nune et al., 1978*).

Variceal veins in the oesophageal and gastric submucosa are part of the porto systemic channels which develop when pressure increases in the portal venous bed. In these sites, varices may rupture and bleed, irrespective of the cause of portal hypertension (PH) (*Burroughs, 1992*).

Variceal bleeding which is the most serious complication of PH, may complicate previously diagnosed cirrhosis or may be the first manifestation of chronic liver disease (*Burroughs, 1992*).

Burroughs (1992). found on average risk of bleeding of 30% in cirrhotics having varices with 50% mortality within 6 weeks.

Within the last decade, endoscopic injection sclerotherapy (EIS)

has become the first line and often the definitive treatment for variceal bleeding (*Cello et al., 1986*).

Complications of sclerotherapy, however, occur in up to 20% or more of patients (*Schuman et al., 1987*). Although an accepted treatment for bleeding oesophageal varices, its various local and systemic complications may limit its effectiveness. Therefore, endoscopic elastic band ligation of oesophageal varices (EVL) was developed based on the technique of band ligation of haemorrhoids, in an attempt to provide a treatment, at least as effective as sclerotherapy but with fewer adverse side effects. (*Stiegmann et al., 1989*).

Unlike sclerotherapy, no chemical inflammation is induced, and the potential systemic toxicity of sclerosants is avoided (*Stiegmann et al., 1990,A*).

Acute variceal haemorrhage remains a catastrophic event with a high mortality from either haemorrhage itself or hepatic coma precipitated by shock and the increased protein load in the gut. Survival from such an episode is followed by increased risk of recurrence and poor prognosis (*Allison, 1983*).

In cirrhosis, the mortality of bleeding varices is about 40% with each episode & the prognosis is determined by the severity of hepatocellular disease (*Sherlock and Dooley, 1993*).