

# **SPLEEN STIFFNESS AS NON INVASIVE TOOL IN PREDICTION OF PORTAL HYPERTENSION AND GASTRO-ESOPHAGEAL VARICES IN ADULT CIRRHOTIC PATIENTS**

**Thesis**

*Submitted for Partial fulfillment of M.D degree  
in Internal Medicine*

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2016**

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

وَأَنْزَلَ اللَّهُ عَلَيْكَ  
الْكِتَابَ وَالْحِكْمَةَ  
وَعَلَّمَكَ مَا لَمْ تَكُنْ  
تَعْلَمُ وَكَانَ فَضْلُ  
اللَّهِ عَلَيْكَ عَظِيمًا

صدق الله العظيم □

سورة الفاتحة (١-٣)





## Acknowledgement

I would like to begin by thanking ALLAH for his guidance and protection, may this blessing always guide us.

Moreover I would like to express my sincere and profound gratitude to *Prof. Dr. Khaled Zakaria Al Karmoty* Professor of Internal Medicine, Hepatology and Gastroenterology Faculty of Medicine - Ain Shams University for his meticulous supervision, royal encouragement, and valuable advices throughout the work.

I wish to express deep appreciation to *Assist. Prof. Dr. Sherif Sadek Shabana* Assistant Professor of Internal Medicine, Hepatology and Gastroenterology Faculty of Medicine - Ain Shams University for his continuous guidance, unique supervision and kind care.

I am also grateful to *Assist. Prof. Dr. Zainab Ahmed Ali El-Din* Assistant Professor of Internal Medicine, Hepatology and Gastroenterology Faculty of Medicine - Ain Shams University for her kind effort, assistance she offered me throughout the performance of this work.

Also I would like to extend my warmest gratitude to *Dr. Ahmed Samir Abo Halima* Lecturer of Internal Medicine, Hepatology and Gastroenterology Faculty of Medicine - Ain Shams University for his hard and faithful effort that helped me to do this work.

I would also like to thank *Dr. Ahmed Mahmoud Abd- El Aleem* Lecturer of Internal Medicine, Hepatology and Gastroenterology Faculty of Medicine - Ain Shams University for his constant support.

*Dr. Mohamed Hussein Galal*

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### *List of Abbreviations*

<b>AASLD</b> .....	: American Association for the Study of Liver Diseases.
<b>AFP</b> .....	: Alpha feto protein
<b>ALT</b> .....	: Alanine aminotransferase
<b>AST</b> .....	: Aspartate aminotransferase
<b>AUC</b> .....	: Area under the curve
<b>BMI</b> .....	: Body mass index
<b>CO</b> .....	: Carbon monoxide
<b>COX</b> .....	: cyclo-oxygenase
<b>CSPH</b> .....	: clinically significantly portal hypertension
<b>CT</b> .....	: computed tomography
<b>CVC</b> .....	: Caudal Vena Cava
<b>DA</b> .....	: diagnostic accuracy
<b>ET</b> .....	: Endothelins
<b>EVL</b> .....	: Esophageal variceal ligation
<b>FHVP</b> .....	: Free hepatic vein pressure
<b>FT</b> .....	: fibrotest
<b>GI</b> .....	: Gastrointestinal
<b>GOV</b> .....	: Gastro-Oesophageal Varices
<b>HCC</b> .....	: Hepatocellular carcinoma
<b>HE</b> .....	: Hepatic encephalopathy
<b>HSC</b> .....	: Hepatic Stellate Cell
<b>HVPG</b> .....	: Hepatic venous pressure gradient
<b>IGV</b> .....	: Isolated Gastric Varices
<b>IHVR</b> .....	: Intrahepatic venous resistance
<b>INR</b> .....	: International normalized ratio
<b>IVC</b> .....	: Inferior vena cava
<b>kPa</b> .....	: K Pascal
<b>LS</b> .....	: Liver stiffness

*List of Abbreviations (Cont...)*

<b>MAPSS</b> .....	: Multiple acquired porto-systemic shunts.
<b>MELD:</b> .....	: Model for end-stage liver disease
<b>MRI</b> .....	Magnetic Resonance Imaging
<b>NO</b> .....	: Nitric oxide
<b>NPV</b> .....	: Negative predictive value
<b>NSBB</b> .....	: Non selective beta blocker
<b>OV</b> .....	: Oesophageal varices
<b>PBF</b> .....	: Portal blood flow
<b>PH</b> .....	: Portal hypertension
<b>PHG</b> .....	: Portal hypertensive gastropathy
<b>PPG</b> .....	: Portal pressure gradient
<b>PPV</b> .....	: positive predictive value
<b>PV</b> .....	: Portal vein
<b>PVP</b> .....	: Portal vein pressure
<b>SAAG</b> .....	: Serum ascites albumin gradient
<b>SS</b> .....	: Spleen stiffness
<b>TE</b> .....	: Transient elastography
<b>TIPS</b> .....	: Transjugular intrahepatic portosystemic shunt
<b>US</b> .....	: Ultrasound
<b>WHVP</b> .....	: Wedged hepatic vein pressure

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# INTRODUCTION

Portal hypertension (PH) is one of the most important complications of liver cirrhosis. Portal hypertension is defined as increase in the pressure of portal vein and its territory due to increase the resistance, increase blood flow, or both in the portal circulation. The clinical consequences of PH, which include the development of multiple acquired splenomegaly, porto-systemic shunts, esophageal varices, ascites, hepatic encephalopathy, or some combination of these and cause death in patients with liver cirrhosis (*Buob et al., 2011*).

Splenomegaly is a common finding in portal hypertension that should determine changes in the spleen's density because of portal and splenic congestion and/or because of tissue hyperplasia and fibrosis (*Stefanescu et al., 2011*).

Noninvasive methods can be used to evaluate the presence and degree of portal hypertension in patients with cirrhosis, and the diagnostic performance is rather fair. Methods evaluating increased hepatic vascular resistance mainly include the detection of hepatic fibrosis by serum markers and transient elastography. The radiological assessment of hyperkinetic syndrome and measurement portal and splenic veins diameter is performed by ultrasound. The assessment of severe portal hypertension by the presence of varices may be performed with

simple tools such as biological assays, CT scanning, and esophageal capsules (*Thabut et al., 2010*).

Transient elastography (fibroscan) is validated for the diagnosis of significant fibrosis and cirrhosis in chronic hepatitis, in recurrence of hepatitis after liver transplantation, in chronic cholestatic diseases, in alcoholic disease and in nonalcoholic fatty liver disease. Fibroscan is an excellent tool for the early detection of cirrhosis and for the evaluation of portal hypertension (*Victor and Julien, 2010*).

Spleen elasticity should be closely related to portal venous pressure because histologic changes in the spleen would be directly caused by portal hypertension. These changes might be quantified by elastography, so spleen stiffness can be assessed using transient elastography as its value increasing in the liver disease progresses and portal hypertension (*Colecchia et al., 2012*).

## **AIM OF THE STUDY**

The aim of this study is to assess the ability of spleen stiffness measured by transient elastography (fibroscan) in prediction of portal hypertension and gastro-esophageal varices in adult cirrhotic patients.

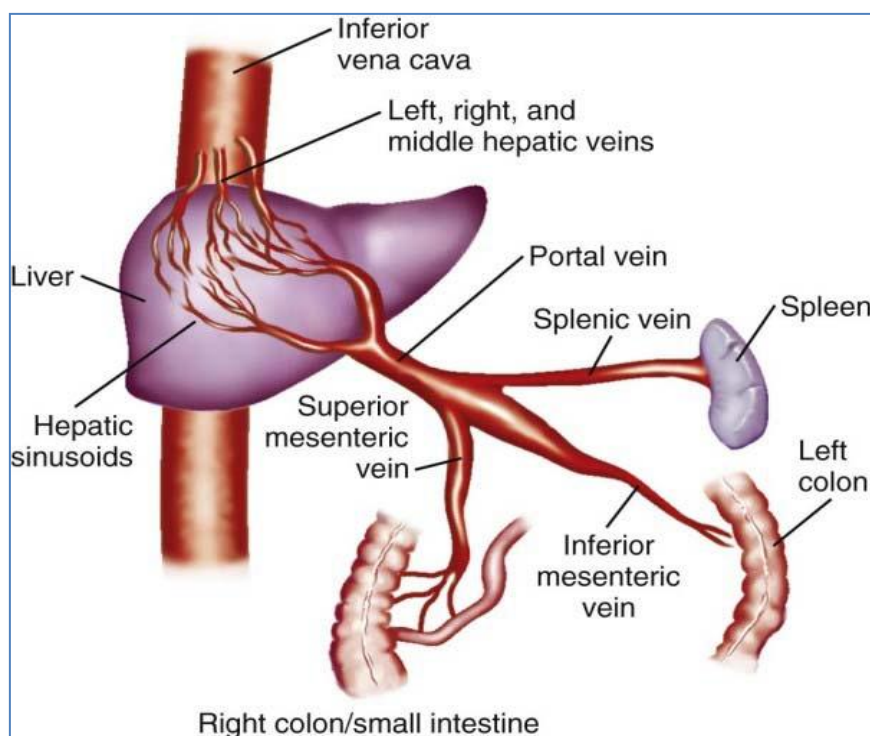
*Chapter 1***PORTAL HYPERTENSION****Definition:**

Portal hypertension is a frequent syndrome – most often caused by chronic liver diseases – which is characterized by an increased portal pressure gradient (PPG; the difference in pressure between the portal vein and the inferior vena cava which represents the perfusion pressure of the liver with portal blood). The increased portal pressure leads to other consequences, such as splenomegaly, growth of an extensive network of portal-systemic collaterals that shunt portal blood flow to the systemic circulation bypassing the liver and development of a hyperkinetic circulatory state. In normal conditions the PPG ranges between 1 and 5 mmHg. Portal hypertension becomes clinically significant (associated with risk of clinical complications) when the PPG increases to 10 mmHg or above. Values between 5 and 9 mmHg represent subclinical portal hypertension (*Berzigottiet al., 2013*).

**The portal venous system:***1) Anatomy of the Portal Venous System:*

The portal system includes all veins that carry blood from the abdominal part of the alimentary tract, the spleen, pancreas

and gallbladder. The portal vein enters the liver at the porta-hepatis in two main branches, one to each lobe; it is without valves in its larger channels. The portal vein is formed by the union of the superior mesenteric vein and the splenic vein just posterior to the head of the pancreas at about the level of the second lumbar vertebra; it courses superiorly and toward the right passing behind the first part of the duodenum and anterior to the inferior vena cava (Fig.1). It extends slightly to the right of the midline for a distance of 5.5 – 8 cm to the porta hepatis (*Mathur, 2008*).



**Fig.(1):** Anatomy of the portal circulation. Blood vessels that constitute the portal circulation and hepatic outflow tracts are depicted (*Sleisenger and Fordtran, 2010*).