Portal Vein and Left Gastric Vein Haemodynamics in Portal Gastropathy with & without Oesophageal Varices

Thesis
Submitted in partial fulfillment for
Master Degree in Internal Medicine
By

Thanaa Abd El-Hamid Nasr
M.B. B.Ch.

Supervised by

Prof. Dr.
Soheir Saeed Sheir
Professor of Internal Medicine
Faculty of Medicine, Ain Shams University

Dr.
Ahmed Shawky El-Sawaby
Assist. Professor of Internal Medicine
Faculty of Medicine, Ain Shams University

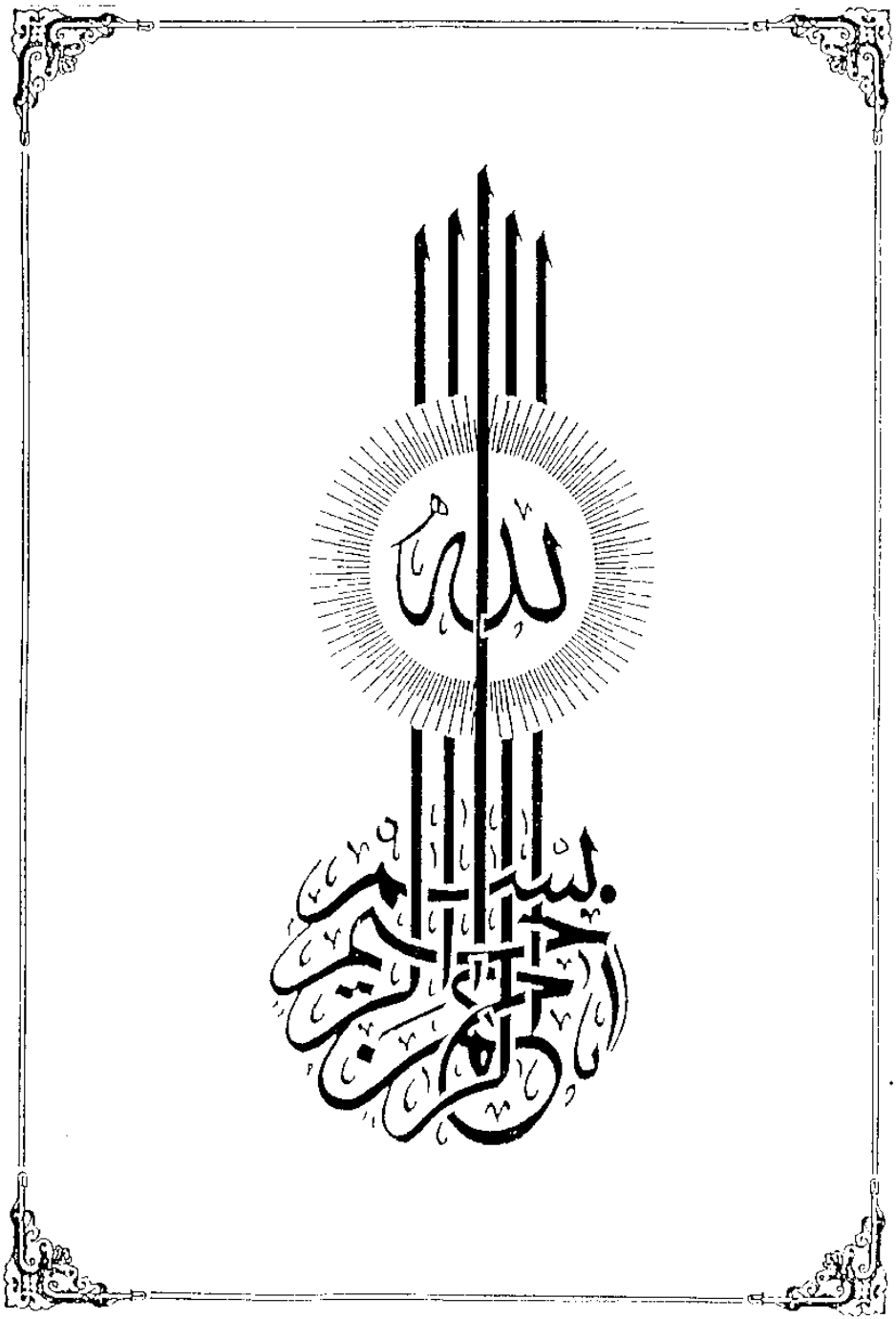
Dr.
Mahmoud Abd El-Megid Othman
Lecturer of Internal Medicine
Faculty of Medicine, Ain Shams University

FACULTY OF MEDICINE
AIN SHAMS UNIVERSITY
1995

"قالوا سبحنك لا علم لنا الا ما
علمتنا انك أنت العليم الحكيم."

صدق الله العظيم

سورة البقرة- آية ٣٢



Acknowledgment

First, and foremost, Thanks to GOD.

I would like to express my sincere thanks and deepest gratitude to Prof. Dr. Soheir Sheir, Professor of Internal Medicine, Faculty of Medicine, Ain Shams University, who offered me encouragement and support throughout this study.

I am greatly indebted to Dr. Ahmed Shawky, Assist. Professor of Internal Medicine, Faculty of Medicine, Ain Shams University, for his active cooperation, valuable advice and expert guidance throughout the entire work.

I am honored to thank Dr. Mahmoud Abd El-Megid, Lecturer of Internal Medicine, Faculty of Medicine, Ain Shams University, for his encouragement and the help he offered me for the completion of this work possible.

LIST OF ERRATA

Page	Line	Wrong	Correct
8	6	reachs	reaches
16	1	abnormlties	abnormalities
19	8	errosions	erosions
20	4	gasric	gastric
28	21	methos	methods
29	21	dimensial	dimensional
33	4	acurate	accurate
35	12	adminstration	Administration
36	2	volme	volume
37	19	acuracy	accuracy
38	17	mesentric	mesenteric
42	10	mesentric	mesenteric
44	6	mesentric	mesenteric
46	6	initialy	initially
59	18	remaing	remaining
95	14	occurance	occurrence

CONTENTS

	Page
INTRODUCTION & AIM OF THE WORK	1
REVIEW OF LITERATURE	3
ANATOMY AND PHYSIOLOGY OF PORTAL SYSTEM	3
PORTAL HYPERTENSIVE GASTROPATHY	15
COLOR AND PULSED DOPPLER SONOGRAPHY OF THE NORMAL PORTAL VENOUS SYSTEM	28
SUBJECTS & METHODS	49
R E S U L T S	56
DISCUSSION	90
SUMMARY & CONCLUSION	96
REFERENCES	98
ARABIC SUMMARY	

LIST OF TABLES

Page

Table (1): Differential diagnosis of gastic mucosal abnormalities in portal hypertension 16
Table (2): P.V. measurement of Group I 63
Table (3): Clinical findings in Gr. II 64
Table (4): Clinical findings in Gr. III 65
Table (5): Liver function test in Group II 66
Table (6): Liver function test in Group III 67
Table (7): Endoscopic findings in Gr. II 68
Table (8): Endoscopic findings in Gr. III 69
Table (9): Color Doppler measurements in Group II 70
Table (10): Color Doppler measurements in Group III 71
Table (11): Color Doppler measurement in Group II & III 72
Table (12): Relation between PHG and sclerotherapy 73
Table (13): Relation between PHG and varices 74
Table (14): Liver function tests in Group II & III 75
Table (15): Comparison of clinical findings in Group II & III 76
Table (16): Real time abdominal U/S 77
Table (17): Upper endoscopic findings in Group II & III 78
Table (18): Real time U/S findings in Gr. II 79
Table (19): Real time U/S findings in Gr. III 80
Table (20): Comparison between Groups I, II, & III regarding haemodynamics 81

LIST OF FIGURES

Page

Fig. (1): Scheme of the causes and sites of block of portal hypertension 10
Fig. (2): Doppler wave forms of portal and hepatic veins 32
Fig. (3): Relation between PHG and Varices 82
Fig. (4): Relation between sclerotherapy and PHG 83
Fig. (5): Comparison of color Doppler measurement of portal vein in the 3 groups under study 84
Fig. (6): Comparison of color Doppler measurement of left gastric vein in groups II & III 85
Fig. (7): Liver size (by abdominal U/S) in Group II & III 86
Fig. (8): Comparison of liver function tests in Group II & III 87
Fig. (9): Doppler ultrasound of portal vein 88
Fig. (10): Doppler ultrasound of left gastric vein 89

INTRODUCTION
&
AIM OF THE WORK

INTRODUCTION

The frequency and importance of gastric mucosal lesion in patients with portal hypertension have been increasingly recognized in recent years (Smart and Triger, 1991). The stomach of the cirrhotic patients with portal hypertension are frequently subjected to a number of alterations which can be differentiated into 4 stages during endoscopic examinations:

I. Superficial reddening on the surface of the gastric rugae.

II. White reticular pattern separating areas of prominent pink oedematous mucosa (Snake - Skin or mosaic pattern).

III. Cherry red spots.

IV. Diffuse bleeding (Nillius and Zipprich, 1991).

Portal hypertension is characterized by a pathological increase in portal venous pressure. Increased vascular resistance to portal blood flow is the initiating factor in portal hypertension. Splanchnic vasodilatation with increased blood flow is an additional factor which contributes to the maintenance and aggravation of portal hypertension (Bosch, et al., 1986b).

Doppler measurements give accurate non invasive estimation of portal blood flow and so this technique may be used to monitor physiological and pathological changes in patients with portal hypertension (Alvarez, 1991).

Aim of the work

The aim of the work is to study the portal hypertensive gastropathy in relation to the portal vein and left gastric vein haemodynamics.

REVIEW OF LITERATURE

ANATOMY AND PHYSIOLOGY OF PORTAL SYSTEM

The portal system includes all veins that carry blood from the abdominal part of the alimentary tract, the spleen, pancreas and gall bladder. The portal vein enters the liver at the porta hepatis in two main branches, one to each lobe; no valves exists in its larger channels. From the liver it is ultimately drained into the inferior vena cava by the hepatic vein, (Dougllas et al., 1950).

The portal vein is formed by the union of the superior mesenteric vein and splenic vein just posterior to the head of the pancreas at about the level of second lumber vertebra. It extends slightly to the right of the midline for a distance of 5.5-8 cm to the porta hepatis. The portal vein has a segmental intra-hepatic distribution accompanying the hepatic artery.

The superior mesenteric vein is formed by tributaries from the small intestine, colon and head of the pancreas, and irrigularly from the stomach via the right gastro-epiploic vein.

The splenic veins (5-15 channels) originate at the splenic hilum and join near the tail of the pancreas with the short gastric vessels to form the main splenic vein. This proceeds in a transverse direction in the body and head of the pancreas, lying below and in front of the artery. It receives numerous

tributaries from the head of pancreas, and the left gastro-epiploic vein enters it near the spleen. *The inferior mesenteric vein*, bringing blood from the left part of the colon and rectum, usually enters its medial third. occasionally, however it enters the junction of superior mesenteric and splenic vein.

Physiology:

Portal blood flow in man is about 1000-1200 ml/min. Portal Oxygen Content: The fasting arterio portal oxygen difference is only 1.9 volumes percent (range o.4-3.3 vol.%) (Smyth et al. 1951). The portal vein contributes 40 ml/min or 72% of the total oxygen supply to the liver. During digestion the arterioportal venous O_2 difference increases due to increased intestinal utilization (Smyth et al., 1951).

The portal vein is therefor an undependable source of O_2, supplying least during digestion when hepatic activity is greatest. So Reynolds (1982) said that portal venous blood differs from most other venous blood in:

a) Being under a slightly higher pressure in order to over-come

the resistance of hepatic sinusoids.

b) Being less depleted in O_2 because of the relatively high blood

flow through the splanchnic area.

c) And containing many nutrient and bacterial waste products from digestive tube that are in route to the liver.

The high pressure hepatic arterial stream and low pressure portal venous system unite at the level of hepatic sinusoids. The portal vein supplies about 2/3 of hepatic blood flow and about 1/2 of the total O2 consumption, while the hepatic artery contributes the remainder (Reynolds, 1982).

A persistent increase above normal portal vein pressure is called portal hypertension. This appears to be due to a primary increase in vascular resistance some where in the portal circuit and is accompanied by dilatation of the venous bed behind the obstruction resulting in stasis and decrease in the amount of blood flowing through the normal vascular channels with reciprocal increase in collateral blood flow around the liver. The final level of pressure in the portal blood depends on the degree of vascular obstruction, the resistance in the collateral vessels and the rate of inflow of blood into the splanchnic bed (Reynolds, 1982). There is no constant pattern of hepatic distribution of portal inflow, so sometimes splenic blood goes to the left and sometimes to the right lobe (Kashiwagi et al., 1980).

Flow is probably stream lined rather than turbulent. Portal pressure in normal person is about 7 mm Hg. (Sherlock, 1985).

Portal vein:

This vessel is merely the upward continuation of the superior mesenteric vein, which changes its name to portal vein after it has received the splenic vein behind the neck of the pancreas. It lies in front of the inferior vena cava, passes upwards behind the pancreas and the first part of the duodenum