Morphological Changes in GIT in Portal hypertension

Essay

Submitted For Partial Fulfillment of Master Degree
In General Surgery

Presented By

Mahmoud Ahmed Hegazy Ahmed
M.B.,B.CH.

Under Supervision of

Prof. Dr.Tewfik Saad Mohamed Fahim

Professor of General Surgery
Faculty of Medicine-Ain Shams University

Prof. Dr.Naglaa Medhat Ebrahim Abou-rabia

Professor of Histology
Faculty of Medicine-Ain Shams University

Dr. Ahmed Elnabil-Mortada

Lecturer of General Surgery
Faculty of Medicine- Ain Shams University

Faculty of Medicine
Ain Shams University
٢٠١٢

Acknowledgement

Praise worthy thanks and all gratitude are due, first and full measure to merciful **ALLAH** who gives and facilitates everything.

No wards can describe my appreciation, gratitude and respect for **Prof. Dr.Tewfik Saad Fahim** Professor of General Surgery - Faculty of Medicine-Ain Shams University for his fatherly support not only during and throughout my essay work, but also throughout my medical career, he was the first hand who learn me how to be a good surgeon .

My sincere gratitude to **Prof. Dr.Naglaa Medhat Ebrahim** Professor of Histology - Faculty of Medicine -Ain Shams University for the supervision, and for great help in all the stages of this work .

I would like to express my deepest gratitude and appreciation to **Dr. Ahmed Elnabil-Mortada** Lecturer of General Surgery - Faculty of Medicine- Ain Shams University who assisted me with his kind supervision, patience, encouragement and his advice all through my work.

Finally , I dedicate this work to soul of **My Mother** , and I also would like to thank my **Dad** who support me in all my life and still do , and I would like to dedicate all my success to my lovely **WIFE** who help me and support me in all my life.

Contents

List of figures :-

Introduction

Portal hypertension is a frequent syndrome characterized by a chronic increase in portal venous pressure and by the formation of portal-systemic collaterals. Its main consequence is massive bleeding from ruptured esophageal and gastric varices. Bleeding is promoted by increased portal and variceal pressure, and is favored by dilatation of the varices ***(Bosch et al, ١٩٨٩).***

Changes in portal pressure are regulated by changes in hepatic vascular resistance, which is normally under neurohumoral control, and portal tributary blood flow. Two theories on the pathophysiology of portal hypertension have been proposed: the 'backward flow' theory, in which portal hypertension is attributable to increased resistance to portal venous flow, and the 'forward flow' theory, in which increased splanchnic blood flow maintains portal hypertension despite extreme portal-systemic shunting ***(Okumura et al, ١٩٨٩).***

The site of increased intrahepatic resistance is variable and is dependent on the disease process. The site of obstruction may be: pre-hepatic, hepatic, and/or post-hepatic. Peripheral vasodilatation initiates the classical profile of decreased systemic resistance, expanded plasma volume, elevated splanchnic blood flow and elevated cardiac index ***(Tarun et al, ١٩٩٧).***

The therapy of portal hypertension depends to a significant extent on its clinical manifestation. In cases of acute haemorrhage from oesophageal varices, the objective of the therapy is to stop the haemorrhage and to decrease the pressure

and the reflux within the portal vascular bed. Urgent sclerotisation under the simultanous pharmacologic decrease of portal hypertension is successful in ٩٣-٩٥% ***(Kupcová et al, ١٩٩٨).***

There are many morphological changes in GIT in portal hypertension, in upper and lower GIT and in hepato-pactreatco-biliary system.

Portal hypertensive gastropathy (PHG) occurs as a complication of cirrhotic or non-cirrhotic portal hypertension. Although the pathogenesis of PHG is not completely understood, evidence suggests that the key factor for the development of PHG is portal hypertension. PHG is clinically important because it may cause acute massive or insidious blood loss ***(Cubillas & Rockey, ٢٠١٠).***

In patients with liver cirrhosis and portal hypertension, Portal hypertension colopathy is thought to be an important cause of lower GIT hemorrhage ***(Ito et al, ٢٠٠٥).***

Splenomegaly is a frequent finding in patients with liver cirrhosis and portal hypertension and may cause hypersplenism. The occurrence of thrombocytopenia in those patients can be considered as an event with multiple etiologies ***(Djordjević J et al, ٢٠١٠).***

Small bowel mucosa in portal hypertension was recently assessed extensively ***(Misra et al, ١٩٩٧).***

Many endoscopic abnormalities are mentioned in this setting such as anorectal and recto-sigmoid varices , hemorrhoids, multiple vascular ectasia like lesions, and nonspecific inflammatory changes ***(Bini EJ et al, ٢٠٠٠).***

Aim of the work

- The aim of the present study is to review the morphological changes in gastro-intestinal tract in portal hypertension disease.

Chapter ١

A - Surgical anatomy and embryology of portal vein:-

- The portal system includes all the veins which drain the blood from the abdominal part of the digestive tube (with the exception of the lower part of the rectum) and from the spleen, pancreas, and gall-bladder. From these viscera the blood is conveyed to the liver by the portal vein.

- The portal vein (vena portae) is about ٨ cm. in length, and is formed at the level of the second lumbar vertebra by the junction of the superior mesenteric and lienal veins, the union of these veins taking place in front of the inferior vena cava and behind the neck of the pancreas.

- It passes upward behind the superior part of the duodenum and then ascends in the right border of the lesser omentum to the right extremity of the porta hepatis, where it divides into a right and a left branch, which accompany the corresponding branches of the hepatic artery into the substance of the liver.

- **In the lesser omentum** it is placed behind and between the common bile duct and the hepatic artery, the former lying to the right of the latter**.**

- **It is surrounded by the hepatic plexus of nerves, and is accompanied by numerous lymphatic vessels and some lymph glands.**

- **The right branch** of the portal vein enters the right lobe of the liver, but before doing so generally receives the cystic vein**.**

- **The left branch,** longer but of smaller caliber than the right, gives branches to the caudate lobe, and then enters the left lobe of the liver**.**

- **The ligamentum teres (obliterated umbilical vein),** and is united to the inferior vena cava by a second fibrous cord, the ligamentum venosum (obliterated ductus venosus).

Tributaries of the portal vein

- The tributaries of the portal vein are: Lienal , Superior Mesenteric, Coronar , pyloric, cystic and para-umbilical veins .

A - The Lienal Vein (*v. lienalis; splenic vein*) : -

- Commences by five or six large branches which return the blood from the spleen. Tributaries of The lineal vein are The short gastric veins , The left gastroepiploic vein , The inferior mesenteric veins and The pancreatic veins.

B - The Superior Mesenteric Vein (*v. mesenterica superior*)

- Returns the blood from the small intestine, from the cecum, and from the ascending and transverse portions of the colon.

C - The Coronary Vein (*v. coronaria ventriculi; gastric vein):*

- Derives tributaries from both surfaces of the stomach.

D - The Pyloric Vein :

E - The Cystic Vein (*v. cystica*):

- Drains the blood from the gall-bladder, and, accompanying the cystic duct, usually ends in the right branch of the portal vein .

F - Parumbilical Veins (*vv. parumbilicales*)

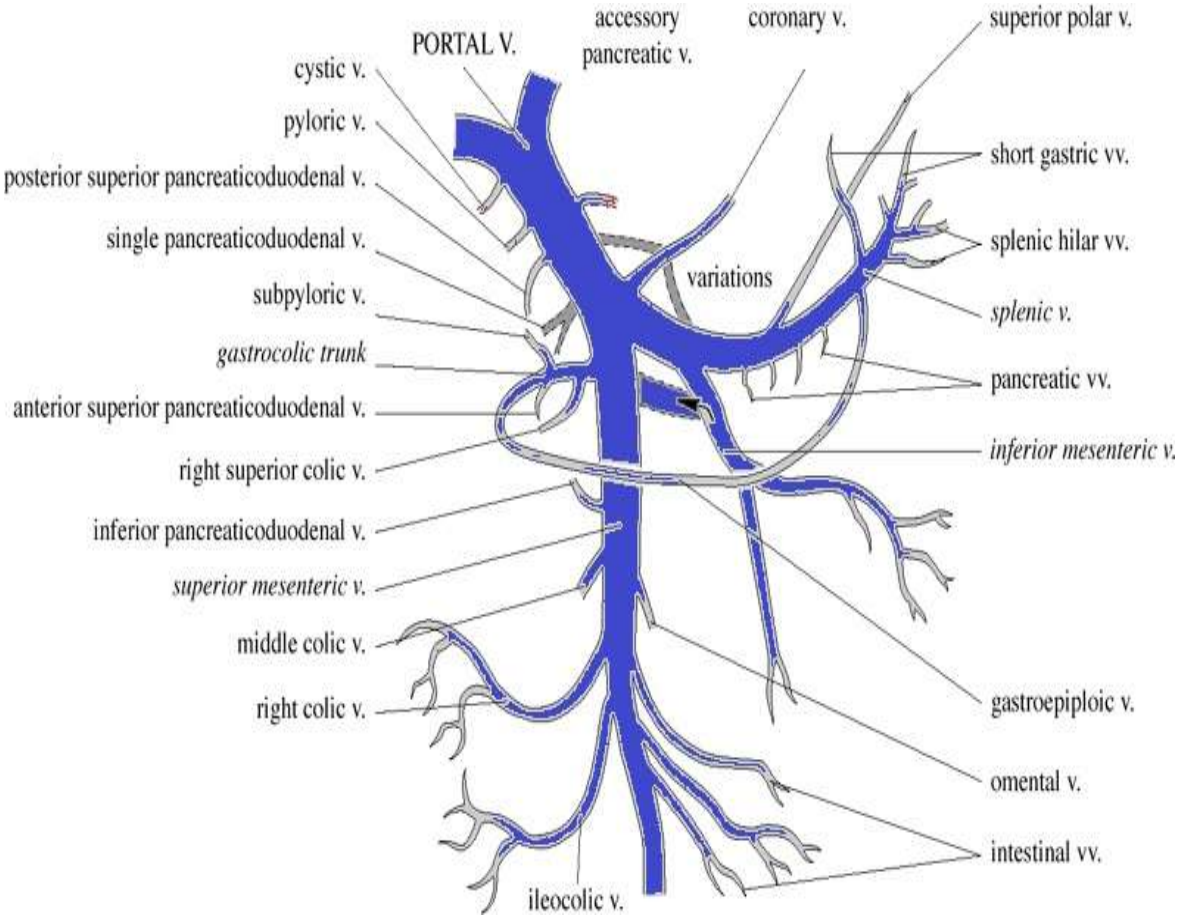
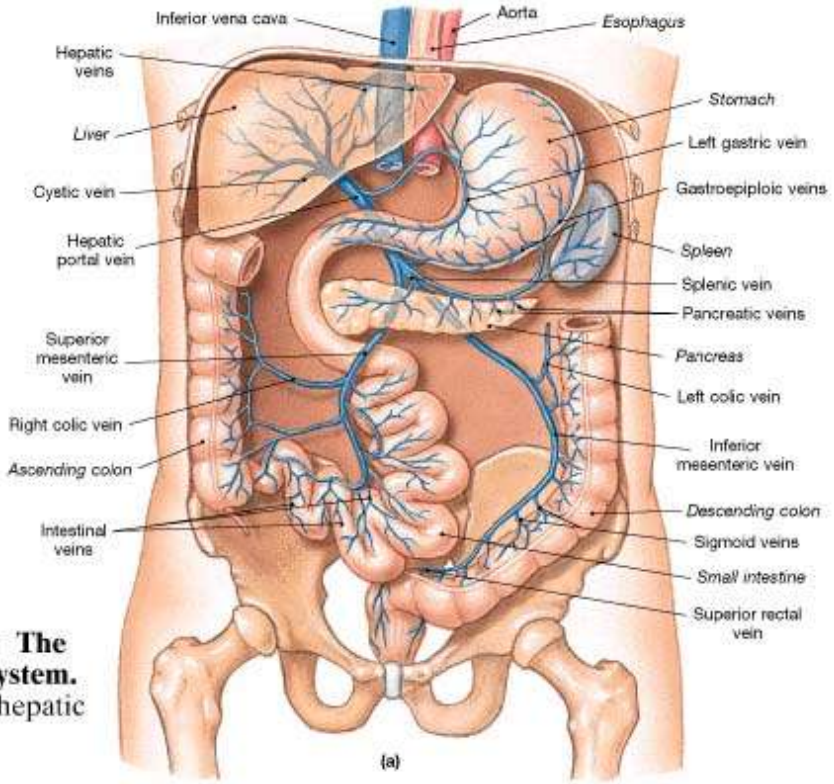


Fig ١٠ : anatomy of portal hypertension and its tributaries .



•FIGURE 21-35 The Hepatic Portal System. **(a)** Vessels of the hepatic portal system.

Fig ٢ : anatomy of portal hypertension and its Tributaries

Embryology of the Portal System :-

- The portal system first appears, arteries and biliary ducts develop subsequently. In hamster, Nettelblad [٦] noted the first lobulation and the disposition of the vitelline and umbilical veins in his stage ٣ (٩th day, ٦th hour).
- Arteries and biliary ducts appear at stage ١٢ (٢١st day, ٥th hour), only when the portal branching is formed. In man, Lassau [٩] found buds of the future right and left hepatic ducts in an ١٨-mm embryo (٤٠–٤٢ days, Streeter H = XX), and Martin and Convert [١٠] detected right and left hepatic ducts in a ٢٦-mm long embryo (٤٤–٤٦ days, H = XXII): the portal and hepatic vein systems are fully developed.
- Shah and Gerber [١١] detected the first appearance of small ductal plate cells in the mesenchyma along the branches of the portal vein at the ٤th week of gestational age.
- Since the portal vein branching is first organized and since the biliary (and arterial) branching appears later and is dependent on the portal branches, portal vein segmentation should strongly be recommended.

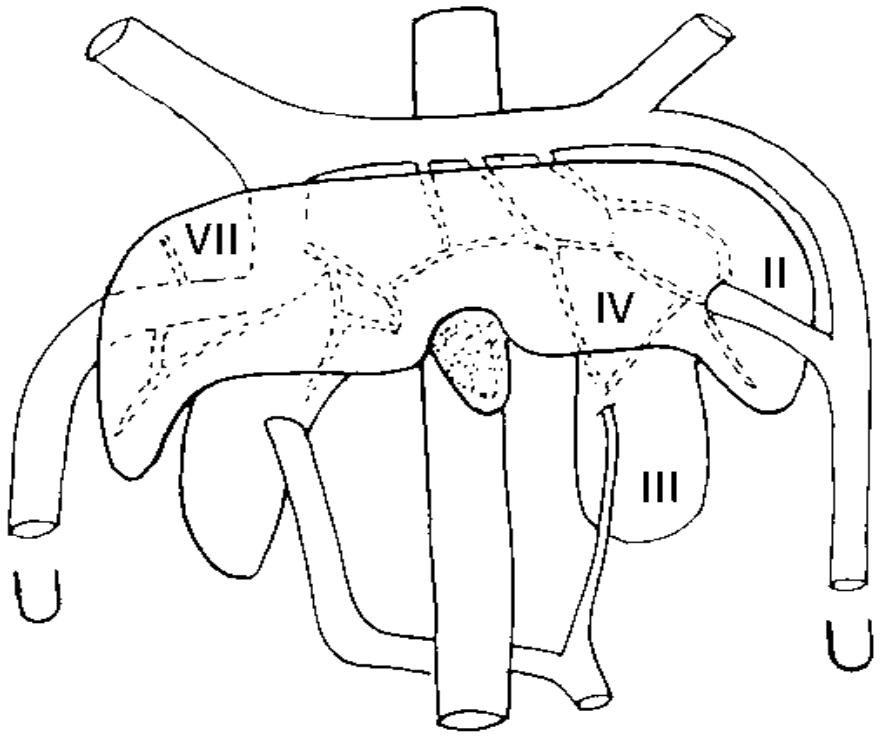


Fig ٣ . Formation of the new left umbilical vein in hamster (Nettel- blad's stage The development is more evident in a nonlobated liver. Two lateral lobes (future segments II and VII) from around the vitelline veins. A large middle lobe (future right and paramedian [posterior and anterior] sectors) from around the cholecystic axis; between the left lateral and the middle lobes is a left interlobar fissure (equivalent to the left portal fissure in nonlobated livers). A branch from the left umbilical vein *enters the left portion of the middle lobe* (left paramedian sector). The branch will partly irrigate the left liver and is located between segment IV on the right and segment III on the left .

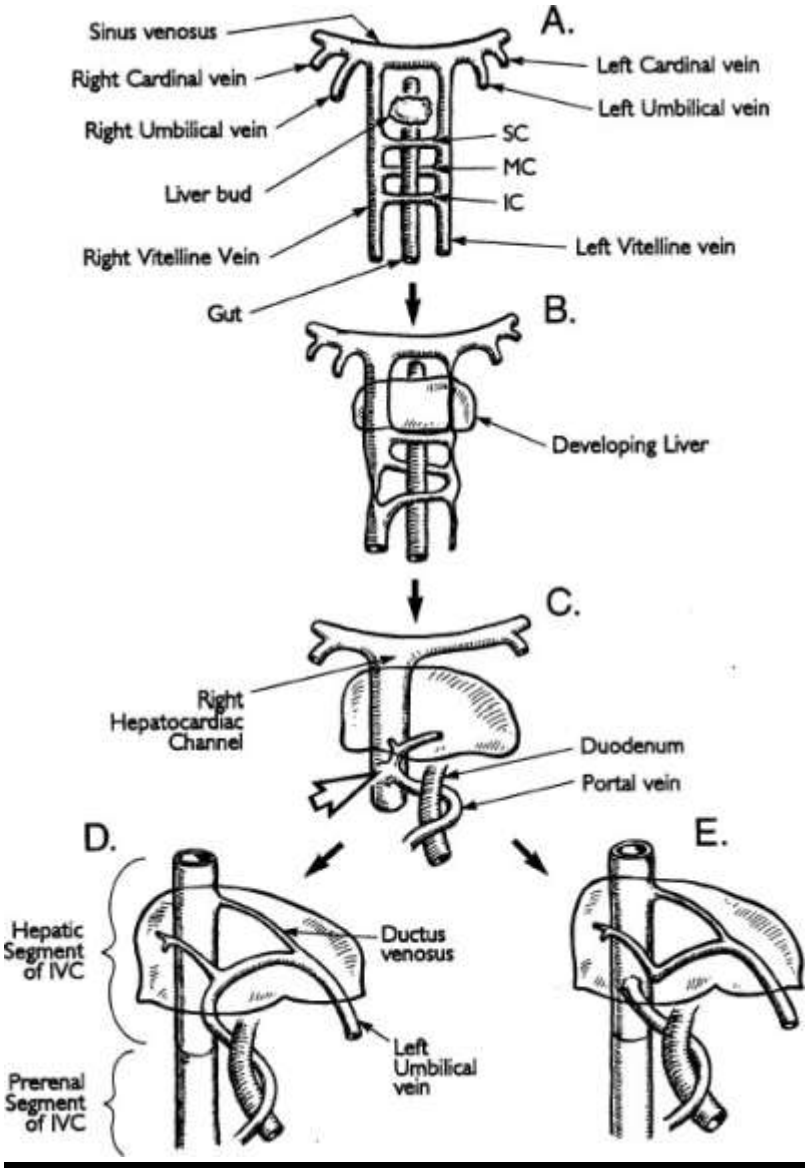


Fig ٤ : These schema show the embryological development of the portal vein and the embryological pathology leading to a congenital portosystemic shunt. Normal development shows *A*→*B*→*C*→*D*. Abnormal development shows *A*→*B*→*C*→*E*. The large white arrow at *C* indicates the part of the right vitelline vein that becomes the portal vein and disconnects from the upper part of the right vitelline vein, which becomes the hepatic segment of the inferior vena cava. SC, superior communication; MC, middle communication; IC, inferior communication; IVC, inferior vena cava.