CAVERNOUS TRANSFORMATION OF PORTAL VEIN: A RARE ANATOMIC DEVELOPMENT WITH EMPHASIS ON ANATOMY AND PHYSIOLOGY OF PORTAL BLOOD FLOW


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ABSTRACT

Portal vein contributes to more than 70 % blood flow to the liver the significant increases in portal venous blood pressure is associated with major systemic manifestations. Having a knowledge about Portal Blood supply is important to any clinician, gastroenterologist or any Hepatic Surgeon. The Article outlines the rare development of Portal Cavernoma in a patient who had Portal Hypertension. Portal cavernoma is a consequence of portal vein thrombosis. We present a case of a lady with mild Right hypochondriac pain who on evaluation was found to have Cavernomatous transformation of Portal vein without any other abnormality.

KEYWORDS: Portal Vein; Cavernoma; Hepatic; Vitelline; Caput; Varices; Porto Systemic.

INTRODUCTION

The Portal vein is unique in feature that it is one of the most important source of blood supply to liver and even surpasses the hepatic artery as far as the dynamics of blood flow to liver are concerned. The Relative blood flow and supply to the Liver through Portal Vein is more than the Hepatic artery or Hepatic Veins. Further the Neuroregulatory control of Portal Blood Flow is highly complex and intricate. The Normal diameter of Portal vein varies from 7-15mm but sometimes can get profoundly increased and transformed into a multiple channels a sign of cavernous transformation.

DISCUSSION

The Embryogenesis of the Portal vein starts with the development of Vitelline veins after the yolk sac disappears, these newly and primitively developing vitelline veins regress almost totally but are seen to persist only at a certain part where the future portal vein is to be formed. The Latest concepts of Liver Segments is based upon the general relationships between the hepatic veins and portal vein [1]. This can be demonstrated by injection of methylene blue. Under normal circumstances The portal vein provides about more than 70 percent of the liver’s blood supply. The Important Point ie. junction of the superior mesenteric and splenic veins forms the portal vein posterior to the neck of the pancreas [2]. The Normal portal vein then passes superiorly, posterior to the first part of the duodenum at the level of the second lumbar vertebra. Normally the Portal vein
ranges from 1 to 3 cm. in diameter and 5 to 8.5 cm. in length.

The characteristic feature of portal vein includes the it is the one vein which begins and also ends in capillaries. The areas from which the blood is drained by the portal vein are the Abdominal part of alimentary tract, Spleen, Gall bladder and the Pancreas.

Anatomically the Portal vein is divided into 3 parts: Infraduodenal, Retroduodenal and the Supraduodenal part.

**Fig. 1:** USG demonstrating Portal Cavernoma.

Further it is seen to divide the into right and left branches at the porta hepatis. There can be three or rarely four branches of Portal vein and that can be an anatomic variation to be taken notice of. The portal vein usually passes behind the bile duct and hepatic artery in the hepatoduodenal ligament. The Main portal trunk divides into left and right hepatic branches in the portal fissure. On measurement it is seen that usually the left branch of the portal vein is longer than its right counterpart and the right branch of the portal vein divides into anterior and posterior segments approximately at the point of entry into liver parenchyma. Further it can be observed that The portal vein further branches and subdivides into small veins and venules, which finally enter to open into pools of blood referred to as hepatic sinusoids. It has been observed that the smaller branches of the portal vein connect the liver with the systemic venous system which are of insignificant value Under normal physiological circumstances. Under Pathological conditions and in cases of portal hypertension these portosystemic communications open up and develop into large channels with increased collateral flow. The most important natural portosystemic anastomoses include esophageal varices [3], caput medusae, hemorrhoids and retroperitoneal communications, including connections to the renal and adrenal veins.

The sites of portal-systemic communications the portal and systemic veins forming these are:

<table>
<thead>
<tr>
<th>Sites</th>
<th>Portal vein</th>
<th>Systemic vein</th>
</tr>
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<tbody>
<tr>
<td>Umbilicus</td>
<td>Left branch of portal vein through paraumbilical vein</td>
<td>Veins of anterior abdominal wall</td>
</tr>
<tr>
<td>Lower end of esophagus</td>
<td>Oesophageal tributaries of left gastric</td>
<td>Oesophageal tributaries of the accessory hemiazygous vein</td>
</tr>
<tr>
<td>Anal canal</td>
<td>Superior rectal vein</td>
<td>Middle and inferior rectal vein</td>
</tr>
<tr>
<td>Bare area of liver</td>
<td>Hepatic vein</td>
<td>Phrenic and intercostals veins</td>
</tr>
<tr>
<td>Posterior abdominal wall</td>
<td>Veins of duodenum ascending and descending colon</td>
<td>Retroperitoneal veins of abdominal wall and renal capsule</td>
</tr>
<tr>
<td>Liver</td>
<td>Rarely ductusvenosus remains patent and then connects left branch of portal vein</td>
<td>Inferior vena cava</td>
</tr>
</tbody>
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Under Microscopic study The terminal branches of the the portal vein and the hepatic vein do not meet but are separated by a space between them filled with hepatic cell plates and
sinusoids. These branches progressively become smaller as they penetrate the liver substance, while the terminal hepatic veins connect with the sinusoidal bed, piercing through closely applied cell plates [4].

This lady with mild Right hypochondriac pain who on evaluation was found to have Cavernomatous transformation of Portal vein without any other abnormality.

Following portal vein obstruction the portal vein is replaced by a numerous tortuous venous channels instead of a single portal vein.

The non recanalization of portal vein after obstruction leads to the collateral vein development and these veins become dilated and serpigenous. The resultant development of portal venous hypertension can cause a variety of changes in liver including either an atrophy of liver segments or hypertrophy if associated with cirrhosis of liver.

Ultrasound is an effective modality for diagnosis in the first place and can show numerous tortuous vessels. Ultrasound fails to demonstrate a normal portal vein caliber and instead multiple serpentine channels are seen. Color Doppler confirms the presence of portal venous blood flow within these serpentine, tortuous channels. A multiphase CT scan and MRI [5, 6] also confirms the diagnosis with calcification sometimes. All these changes take a time span extending from months to years. The Cavernous transformation is associated with Behcets disease, Polycythemia vera, Factor V Leidin Mutations, Myelofibrosis, Bdd Chiari Syndrome [7, 8].

**CONCLUSION**

The Normal Anatomy of Portal Vein and the Physiology and Pathophysiological Changes in Blood flow through Portal Vein are very important and can present in a variety of Ways and Multiple Correlated Diseases should be excluded as causes of Portal Venous Hypertension which can reduce the chances of Cavernous Formation of Portal Vein.

**Conflicts of Interests: None**

**REFERENCES**


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