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PORTAL HYPERTENSION: AN AYURVEDIC VIEW

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ABSTRACT

Portalcaval Anastomosis is a specific type of anastomosis that occurs between the veins of portal circulation and those of systemic circulation. Portal hypertension is defined as sustained elevation of portal pressure more than 12 mm Hg. The mechanism behind causation of portal hypertension are two, (1) increased intrahepatic resistance to the passage of blood flow through the liver due to cirrhosis and (2) increased splanchnic blood flow secondary to vasodilation within the splanchnic vascular bed. Portal hypertension is directly responsible for the two major complications of cirrhosis: variceal hemorrhage and ascitis. These varices develop at sites where the systemic and portal circulations have common capillary beds. The principal sites are oesophageal varices, haemorrhoides, caput medusa and retroperitoneal anastomoses. The development of oesophago-gastric varices which is frequently manifested by massive haematemesis is the most important consequence of portal hypertension. Bleeding from haemorrhoids is usually not as serious a complication as haematemesis from oesophageal varices.

Similar concept has been explained in our classics in the context of *Raktapitta*. Here an effort is made to understand the Ayurvedic concept of Portal Hypertension.

Key words: *Portal Hypertension, Raktapitta*

INTRODUCTION: Portal Hypertension is a hypertension (high blood pressure) in the hepatic portal system, which is composed of the portal vein and its branches and tributaries. The portal vein drains the blood from the abdominal part of alimentary tract, except the lower part of rectum and canal. It also receives the veins from the spleen, pancreas and gall bladder. These veins unite to form the trunk of portal vein which circulates through the liver, divides into capillary like sinusoids and finally drains into the inferior venacava via hepatic veins.

Formation of portal vein: Portal vein is formed by the union of superior mesenteric and splenic veins behind the neck of pancreas, infront of the inferior venacava, at the level of L2 vertebra.

Collateral Anastomosis between Portal and Systemic veins (Porta-Caval Anastomosis):

At the lower end of oesophagus: Oesophageal tributaries of left gastric vein (portal system) communicate with oesophageal tributaries of Hemiazygos veins. In portal obstruction these veins are distended producing oesophageal varecis. Sometimes the wall of the veins ruptures and produces emesis of blood.

At the lower end of Rectum and Anal Canal: Superior rectal vein (portal system) communicates with middle and inferior rectal vein (systemic vein) close to the pectinate line of anal canal. In portal obstruction radicles of superior rectal vein in the anal columns are distended producing internal rectal piles.

At the umbilicus: The para-umbilical vein (portal-system) Communicates with the following systemic veins.

1. **From above**-Superior epigastric and lateral thoracic veins.
2. **From below**-Superficial epigastric and inferior epigastric veins.
3. **From the sides** -posterior intercostal and lumbar veins.

In portal obstruction, the tributaries of systemic veins around the umbilicus are distended like the spokes of a wheel. This phenomenon is known as the Caput Medusa**.**

PORTAL HYPERTENSION: Portal hypertension is defined as sustained elevation of portal pressure more than 12 mm Hg[1].

Portal hypertension is caused by a combination of two simultaneously occurring hemodynamic processes.

1. Increased intrahepatic resistance to the passage of blood flow through the liver due to cirrhosis and regenerative nodules.
2. Increased splanchnic blood flow secondary to vasodilatation within the splanchnic vascular bed.

The causes of portal hypertension are usually subcategorized as prehepatic, intrahepatic and post hepatic[2].

1. **Prehepatic**: causes of portal hypertension are those affecting the portal venous system before it enters the liver. They include portal vein thrombosis and splenic vein thrombosis.

2. **Posthepatic**: causes encompass those affecting the hepatic vein and venous drainage to the heart. They include veno-occlusive disease, and chronic right sided cardiac congestion.

3. **Intrahepatic**: causes account for over 95% of cases of portal hypertension and are represented by the major forms of cirrhosis. Intrahepatic causes of portal hypertension can be further subdivided into presinusoidal, sinusoidal and postsinusoidal[3].

a) **Presinusoidal** causes include congenital hepatic fibrosis.

b) **Sinusoidal** causes are related to cirrhosis from various causes.

c) **Postsinusoidal** causes include veno-occlusive disease.

Major sequelae of the Portal Hypertension[4].

Irrespective of the mechanisms involved in the pathogenesis of portal hypertension, there are 4 major clinical consequences- Ascites, Varices, Splenomegaly and Hepatic Encephalopathy.

Varices (collateral channels or porto-systemic shunts[5])

As a result of rise in portal venous pressure and obstruction in portal circulation within or outside the liver, the blood tends to bypass the liver and return to the heart by development of porto-systemic collateral channels. These varices develop at sites where the systemic and portal circulations have common capillary beds. The principle sites are as under.

1) Oesophageal varices: The development of oesophago-gastic varices which is frequently manifested by massive haematemesis is the most important consequence of portal hypertension.

2) Haemorrhoids: Development of collaterals between the superior, middle and inferior haemorrhoidal veins resulting in haemorrhoids is another common accompaniment. Bleeding from haemorrhoids is usually not as serious a complication as haematemesis from oesophageal varices.

ACCORDING TO AYURVEDA: There are two *Gati* in *Raktapitta* for the *Pravartana* of *Dustarakta*. One is from

Amashaya i.e., *Urdhvagati* and another one is from *Pakvashaya* i.e., *Adhogati*. *Dalhana* while commenting on this says that *Yakrit* and *Pleeha* are the *Moolasthan* of *Rakta*. Hence *Rakta* will not come to *Amashaya* or *Pakvashaya* until and unless it gets vitiated by some other factors. Thus it is clear that the *Prakupita Rakta* attains *Gamana* and starts *Pravartana* from *Amashaya* i.e., *Urdhvagati* and *Pakvashaya* i.e., *Adhogati*[6].

DISCUSSION: Portal vein and its tributaries are devoid of valves. It acts as reservoir of blood and about one third of the entire volume of blood may be stored in portal system. In portal obstruction, particularly due to cirrhosis of liver, back-flow of portal blood takes place through the radicles of caval system before the blood reaches the right atrium , this is the cause for Portal hypertension which leads oesophageal varices and Internal haemorrhoids**.**

Varicose veins in the esophagus and stomach bleed easily and sometimes massively, and less commonly in the rectum. Bleeding from these veins may result to death. The same concept is present in Ayurveda as *Raktapitta*. It is basically of two types *Urdhvagata Raktapitta* and *Adhogata Raktapitta*[7], these two may be consider as Oesophageal Varices and Internal Haemorrhoids respectively.

CONCLUSION: The present study attempts to integrate the knowledge of traditional Ayurvedic concepts with the contemporary science. This may be useful for utilizing the ancient medical science in perspective. So Portal Hypertension is considered as *Raktapitta* in Ayurvedic view.

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