Intrahepatic portosystemic venous shunt (PSVS)

Intrahepatic portosystemic venous shunt (PSVS) is a rare condition defined as a communication between the intrahepatic portal vein and systemic veins, including the hepatic vein and perihepatic vein, via an anomalous intrahepatic venous channel. After the introduction of CT, MR imaging, and sonography, intrahepatic portosystemic venous shunt has been encountered more frequently.

Several appearances of intrahepatic portosystemic shunts have been described. The most frequently reported is between the right portal vein and the IVC. This type of portosystemic shunt typically occurs in the clinical setting of portal hypertension. Other appearances of intrahepatic portosystemic shunts include multiple diffuse communications between peripheral portal and hepatic veins, a single communication between a portal vein branch and a hepatic vein in one hepatic segment, and a single communication between a portal vein branch and a hepatic vein through an aneurysm.

42 cases of intrahepatic portal venous to hepatic venous shunts have been reported in the English-language literature. Portal vein aneurysms were reported in 29 (69%) of these 42 cases. Most of these cases (76%) were not associated with liver cirrhosis. Because of the low rate of coexisting liver cirrhosis and high rate of coexisting anomalies, intrahepatic portal venous–hepatic venous shunts are likely to be of the congenital origin type.

In an early stage of embryologic development, anastomoses exist between the subcardinal venous system and vitelline venous system. The right subcardinal vein becomes a part of the hepatic segment of the inferior vena cava. The vitelline vein is broken to the vitelline sinusoids, which become the intrahepatic portal vein branches and the hepatic veins. It is possible that most portosystemic venous shunts either exist or develop from embryonic vascular remnants, including the right vitelline vein and the vitelline sinusoids. A ductus venosus is one of the possible causes that may produce a communication between the left portal vein and the inferior vena cava. PSVS can develop as the intrahepatic collateral pathways in the presence of cirrhosis and portal hypertension.

On gadolinium enhanced MRI obtained during the portal venous phase, the communication between a portal vein branch and the hepatic vein can be demonstrated. Another imaging finding is an early and asymmetrical enhancement of a hepatic vein in the late arterial phase. Contrast enhanced CT can also demonstrate the abnormal communication.

Clinical manifestations of intrahepatic portosystemic venous shunt depend on the shunt flow; a highflow shunt might cause hepatic encephalopathy and hypoglycemia.

Conservative therapy (restriction of protein, ingestion of lactulose, oral administration of nonabsorbable antibiotics), surgery (portal vein ligation or hepatic lobectomy), and transcatheter embolization have been used for the treatment of intrahepatic portosystemic venous shunt.
References:

