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Portal vein abnormalities: an imaging review

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ABSTRACT

The portal vein is the main vascular channel of the liver and is affected by many pathologies. Imaging plays an important role in the detection and characterization of these abnormalities, guiding the surgeon and the interventional radiologist in planning further management. We discuss the imaging appearances of various abnormalities affecting the portal vein and the imaging modalities used in their diagnosis. We also briefly discuss the radiological interventions done in some of these cases.

1. Introduction

The portal vein (PV) is the vascular channel which supplies about 80% of blood to the liver. It receives most of its blood from bowel and spleen through the superior and inferior mesenteric veins and the splenic vein respectively. Many pathologies, both benign and malignant, may involve the PV [1]. Imaging plays an important role in the detection and characterization of these abnormalities, providing guidance to the surgeon and/or interventional radiologist in planning the treatment whenever necessary. The imaging modalities used include ultrasonography (USG) including color Doppler, computed tomography (CT), magnetic resonance imaging (MRI) and digital subtraction angiography (DSA). We review the imaging appearances of normal PV and various abnormalities affecting the PV and also briefly discuss some of the relevant radiological interventions.

2. Normal anatomy

The portal vein is formed by the union of the splenic vein (SV) and the superior mesenteric vein (SMV), the union being called portosplenic confluence. It measures 9–13 mm in diameter in adults and is about 8 cm long [2]. Distally it divides into right and left branches; the right PV further divides into anterior and posterior divisions (Fig. 1A & B) [3]. The right anterior division supplies segments V and VIII and right posterior division supplies segments VI and VII of the liver. The left PV gives branches to segments I (caudate lobe), II, III and IV. The normal blood flow is hepatopetal i.e., towards the liver.

Few tributaries drain directly into the PV, including left gastric vein and posterior superior pancreaticoduodenal vein [4]. Many sites of portosystemic shunts exist in the body which open up or dilate when there is portal hypertension. These include periesophageal, perirectal, retroperitoneal, splenic hilar, perigastric and periumbilical regions.

3. Abnormalities of the portal vein

Abnormalities of the PV are divided into congenital and acquired. Congenital abnormalities are uncommon and some of them are incidentally detected. Acquired pathologies are usually symptomatic. The diseases may affect either intrahepatic branches or extrahepatic segments of the PV or both and may additionally involve the SMV or SV. The various pathologies involving the PV are classified in Table 1.

3.1. Congenital abnormalities

3.1.1. Anatomical variations

Variations in the anatomy of the PV are seen in about 20–35% of the population [3]. Three common anatomies have been described in literature, with type I being normal (Fig. 1C) (3,5). The two common variations are trifurcation of the PV (type II), where the PV divides into right anterior, right posterior and left branches (9–10%) and early branching of the right posterior PV from the main PV (type III) (13–24%) (Fig. 1D & E) [5]. Another less common variation is early division of the right PV. Hypoplasia and absence of the right or left PV and non-branching of the PV are very rare and frequently associated with atrophy of ipsilateral hepatic lobe and hypertrophy of contralateral lobe [6]. These anatomical variations most often are

Abbreviations: LPV, Left portal vein; MPV, main portal vein; RA, right anterior; RP, right posterior; RPV, right portal vein; SMV, superior mesenteric vein; SV, splenic vein; IVC, inferior vena cava

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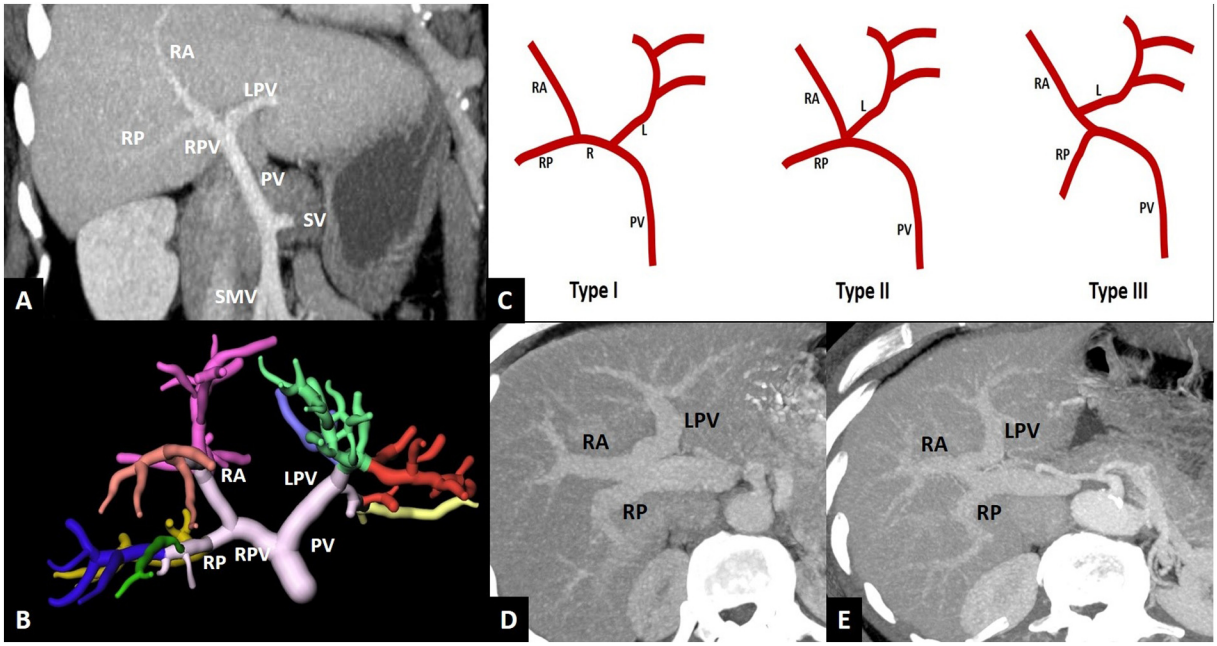

Fig. 1. A & B: Coronal maximum intensity projection (A) and volume rendered (B) CT image showing the PV and its branches. **C**: Schematic diagram showing three common types of anatomy of the PV. **D**: Type II variation. Maximum intensity projection CT image showing trifurcation of the portal vein into right posterior, right anterior and left portal veins. **E**: Type III variation. Maximum intensity projection CT image showing early origin right posterior branch from the main portal vein. LPV – Left portal vein; MPV – main portal vein; RA – right anterior; RP – right posterior; RPV – right portal vein.

Table 1
Abnormalities of portal vein

Congenital	Anatomical variations
	Abernathy malformation
	Congenital intrahepatic portosystemic shunt
Acquired	Thrombosis – bland and tumor
	Septic thrombophlebitis
	Extrahepatic portal vein obstruction
	Intraluminal gas
	Stenosis or occlusion
	Aneurysms
	Peliosis hepatis
	Arterioportal shunts
	Portosystemic shunts
Radiological interventions	Angioplasty and stenting
	Transjugular intrahepatic portosystemic shunt
	Portal vein embolization
	Balloon-occluded retrograde or antegrade transvenous obliteration (BRTO/BATO) of varices

incidentally detected and usually have little clinical significance. Their importance arises when PV embolization or hepatectomy becomes necessary and knowledge of these variations help in proper planning of the procedure.

3.1.2. *Abernethy malformation*

Abernethy malformation (also called congenital extrahepatic portosystemic shunt) is a condition where there is an extrahepatic shunt between the PV and a systemic vein, most commonly the inferior vena cava (IVC) [7]. Other systemic veins into which the shunt may drain are the renal, iliac or azygous veins or the right atrium [8]. This shunt results in complete or partial bypass of blood from the PV into the systemic circulation. The affected patients may be asymptomatic or may present with features of cardiac failure, pulmonary arterial hypertension or rarely, hepatic encephalopathy.

Abernethy malformation is classified into two types [9]. In type I there is no intrahepatic branching of the PV with complete shunting of blood from the PV into the systemic circulation (Fig. 2). This is further divided into type Ia (the SV and SMV draining separately into a systemic vein) and type Ib (common trunk of the PV draining into a systemic vein). In Type II, the shunting between the PV and the systemic circulation is partial (side to side shunt) and intrahepatic PV branches are present (Fig. 3). Congenital anomalies are more commonly associated with type I and include congenital heart and renal anomalies, gastrointestinal, skeletal and vascular anomalies [8, 10].

USG often shows a dilated main PV leading into IVC, with blood flow from the PV to the IVC on color Doppler. The SV or the PV show triphasic or biphasic spectral waveform due to transmission of pulses from the right atrium. Intrahepatic PV branches may or may not be seen depending on the type of the malformation [7, 8]. Hepatic artery is often hypertrophied. Cross sectional imaging with contrast enhanced CT scan or MRI is useful in better defining the length, location and size of the shunt, showing intrahepatic PV branches if present and demonstrating liver lesions (e.g., hyperplastic nodules with appearance similar to focal nodular hyperplasia), if any (Figs. 2 & 3) [7]. Balloon occlusion of the shunt with diagnostic angiography or shuntogram through transjugular or transfemoral routes may be performed. These images can outline the location, size, and length of the shunt and are important for planning endovascular or surgical management. In cases where imaging shows Type I anomaly, liver biopsy should be done for confirmation as demonstration of portal radicles on histology changes the diagnosis (to type II) and the management [7].

Management of patients with Type I anomaly is difficult. Staged surgical ligation of the shunt may be done with moderate success rates [11, 12]. Rarely, liver transplantation is done. In type II, as intrahepatic branches are present, the shunt can be successfully blocked either by surgical ligation or by radiological intervention using coils or vascular plugs [13].

3.1.3. *Congenital intrahepatic portosystemic shunt*

Congenital intrahepatic portosystemic shunt (CIPS) is a rare anomaly where there is intrahepatic communication between the branches of the PV and the hepatic vein [14]. Park et al., classified this anomaly into 4 types based on the number of shunts (single or multiple), the location (single or multiple segments) and the presence or absence of an aneurysm [15]. These shunts develop due to persistence