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ANATOMIC VARIATIONS

Anatomic variations: a rare variant of formation of the portal vein

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Abstract
The portal vein derives from the vitelline veins, a component of the extraembryonic venous system, and is normally formed by the confluence of the superior mesenteric and splenic vein. The knowledge of the anatomy of the portal vein and its abnormalities is important for interventional and surgical procedures. Variant portal architecture is a common finding during imaging studies. Ultrasonography, computed tomography and magnetic resonance are non-invasive methods for studying and understanding portal vein's anatomy and abnormalities. We describe a rare case of variation in the formation and course of the portal vein. To the best of our knowledge, there is no evidence of this kind of abnormality in literature.

Keywords Portal vein anomalies · Embryonic development · Computed tomography · Interventional procedures

Introduction

The portal vein (PV) is formed by the confluence of the superior mesenteric and splenic veins. Normally, the PV is anterior to the inferior vena cava, and posterior to the pancreas and to the first part of the duodenum. At the hepatic hilum the PV divides into two branches, the right portal vein (RPV) and the left portal vein (LPV). The RPV then divides into the right anterior portal vein (RAPV) and the right posterior portal vein (RPPV). The LPV carries blood to the II, III and IV segments of the liver; the RPV supplies the V, VI, VII, VIII segments. The I segment is supplied by both left and right branches.

The PV carries blood from the digestive tracts (except the lower part of the rectum and the anal canal), the spleen, the pancreas and the gallbladder [2–3].

Imaging plays an important role in the detection of portal vein abnormalities, guiding both radiological and surgical procedures [1].

Here, we describe a rare case of variation in the formation and course of the portal vein.

Case report

A 70-year-old woman was admitted to our hospital with abdominal pain. She underwent appendicectomy in the adolescence and, shortly after, resection of the terminal ileum for acute abdomen due to abdominal adhesions. She had a history of hyperlipasemia and hyperamylasemia of unknown aetiology, without specific abdominal symptoms. At admission, blood tests showed normal liver tests, otherwise confirming the known alteration of the pancreatic enzymes. An US examination of the abdomen identified vascular abnormalities of the portal venous system, and dilatation of the inferior vena cava. To acquire a comprehensive evaluation, contrast-enhanced computed tomography (CT) was carried out and performed in unenhanced, arterial, venous and delayed phases, using 64-channel scanners (Somatom Definition Dual Source, Siemens Healthcare). A nonionic contrast medium (iomeprol, 400 mgI/ml, Iomeron 400; Bracco Imaging, Milan, Italy) was used, at a dose of 2 ml/Kg.

The contrast medium was warmed to 37 °C and administered with a dual-chamber power injector (Stellant D CT;

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Medrad, Indianola, Pa) at a rate of 3 ml/s through an intravenous catheter inserted into an antecubital vein. This was followed by a 30-ml saline flush at the same injection rate.

The arterial phase scanning was determined using the bolus-tracking CT angiography technique with a threshold of 100 HU; the scan delay for venous phase scanning was 60 s. All acquisitions were obtained with an automatic exposure control system (Care Dose 4D; Siemens AG, Healthcare Sector) and slice thickness/reconstruction interval, 2 mm/1.5 mm for unenhanced and 1 mm/1 mm for the other phases.

The CT-study showed both an unusual development of the PV, hepatic veins and a variant in the position of the duodenum. The PV was located in the right upper quadrant, on the outer face of the liver. Hepatic veins had a peculiar development: three hepatic veins run on the upper outer surface of the liver, draining blood from II, III, IVa, VII and VIII segments; VI segment was drained by a single hepatic vein, which run on the outer lower surface of the right lobe; IVb and V segments were drained by a separate hepatic vein, normally developed inside liver parenchyma; first segment was drained by an independent vein. The duodenum was normally positioned anteriorly to the PV but was deployed, with the pancreas, in the right upper quadrant. The RPPV originated as the first branch of the portal vein (type 3 malformation) [1] (Figs. 1, 2, 3).

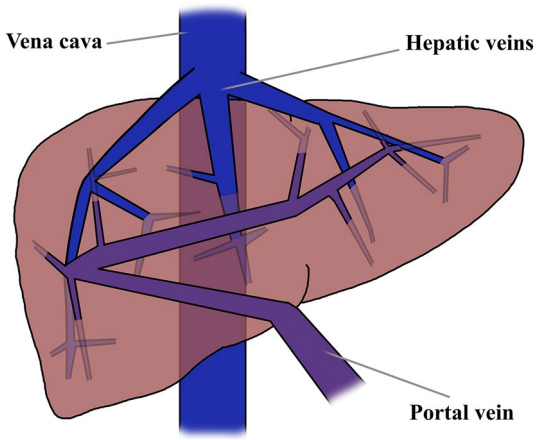

Fig. 2 Schematic drawing representing the hepatic venous system of the case patient. Portal vein runs on the right outer surface of the liver and separates into lobar branches which themselves run on the liver surface before further dividing into segmental branches, that deepen inside liver parenchyma. Hepatic veins run outside liver and divide into segmental branches, penetrating parenchyma from the outer surface

Discussion

The PV is normally formed in 4–10 weeks of embryonic development. At the third week the embryo presents the existence of three pairs of vein, the omphalomesenteric vein (also called vitelline veins, that carry blood from the yolk sac to the heart), the umbilical veins (that carry oxygenated blood from the chorionic vill of the placenta to the embryo) and the cardinal veins. Between the 4th and 12th weeks of gestation, during the development of the liver, the portal venous system arises both from the vitelline venous system and the umbilical venous system. The vitelline veins form anastomotic network around the duodenum, composed by caudal–ventral, dorsal and cranial–ventral anastomoses, that enter the septum transversum or primitive liver forming hepatic sinusoids that drain into the sinus venosus or primitive heart [4]. Then, the yolk sac disappears with vitelline veins, the caudal–ventral

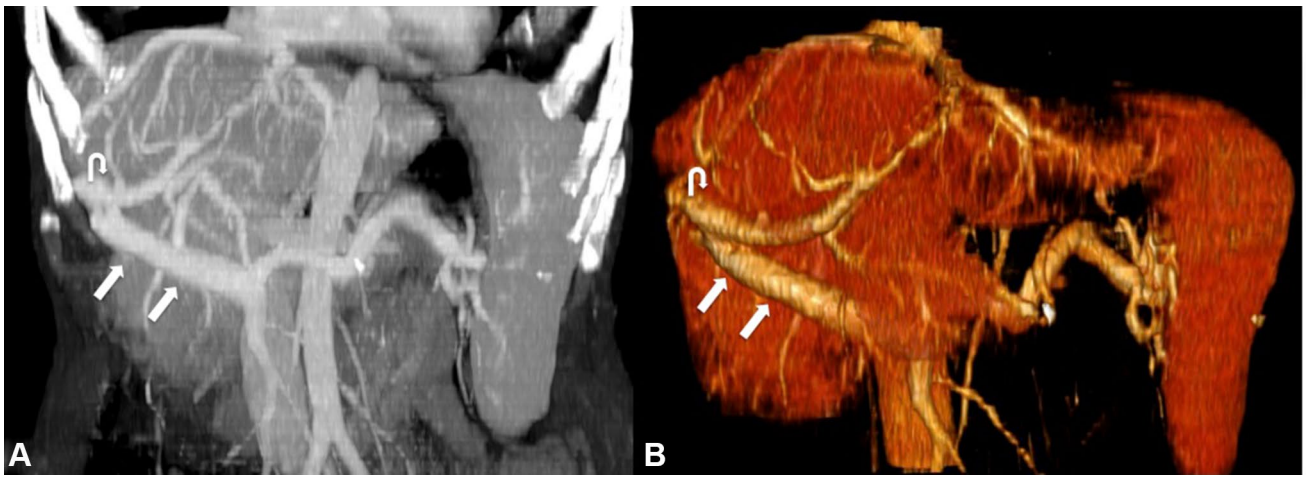

Fig. 1 MIP (**a**) and VRT (**b**) reconstruction images from the venous phase of the CT study demonstrating the route of the portal vein on the outer side of the liver (straight arrows) and the origin of the RPPV (curved arrows)

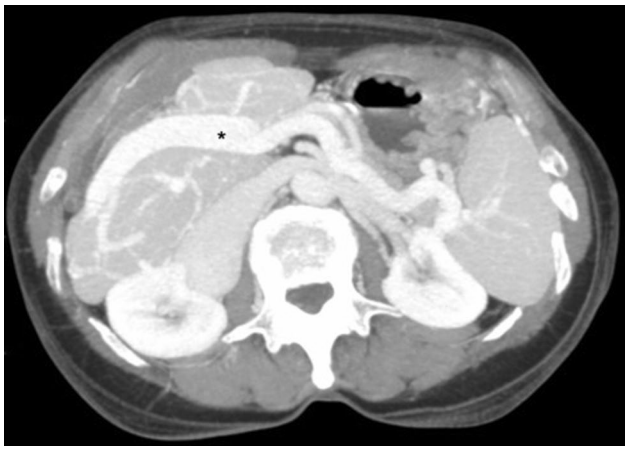

Fig. 3 MIP axial reconstruction from the venous phase of the CT study demonstrating the route of the portal vein (asterisk) from the hilum through the outer surface of the liver

anastomosis regresses and the dorsal anastomosis becomes the portal vein [3].

Probably, our patient presents an embryological disorder: in fact, we might assume that the vitelline veins did not enter the septum transversum but formed a network around the primitive liver rather than inside.

This is also conceivable because the duodenum and the pancreas were deployed in right upper quadrant even if the duodenum was normally placed anteriorly the PV. She presents, even, the type 3 malformation according to Covey's classification.

To the best of our knowledge, there is no evidence of other cases with similar vascular variations in the literature.

The comprehension of embryologic development of the portal vein is crucial to understand the imaging findings and eventual variants.

This is important for clinical management and moreover for an adequate planning of surgical, percutaneous or vascular interventional procedures.

Author contributions AS: manuscript writing. CS: clinical data collection and manuscript writing. SS: literature research and scheme drawing. FC: literature research. SC: management. AB: management and manuscript writing.

Compliance with ethical standards

Conflict of interest None of the authors have any conflicts of interest to declare.

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