

# Duplication of the portal vein and the implications for procedural planning

OL Chan \*, YS Lee, CH Ho, CC Lee, CC Cheung

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A 72-year-old man with recurrent hepatitis B virus–related hepatocellular carcinoma was referred for right portal vein embolisation (PVE) prior to right hepatectomy. He had Child-Pugh class A cirrhosis, with calculated indocyanine green–R15 of 8%. Portal embolisation was indicated due to the presence of multiple co-morbidities and marginal future liver remnant volume of 35%.

Preprocedural computed tomography revealed duplication of the portal vein (DPV) [Fig 1]. The anatomy and feasibility of the procedure was discussed with hepatic surgeons. Right PVE was successfully performed with n-butyl cyanoacrylate glue. Left hepatic lobe hypertrophy from 430 cm<sup>3</sup> to 560 cm<sup>3</sup> was achieved. The patient subsequently underwent an uneventful right hepatectomy.

Portal vein embolisation is a commonly adopted strategy to induce future liver remnant hypertrophy prior to hepatectomy. Knowledge of the portal venous anatomy and its variants is vital for treatment planning. Duplication of the portal vein is a rare congenital anomaly that has been described only in case reports. It is related to the spectrum of vitelline vein regression anomaly with pathogenesis believed to be failed regression of the left cranial part of the vitelline vein (Fig 2a-e).<sup>1</sup> A variation of DPV has been reported; some authors describe two portal veins arising separately without extrahepatic communications,<sup>2</sup> while some describe an additional portal vein arising anomalously from either the superior mesenteric vein or the splenic vein (Fig 2f-i).<sup>3</sup> The latter was evident in our patient (Fig 1).

Another anomaly with double channel portal vein is portal vein fenestration in which there is a small fenestration at the mid portion of the main portal vein.<sup>4</sup> The exact pathogenesis and its relationship with portal vein duplication remains unknown.

In the presence of DPV, there was altered flow dynamic with preferential opacification of the right or left portal vein branches depending on different catheter tip positions (Fig 3). There was preferential flow towards the left portal branches at the intrahepatic communication at the hepatic hilum, giving a narrow safety margin for embolisation to prevent non-target embolisation of the left portal vein that could jeopardise the future liver remnant.

Our patient successfully underwent PVE without complication. The degree of hypertrophy was similar to that reported in local cohorts.<sup>5</sup> Surgeons discussed whether the anomalous portal vein could be embolised to improve the efficacy of PVE but there was also a risk of jeopardising venous return from small branches of the superior mesenteric vein that may worsen liver function.

During hepatectomy, DPV was confirmed (Fig 4). It did not affect surgical planning and the patient underwent right hepatectomy uneventfully.

Duplication of the portal vein is a rare congenital anomaly. Because of the possible altered flow dynamics, it is important to identify this anomaly on preprocedural imaging and arrange multidisciplinary team discussion to plan PVE and ensure a safe and effective procedure.



FIG 1. Axial image (a), coronal maximum intensity projection image (b) and three-dimensional reconstruction image (c) from contrast computed tomography of the abdomen showing duplication of the portal vein. The anatomical portal vein (arrowheads) arises from the confluence of the superior mesenteric vein and the splenic vein with a retroduodenal and retropancreatic course. The anomalous portal vein (arrows) arises from the superior mesenteric vein with a retroduodenal prepancreatic course. There is both intrahepatic and extrahepatic communication of the duplicated portal vein

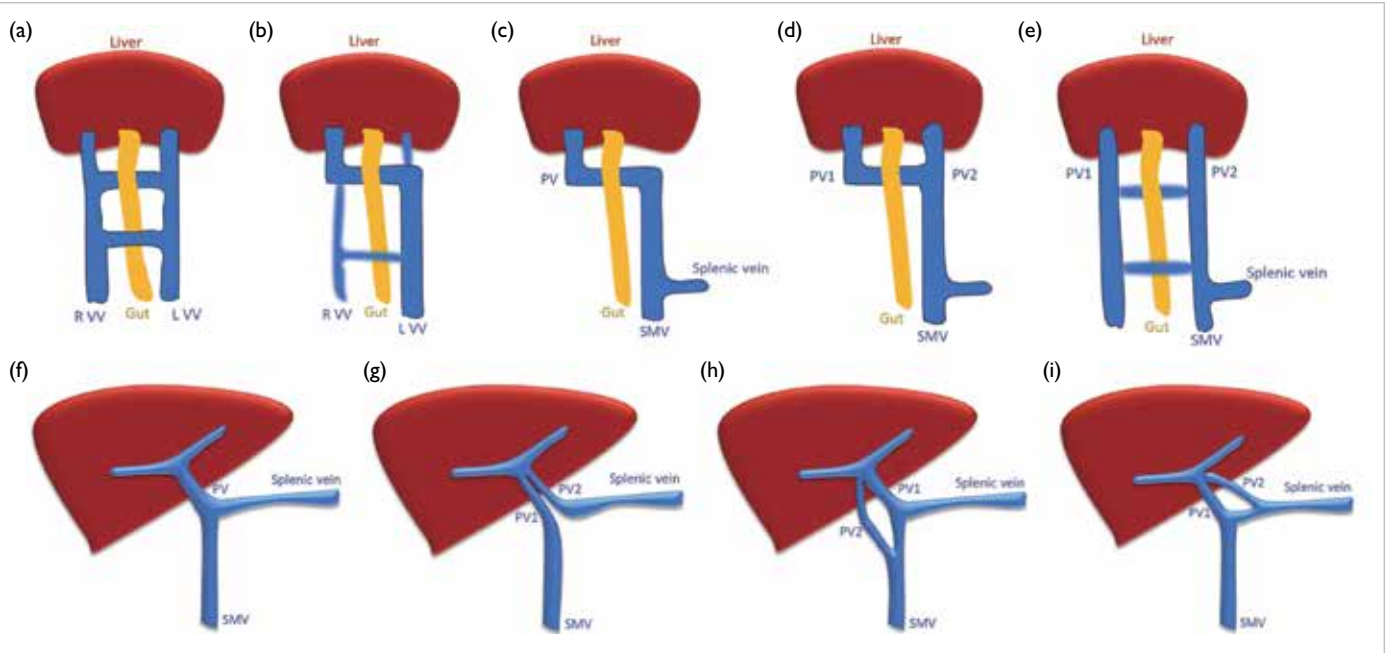


FIG 2. Schematic diagram illustrating the embryology of portal vein (PV) development and postulated pathogenesis of duplication of the portal vein (DPV), normal PV anatomy and various types of DPV described in the literature.<sup>1,3</sup> (a) The paired vitelline veins (VVs), ie, the right vitelline vein (R.V.V.) and the left vitelline vein (L.V.V.), anastomose with each other around the primitive gut. (b, c) The caudal part of the R.V.V. and the cranial part of the L.V.V. degenerate to become the PV. (d) Duplication of the portal vein (the anatomical PV [PV1] and the anomalous PV [PV2]) due to failure of the cranial part of the L.V.V. to degenerate. (e) Duplication of the portal vein due to abnormal degeneration of the VV anastomoses. (f) The normal anatomy of a PV formed by the confluence of the superior mesenteric vein and splenic vein. (g) One variant of DPV, with PV1 arising from the superior mesenteric vein and PV2 arising from the splenic vein, without extrahepatic communication. (h) Another variant of DPV with an additional PV2 arising from the superior mesenteric vein. (i) Another variant of DPV with an additional PV2 arising from the splenic vein  
Abbreviation: SMV = superior mesenteric vein

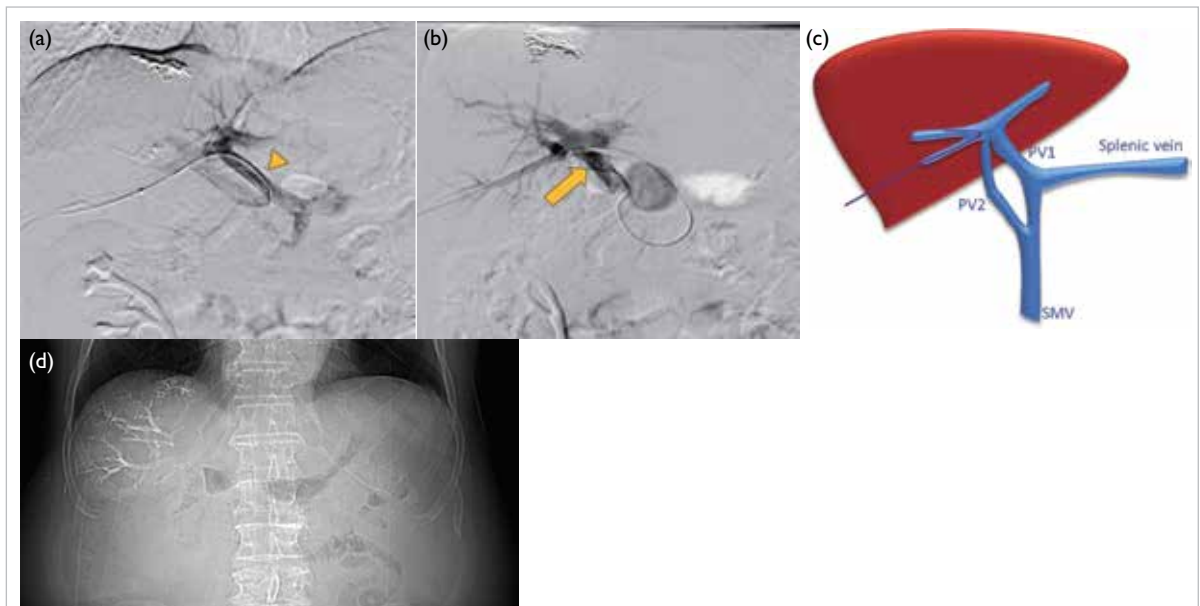


FIG 3. Right portal vein embolisation via the ipsilateral percutaneous approach. (a) An angiographic catheter was passed through the intrahepatic communication to the anatomical portal vein (arrowhead). Portography shows opacification of duplicated portal vein and left portal branches. (b) The catheter was passed through the extrahepatic communication to the anomalous portal vein (arrow). Portography shows preferential opacification of the right portal branches, the intrahepatic communication and some of the left portal branches. (c) Schematic diagram illustrating the catheter position during glue embolisation. The catheter is directed towards the right portal vein branches without bypassing the intrahepatic or extrahepatic communications of duplication of the portal vein (purple kinked line). (d) Post-portal vein embolisation. The radio-opaque branching pattern of glue cast at the right-side portal veins  
Abbreviations: PV1 = anatomical portal vein; PV2 = anomalous portal vein; SMV = superior mesenteric vein

### Author contributions

Concept or design: All authors.  
 Acquisition of data: All authors.  
 Analysis or interpretation of data: OL Chan, YS Lee.  
 Drafting of the manuscript: OL Chan, YS Lee.  
 Critical revision of the manuscript for important intellectual content: All authors.

All authors had full access to the data, contributed to the study, approved the final version for publication, and take responsibility for its accuracy and integrity.

### Conflicts of interest

All authors have disclosed no conflicts of interest.

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### Ethics approval

This study was approved by the Central Institutional Review Board of Hospital Authority, Hong Kong (Ref No.: CIRB-2023-064-1). Written informed consent was obtained from the patient for publication of this article.

<sup>1</sup> OL Chan \*, MB, BS, FRCR

<sup>1</sup> YS Lee, FRCR, FHKAM (Radiology)

<sup>1</sup> CH Ho, FRCR, FHKAM (Radiology)

<sup>2</sup> CC Lee, FRCS, FHKAM (Surgery)

<sup>2</sup> CC Cheung, FRCS, FHKAM (Surgery)

<sup>1</sup> Department of Radiology and Nuclear Medicine, Tuen Mun Hospital, Hong Kong SAR, China

<sup>2</sup> Department of Surgery, Tuen Mun Hospital, Hong Kong SAR, China

\* Corresponding author: col950@ha.org.hk

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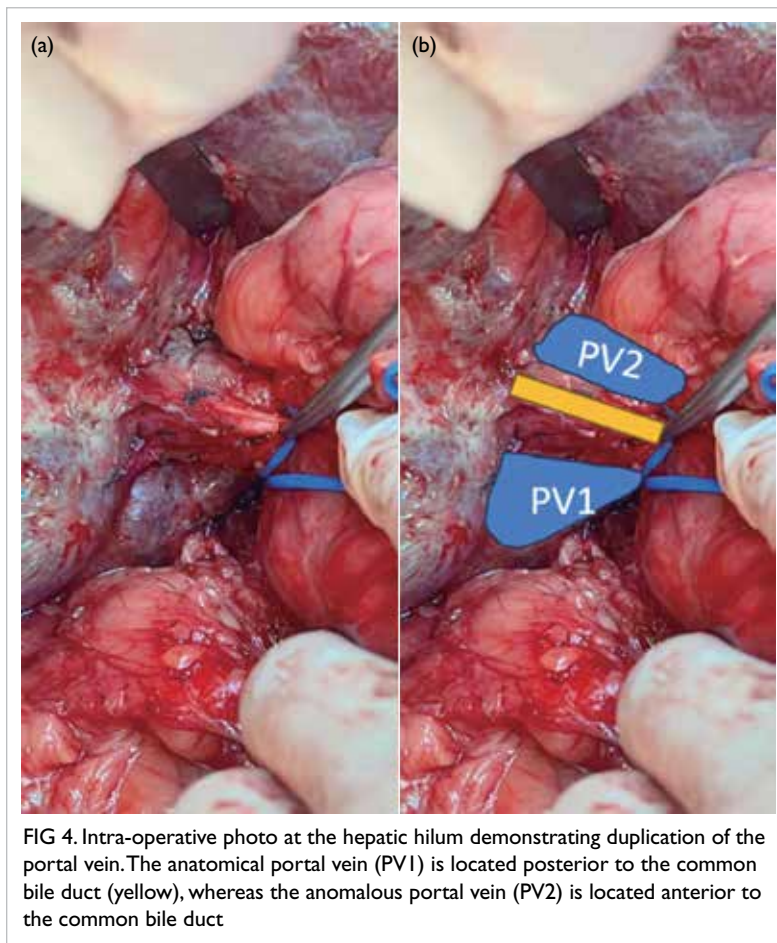


FIG 4. Intra-operative photo at the hepatic hilum demonstrating duplication of the portal vein. The anatomical portal vein (PV1) is located posterior to the common bile duct (yellow), whereas the anomalous portal vein (PV2) is located anterior to the common bile duct