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Categorization of differing types of non-tumoral portal vein thrombosis based on the location and hemodynamic changes during liver transplantation

To the Editor:

We read the paper "Novel classification of non-malignant portal vein thrombosis: A guide to surgical decision-making during liver transplantation" with great interest.[1] This is an excellent and inspiring study proposing a new tailored classification of non-tumoral portal vein thrombosis (PVT) in candidates for liver transplantation (LT), incorporating anatomic and functional parameters (collateral circulations).

In the novel classification system of non-malignant PVT proposed by Prashant Bhangui *et al.*, PVT was dichotomised into non-complex (Yerdel grade 1-3) and complex PVT (Yerdel grade 4).[1] The portal inflow reconstruction can be classified as physiological or non-physiological based on whether it could solve the problem of pre-existing prehepatic portal hypertension (PHT). Physiological portal reconstruction drains the splanchnic blood flow to the graft, thus helping to ameliorate prehepatic portal hypertension after LT. Subsequently, even in those with complex PVT, the presence of remarkable portosystemic collaterals or shunts (splenorenal shunt, enlarged left gastric vein or pericholedochal varix) could also achieve physiological portal reconstruction.[2]

The tailored surgical strategies for portal inflow reconstruction differ not only depending on the splanchnic vascular structures but also on hepatic hemodynamic changes. In whole-organ allografts, the portal vein flow (PVF) should be at least 1,000 ml/min to maintain adequate graft perfusion.[3,4] In transplant recipients with remarkable collateral vessels, ligation of the major portal systemic shunts is essential if the PVF is <1,000 ml/min, in order to prevent portal flow steal.[5]

Therefore, we are of the opinion that non-tumoral PVT can be categorized into five types according to the site, presence of patent collaterals and the graft hemodynamic status in the setting of LT (Fig. 1). The tailored classification strictly corresponds to patterns of PV inflow reconstruction and graft inflow modulation.

In type I, PVT localizes above the confluence of the splenic vein and the superior mesenteric vein (SMV), without enlarged collaterals; in such cases anatomical porto-portal anastomosis without interruption of collaterals can be performed. In type II, PVT localizes above the confluence of the splenic vein and the SMV and presents with remarkable collaterals. Interruption of the collaterals is required if portal perfusion is <1,000 ml/min. In individuals with a surgically created portosystemic shunt, patent surgically created shunts should be dismantled intra-operatively. Additionally, in the presence of a splenorenal shunt, it is also

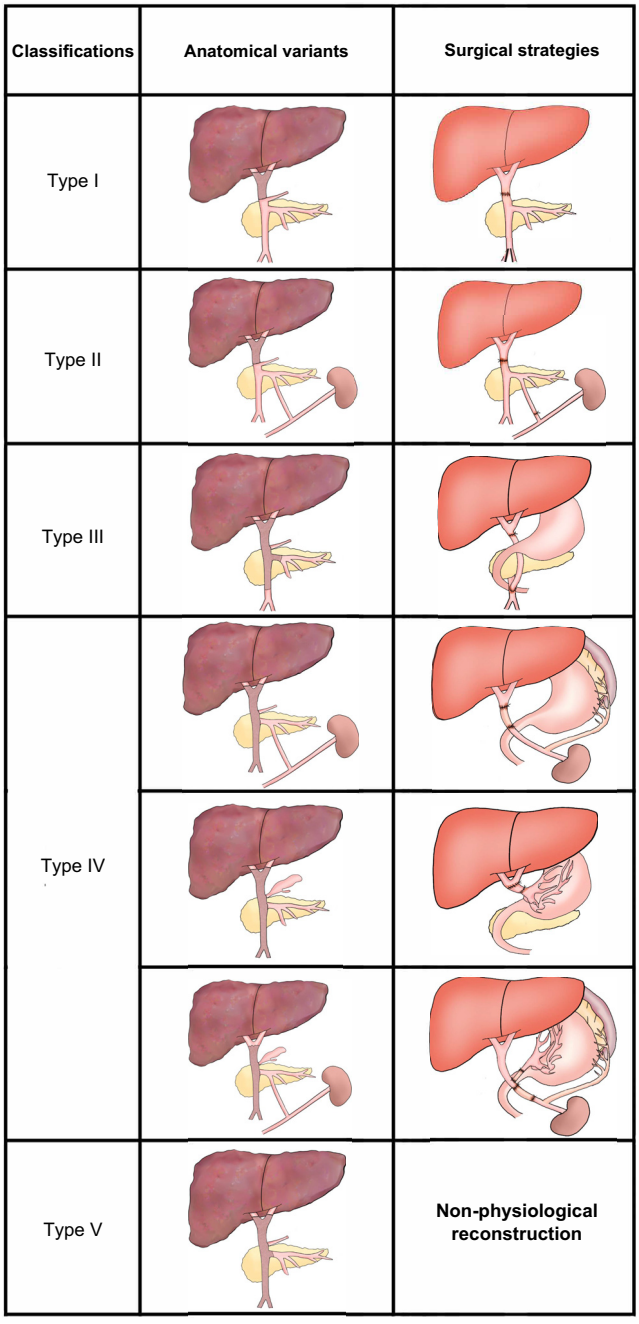

Fig. 1. Categorization of differing types of non-tumoral portal vein thrombosis and tailored technical modifications in the reconstruction of portal inflow corresponding to the anatomic patterns.

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feasible to ligate the left renal vein rather than the shunt itself because there are many collaterals in general. In type III, PVT extends below the confluence of the splenic vein and the SMV, but with a patent proximal SMV; in such cases the recipient SMV can still be used as the main portal influx source by interposing a vascular graft between the SMV and graft portal vein. This conduit is placed anterior to the pancreas and posterior to the pylorus. Interruption of the collaterals is required in the presence of a spontaneous splenorenal shunt or an enlarged gastric vein. Another surgical strategy for Type III PVT is thrombectomy with eversion followed by anatomical porto-portal anastomosis when feasible. In Type IV, PVT involves the whole SMV but with enlarged patent portosystemic shunts (either spontaneous or surgically created); these collateral vessels can be used as portal influx vessels, including renal (left renal vein), gastric (left gastric vein), peri-choledochal, and rarely dilated mesenteric vein tributaries, such as the right gastroepiploic and right and middle colic veins. In the presence of both an enlarged gastric vein and a splenorenal shunt, the prime surgical strategy is a portal inflow reconstruction using the enlarged gastric vein combined with the ligation of the left renal vein to avoid the steal phenomenon. When portal perfusion is <1,000 ml/min, a double anastomosis (such as a combined reno-portal and coronary-portal anastomosis) can be considered to ensure adequate portal inflow. In type V, PVT entails the complete thrombosis of both the porto-mesenteric and collateral veins; in such cases a non-physiological reconstruction can be considered, such as reno-portal, cavoportal, portal vein arterialisation, multivisceral transplantation and heterotopic transplantation. Usually, porto-caval hemi-transposition is used to restore adequate portal flow. In some cases, a liver-intestinal transplant will be necessary to solve the problem. In this situation, the pre-existing pre-hepatic portal hypertension will not be solved and could even be worsened by liver transplantation.

In conclusion, the proposed new categorization of PVT, incorporating the location of the splanchnic thrombosis, the extent of the collateral vessels and hemodynamic changes may allow for better preoperative planning and intraoperative tailoring of portal reconstruction and graft inflow modulation.

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Conflicts of interest

The authors have no conflicts of interest to declare.

Please refer to the accompanying ICMJE disclosure forms for further details.

Authors' contributions

SS Zheng is the guarantor. Z Yang and SS Zheng designed the study and wrote the draft. J Lerut critically revised the manuscript and provided valuable comments. All authors approved the final version of the manuscript.

Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jhepr.2022.100548.

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