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Letter to the Editor

Comment on Impact of Middle Hepatic Artery Reconstruction after Living Donor Liver Transplantation using the Left Lobe

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To the Editor;

We read the study published by Harada and colleagues with great interest.[1] The authors suggested that reconstruction of both the middle hepatic artery (MHA) and the left hepatic artery (LHA) in left lobe living donor liver transplantation (LL-LDLT) is a safe strategy that prevents biliary stricture (BS) if the graft has both LHA and MHA stumps. This has been emphasized in other studies as well.[2,3] We would like to emphasize several points regarding the results of this study.

The authors have stated that "hepatic arterial reconstruction (HAR) plays a more important role in duct-to-duct anastomosis than in hepaticojejunostomy after liver transplantation (LT) because the arterial blood supply to the anastomosis site and graft bile duct in duct-to-duct anastomosis originates only from the reconstructed HA…" in 'paragraph 3 of the introduction section. In our opinion this is an insufficient statement.[1] The arterialization of duct-to-duct anastomosis depends on both patent graft hepatic artery and also patient recipient arterial supply to the extrahepatic biliary tree. In Figure 1 of the mentioned article, it is clearly seen that RHA and MHA of the recipient was ligated in group B and BS was common. This is not a surprise because the recipient biliary system received an arterial supply from the capillaries from pancreaticoduodenal artery of the gastroduodenal arterial trunk which is not adequate. We are a center of excellence in living donor liver transplantation (LDLT) and we perform 300 cases annually. Currently, we are preparing a study evaluating the preservation of the biliary supply during HAR and we have found that biliary complications are significantly reduced when the vascular supply of the biliary tract is preserved (unpublished data).

In Group B, left lobe liver grafts had dual arterial supply and the authors have anastomosed the dominant artery. Once the pulsatile backflow was observed from the accessory artery and it was ligated. In addition, segment IV arterial flow in the hepatic parenchyma was determined by Doppler ultrasonography. We would like to as the authors whether the intraparenchymal arterial network was thrombosed in the late period following LDLT? Why didn't the authors attribute the intraparenchymal biliary abscess to

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biliary ischemia in segment IV. It is clear that segment Iva is perfused through MHA in many cases.[4] Histopathological analysis of the circumferential biliary duct biopsy following HAR could give an idea regarding the arterial supply of the biliary ducts. Furthermore, the effects of MHA ligation can be evaluated by obtaining biopsies in the late period from segment IV parenchyma.

HAR in left lobe liver transplants is technically challenging. The bulk of the left lobe liver grafts hinder the recipient hepatic arteries during HAR. Often it is necessary to make HAR before portal vein anastomosis and graft perfusion because it is technically easier. HAR using operative microscope is usually technically difficult. In another study of the same group performing double anastomoses increased the operative times by an average of 80 minutes compared to single anastomoses.[2] We prefer to anastomose the left hepatic artery to common hepatic artery of the recipient and we ligate the accessory artery once we observe the pulsatile backflow. Recipient bile ducts receive the arterial supply from the gastroduodenal artery, proper hepatic artery and the right hepatic artery. If the previous studies of the authors are analyzed, it is obvious that gastroduodenal axis supplies the extrahepatic biliary tree of the recipient but not the graft.[2,3] We have seen that authors have used gastroduodenal artery and left gastric artery (without common hepatic artery connection) for HAR of left lobe liver grafts. We believe that this reconstruction cannot supply the liver adequately.

The studies show that early biliary leaks play an important role in consequent biliary stricture. Biliary ischemia initially manifests itself as biliary leak but in this study the biliary leak rates of all three groups are simila.[1] We believe that this is due to a lack of consensus in definition of biliary leaks and strictures. On the other hand, biliary stricture observed in this study may be caused by other reasons involving technical aspects.

Disclosures

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

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