Editorial

Filling the diagnostic gap in follow-up after liver transplantation

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Liver transplantation has long evolved from an experimental procedure to a standard therapeutic option in patients suffering from end-stage liver disease, hepatocellular carcinoma or acute liver failure (1). Outcome after transplantation has considerably improved over the last decades. However, whereas early (<90 days) survival has improved, late survival (>3 years) has not changed by the same magnitude (2,3). The increase in early survival is most likely due to improved surgical technique and perioperative management. Late survival in many cases is not impaired by liver disease but cardiovascular diseases and de novo malignancy. This becomes even more important as the prevalence of the metabolic syndrome rises and reinfection of the graft with Hepatitis B or C Virus is no longer of concern.

There is, however, a significant proportion of patients that experience graft loss or death in the "intermediate time" that is not the perioperative phase nor long-term. The magnitude of this group may comprise around 10% of the patients transplanted. During this intermediate period survival is often limited by liver disease. Thus, for the hepatologist it is important to follow patients in the intermediate post-transplant time to early detect patients at risk. Reasons for graft loss or death are specific procedure related such as rejection, biliary anastomosis stenosis or hepatic artery stenosis. The presence of these conditions can typically be detected by routine laboratory and imaging follow-up. Some patients, however, will develop hepatic injury or portal vein anastomosis stenosis that leads to the sequalae of chronic liver disease such as ascites, hepatic encephalopathy or hepatorenal syndrome. Many of these conditions are characterized by an increase in portal venous pressure and will likely be missed by laboratory or imaging follow-up in early stages. The gold-standard to determine the portal pressure is the invasive measurement of the hepatic-venous pressure gradient. Because of the invasive nature of the method, its value as a screening method is limited (4,5).

Transient elastographie has been long evaluated as a useful tool in the assessment of chronic liver disease, markedly for the non-invasive determination of the stage of fibrosis and thus as a predictor to develop complications such as ascites or esophageal varices (6). The method has also been applied to determine spleen stiffness as an indirect marker of portal hypertension. Furthermore, there is a growing body of evidence showing that a reduction in spleen stiffness following liver transplantation truly reflects a decrease in portal hypertension (7,8).

In their present study, Friedrich *et al.* (9) have taken that idea one important step further. In their study, they could demonstrate that an elevated spleen stiffness 3 months post liver transplantation does identify patients that will develop clinical apparent disease many months later. This finding is even more important as an increase in spleen stiffness even translated into decreased survival. More importantly, the authors could even demonstrate that an increase in spleen stiffness selectively identified patients that developed portal hypertension as opposed to those that suffered from biliary strictures or acute rejection episodes. Given this information, an easily available and non-invasive method could be implemented into routine follow up after liver transplantation and fill a diagnostic gap to identify patients that will develop severe liver disease related complications on a mid-term time scale.

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