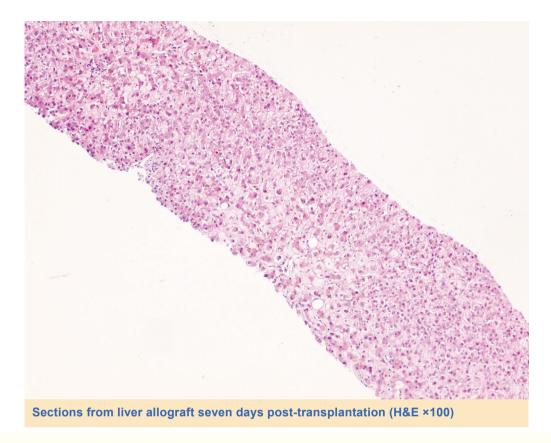
Liver Injury in a Liver Transplanted Patient

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five-year-old patient, known case of cirrhosis secondary to tyrosinemia underwent liver transplantation. Liver of the deceased donor—a 20-year-old man who had sustained head injury in a motor cycle accident—was transferred from another city. The recipient received a segment of the donor's liver after split liver transplantation. Postoperative period was uneventful. However, seven days post-transplantation, the liver enzymes were still high (ALT: 250 IU/L and ALT: 320 IU/L). Histopathologic examination of the transplanted liver biopsy is shown in the above photomicrograph. The patient improved after two weeks and discharged from the hospital in good condition.

WHAT IS YOUR DIAGNOSIS?

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DIAGNOSIS: PRESERVATION/REPERFUSION INJURY

dvances in organ preservation have reduced preservation injury. Nevertheless, when storage time exceeds 10 to 12 hours, post-transplantation complications, due to preservation/reperfusion injury, become more common [1].

Ischemic injury to the graft is divided into cold ischemia—secondary to prolonged preservation—and warm ischemia, which occurs during implantation. While the former type causes endothelial injury, the latter mostly causes hepatocyte damage [2].

Preservation/reperfusion injury occurs during the first two weeks of the operation [3]. The contributing factors to this injury are related to hepatocytes, sinusoidal cells and bile duct factors [1]. Histologic features include hepatocyte ballooning, spotty necrosis, neutrophilic aggregates and cholestasis—which are mostly marked in the perivenular region for the higher susceptibility of this area to ischemic injury [4].

This diagnosis should be considered in all patients with elevated liver enzymes during the first weeks post-transplantation and can be distinguished by the pathologist in the biopsy from the cholestasis of acute rejection, bile duct obstruction, drug toxicity and sepsis [4]. Cholestasis accompanied by portal edema and ductular reaction should prompt assessment of the biliary anastomosis and is indicative of bile

duct obstruction. The distinctive pattern of bile ductular cholestasis is that it is usually associated with sepsis. Drug toxicity can mimic every change in the liver and should always be excluded [5].

In our patient, prolonged cold ischemic time (transfer of the liver from another city) and probably small for size graft (split liver transplantation) were predisposing factors.

Functional cholestasis is a reversible disorder which can be diagnosed by biopsy and should always kept in mind in patients with poor primary graft function.

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