

Question 22 – January 8

A 65 year old male with a history of simultaneous liver kidney transplantation for hepatitis C and alcohol-induced cirrhosis 3 years ago is noted to have elevated aminotransferases on routine follow up. He failed pretransplant treatment for Hepatitis C with pegylated interferon and ribavirin, which was stopped early due to severe depression. His AST is 110 U/L and ALT 105 U/L. Bilirubin and alkaline phosphatase are normal. He denies recurrent alcohol use and is compliant with his immunosuppression, which includes tacrolimus 3mg twice daily and mycophenolate mofetil 500mg BID. A recent 12-hour tacrolimus trough level is 4.6 mg/dL (therapeutic goal 3-5 mg/dL). Liver ultrasound with Doppler shows normal hepatic parenchyma, patent vasculature and no biliary ductal dilation. A liver biopsy is performed. Which of the following pathologic findings is most consistent with the cause of his aminotransferase elevation?

- A. Bile duct lymphocytic infiltration and venous endothelial inflammation
- B. Lymphoplasmacytic portal inflammation and interface hepatitis
- C. Mononuclear portal inflammation and acidophilic bodies
- D. Bile ductular proliferation and neutrophilic pericholangitis
- E. Steatosis and hepatocyte ballooning

Answer: C

The clinical presentation is consistent with recurrent HCV in the allograft. Histologic features favoring HCV (over acute rejection) are predominant mononuclear portal based infiltrate, minimal to mild bile duct injury, lack of endotheliitis (venous endothelial inflammation) and presence of acidophilic bodies. Lymphoplasmacytic inflammation and interface hepatitis may be seen post-transplant in recurrent autoimmune or de novo alloimmune hepatitis. Bile ductular proliferation and neutrophilic pericholangitis may be seen early after transplant due to preservation injury of the graft. Steatosis and hepatocyte ballooning are consistent with steatohepatitis, which in this patient would suggest recurrent alcohol use.

Reference

Banff Working Group. Liver biopsy interpretation for causes of late liver allograft dysfunction. *Hepatology*. 2006 Aug;44(2):489-501.