

Introduction

Direct-acting antiviral (DAA) therapy has made it possible to include HCV+ donors to the organ donor pool. Cholestatic hepatitis post-transplant is quite rare with HCV viremia. We present a rare case of fibrosing cholestatic hepatitis appearing in an HCV+ donor liver.

Case Description

A 48-year-old female with history of end stage liver disease due to alcohol abuse underwent deceased donor, orthotopic liver transplant on 2/22/22 (HCV NAT +). Patient did well post operatively with no complications. However, she continued to have up-trending liver enzymes along with elevated bilirubin without any ductal abnormalities on imaging. She subsequently underwent liver biopsy on 3/02/22, showing sinusoidal pressure of 8mmHg indicating no obstruction, though the tissue histology revealed moderate acute cellular rejection, for which she received pulse dose solumedrol with improving LFTs. However, her LFTs started to rise again in a cholestatic pattern and therefore she underwent a repeat liver biopsy on 3/8/2022 which revealed no evidence of rejection but showed bile duct injury with cholestasis. She was started on ursodiol as a result. Considering the donor was HCV+, Hepatitis C RNA, PCR was done on 3/1/22 which was >100000000 IU/mL, at this point patient was started on EPCLUSA (sofosbuvir-velpatasvir). Her repeat viral PCR subsequently showed continued improvement and became undetectable on 4/27/2022. Of note, her LFTs showed significant improvement with the continued use of antiviral therapy. Patient remained stable throughout her post operative course and did undergo repeat liver biopsy twice thereafter on 3/22/22 and 6/8/22 showing no acute cellular rejection, though it showed bile ductular proliferation with mixed inflammatory infiltrate. Table-1 shows the trend in LFTs, and HCV viral load seen post operatively.

Discussion

DAA has proven to be pivotal in helping meet the ever-growing demand for organ donors. The safety of HCV+ liver donor is well established, though developing fibrosing cholestatic hepatitis 2-4 weeks post-transplant is a rare possibility. Our case (see Figure-1), shows portal inflammation and cholestasis; is a solid example of such pathology. In the absence of anatomic complication and acute cellular rejection, prompt testing for viral load should be employed. Thus, timely initiation of DAA can lead to improved clinical outcomes as seen above. Further long-term studies are needed to establish treatment protocols in patients with cholestatic type injury in seropositive donors.

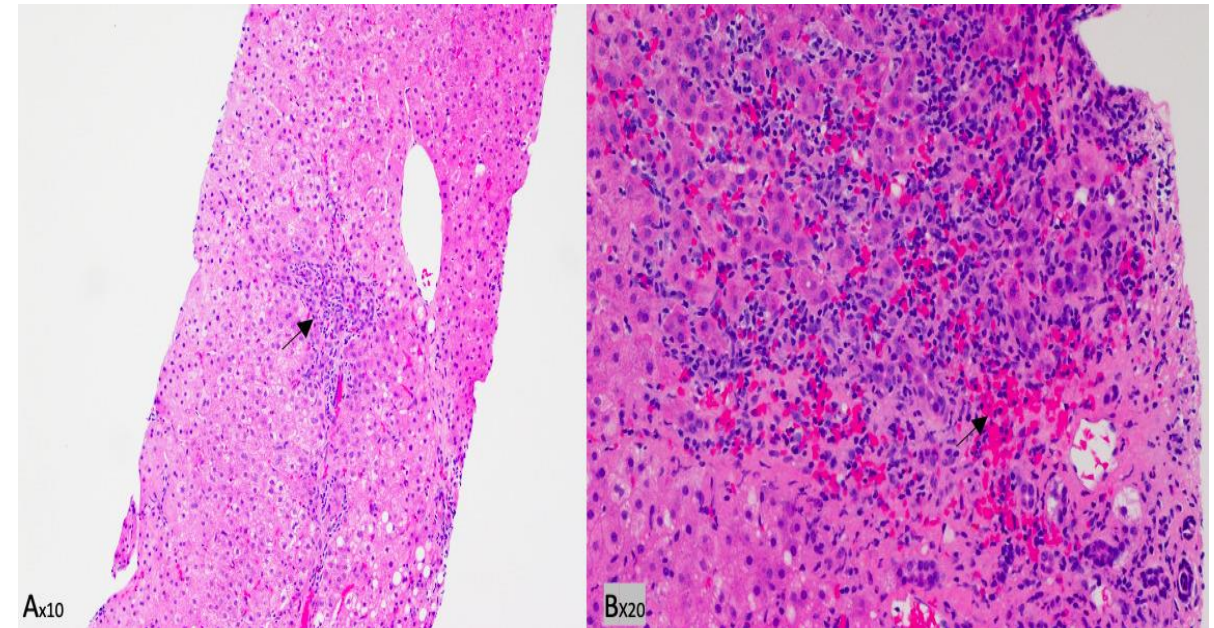


Figure-1:(H&E) A: Arrow pointing at portal tract and ductular reaction which is not diagnostic of acute cellular rejection. B: Arrow pointing towards pigment deposition showing intrahepatic cholestasis.

Table-1:Trend in LFTs and HCV RNA viral load over hospital course following deceased donor HCV+ OLT

Date	Hepatitis C Virus RNA PCR IU/mL	Total Bilirubin mg/dL	Alkaline Phosphatase U/L	AST U/L	ALT U/L
3/1/20222	100000000	13	480	110	101
3/7/2022	2310000	5.8	438	91	80
3/28/2022	248	2.4	285	132	176
4/27/2022	0	0.7	80	23	17

References

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