Leucocytosis before liver transplant, source could be hiding in heart: Case report

Sir,

Liver transplant (LT) for end-stage liver disease (ESLD) requires investigations to rule out infection. Infective endocarditis after solid organ transplant is rare. We present a case of a 47-year-old male with diabetes mellitus and ethanol-related chronic liver disease operated upon for living donor LT with high total leucocyte count (TLC). Immediate preoperative investigations including his preoperative cardiovascular evaluation with transthoracic echocardiography (TTE) were not suggestive of any infection and were unremarkable. The patient was diagnosed with aortic valve vegetation after LT.

The patient developed spontaneous bacterial peritonitis (SBP) while waiting for LT. While ascites and SBP were treated, blood culture grew coagulase-negative *Staphylococcus aureus* and *Staphylococcus haemolyticus*. His TLC was 19,000 cells/mm^3. Appropriate culture-based antibiotics were started and subsequent blood cultures were negative for bacterial growth, but TLC remained high (15,000 cells/mm^3). Haematology workup was done, but no explanation could be found for persistent high TLC in the absence of any other tests suggestive of infection. Antibiotics were stopped more than a week before surgery, and repeat blood and body fluids' cultures, done as protocol 48 h before LT, were negative for bacterial growth.

In view of deteriorating clinical condition, the patient was taken up for LT. Surgery was uneventful. Immunosupression was initiated. On postoperative day (POD) 2, the patient developed atrial fibrillation with respiratory distress requiring endotracheal intubation and mechanical ventilation. Chest X-ray was suggestive of fluid overload. Post-LT heart failure[1] was ruled out. Antibiotics were upgraded and he was weaned off mechanical ventilation on POD 7. On POD 9, he developed *de novo* diastolic murmur. TTE demonstrated aortic regurgitation (AR). Mobile aortic valve vegetation was diagnosed upon transesophageal echocardiography (TEE) [Figure 1]. Blood cultures again grew *Staphylococcus haemolyticus*. An extended course of antibiotic was planned and continued. The patient gradually improved and was discharged home. His AR persists and he has been advised surgical management.

ESLD, long-standing DM, and chronic alcohol intake are all known to cause immune suppression.[2] It is therefore imperative to rule out any infection, in a patient posted for LT. Leucocytosis seen in infection, allergic reaction, and inflammation may also be due to malignant or hereditary causes and due to *Clostridium*

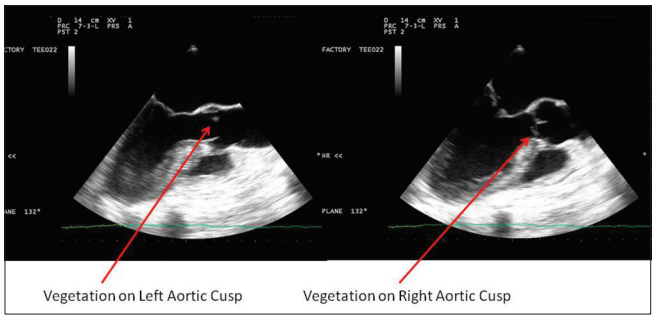

Figure 1: Long axis view of aortic valve with vegetation on aortic valve cusps

difficile infection and were ruled out. Imaging studies are done to plan surgery and to rule out any focus of infection when indicated. While TTE is routinely done for preoperative cardiology workup, TEE is done only upon suspicious finding upon TTE and was not done for our patient.

Our patient was diagnosed with alcohol-induced ESLD, and literature suggests leucocytosis of 15,000–18,000 cells/mm^3 in alcohol induced liver disease.[3] While other plausible explanations were systematically ruled out, alcohol etiology for ESLD was considered as the probable cause for the observed leucocytosis.

In ESLD, architectural aberrations in the extracellular matrix are seen in the myocardium as part of cirrhotic cardiomyopathy which in the presence of high shear stress generated by state of high cardiac output makes the heart susceptible to infections. Intrapulmonary vasodilatations are known to occur in patients with ESLD[4] and may allow microbial seeding of the left heart.

Persistently high TLC before surgery could possibly have been due to infected intracardiac vegetation that must have been too small to be detected upon preoperative TTE and to be suspected in the absence of AR. Undiagnosed preoperative infective endocarditis (IE) is also likely because bacteria consistent with diagnosis of IE were isolated from preoperative blood cultures. While antibiotics administered for positive blood culture probably checked growth and clinical manifestation of the IE before LT, in the postoperative period, pharmacological immune suppression must have allowed microbial growth which caused AR. Literature also suggests infective endocarditis in recipients of dead donor liver graft, though not applicable to our patient.[5]

Whether IE was present before LT or it occurred after surgery, the exact clinical course can only be speculated. However, in view of this case report, and due to high mortality associated with the diagnosis, the authors suggest a closer examination of heart with TEE, as part of preoperative examination before LT in patients with unexplained leucocytosis and to be aware of such possibility in the postoperative period after liver transplant.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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