A case of granulomatous hepatitis due to tuberculosis in a renal allograft recipient

We report a case of an Asian male renal allograft recipient who presented with acute liver failure. He had a fulminant course and succumbed to his illness. His postmortem study showed caseating granulomas in liver suggestive of tuberculosis (TB). Rest of organ systems did not show any evidence of TB. Miliary TB can have a rapid deteriorating course, but TB affecting only liver and causing liver failure sparing other organs has not been described.

Key words: Acute liver failure, granulomatous hepatitis, renal allograft recipient, tuberculosis

INTRODUCTION

Most cases of acute liver failure (ALF) are a result of viral infection or drug toxicity. Early diagnosis of etiology of ALF is crucial for management of the patient and for the institution of specific therapy whenever available. Tuberculosis (TB) is a rare cause of ALF. In areas where TB is endemic, it is to be considered in the differential diagnosis in cases presenting with unexplained ALF.

CASE REPORT

A 47-year-old male renal allograft recipient presented 6 years posttransplant with 7 days history of fever and jaundice. His immunosuppression regimen was cyclosporine, azathioprine, and steroids. His drug doses and regimen remained unchanged over last 2 years. On examination, patient had altered sensorium and icterus at the time of presentation.

Laboratory reports showed a hemoglobin 12.3 g/L, total leukocyte count 2.7 × 10⁹/L and platelet count of 92 × 10⁹/L. Serum total protein 55 g/L, serum albumin 23 g/L, serum glutamic oxaloacetic transaminase 961 IU/L, serum glutamic-pyruvic transaminase 221 IU/L, total bilirubin 323 µmol/L, direct bilirubin 221 µmol/L, blood urea nitrogen 13.2 mmol/L, serum creatinine 120 µmol/L, serum sodium 133 mmol/L, serum potassium 3.6 mmol/L, prothrombin time 25.1s, and international normalized ratio 2.13. Chest X-ray was normal. Ultrasound abdomen showed mild hepatomegaly.

Workup for malaria, leptospira, hepatitis A, B, C, and E, and cytomegalovirus were negative. Blood cultures were sterile. Diagnosed as ALF, patient was treated empirically with antimalarials and antibiotics.

Over next 2 days, he developed disorientation to place and aggressive behavior. He had asterixis, muscular rigidity, brisk deep tendon reflexes, and babinski sign. Patient needed ventilatory support in view of his deteriorating sensorium. He succumbed to the illness before etiology of liver failure was established.

Postmortem examination showed multiple caseating granulomas in the liver [Figures 1 and 2]. Postmortem of rest of the organs, including lungs and brain was normal.

DISCUSSION

We believe that this patient died of ALF and granulomatous hepatitis of tubercular etiology for a number of reasons. Viral hepatitis has been ruled out as much as possible though a possibility...
of other non A, non B, non C, and non E virus cannot be ruled out. In viral hepatitis, we would expect postmortem to show necrosis of hepatocytes. Moreover, presence of granulomas in the liver goes against viral hepatitis as a possible etiology. Patient's drug history remained unchanged over a period of time and so drug-induced hepatitis is unlikely. Postmortem did not show any evidence of biliary obstruction or malignant infiltration. However, we could not demonstrate acid fast bacilli by Ziehl–Nielsen staining.

Though liver involvement as a part of systemic involvement due to miliary tuberculosis is described,[2‑5] granulomatous hepatitis due to TB causing acute liver and sparing other organ systems has not been described in literature. As far as we are aware this is first such case in a transplant recipient to be reported.

Most cases of TB in kidney transplant patients occur as a result of reactivation of quiescent foci of Mycobacterium tuberculosis that persist after initial asymptomatic infection. The increased risk of TB is the result of iatrogenic immunosuppression caused by posttransplant medications such as steroids, calcineurin inhibitors, and antiproliferative agents.[6]

Incidence of TB in renal transplant recipients in this part of the continent is 12.3%. Given such a large incidence it is important to consider TB as a differential diagnosis in patients presenting with unexplained ALF in endemic areas.

REFERENCES

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