Reversible Segmental Portal Hypertension - An Unusual Presentation of Abdominal Tuberculosis in a Renal Transplant Recipient

PP Varma*, AK Seth**, RSV Kumar*

Abstract

Infections are the commonest cause of morbidity and mortality in renal transplant recipients. In India, tuberculosis is one such common infection in these patients and presents with protean manifestations. We report here a case of pyrexia of unknown origin (PUO) and segmental portal hypertension in a renal transplant recipient. Search for the cause of portal hypertension revealed abdominal tubercular lymphadenitis. Treatment with anti-tubercular therapy caused regression of segmental portal hypertension.

INTRODUCTION

In Indian scenario, tuberculosis is a common infection in renal transplant recipients, with a reported incidence of 10-15%.1,2 Besides the lung, which is the commonest site affected, involvement of unusual sites e.g. bone, skin, etc. is not uncommon.1 Segmental or sinistral or left-sided portal hypertension occurs due to compression or infiltration of the splenopetal venous axis because of malignant infiltration, peripancreatic lymphadenitis or chronic pancreatitis. To the best of our knowledge, no case of segmental portal hypertension due to tuberculosis has been reported in renal transplant recipient.

CASE REPORT

A 45 year aged lady received renal transplant from her haplo-matched brother in April, 1994. Her basic disease was benign nephrosclerosis. Post-transplant period was uneventful. After a year of triple-drug immunosuppressive therapy, cyclosporine was withdrawn and she was continued on azathioprine and prednisolone. She never had a rejection episode and basal creatinine was 0.7-1 mg/dl.

In August, 1998, she presented with low-grade fever of four weeks duration. Clinical evaluation was as unremarkable. Investigations revealed: ESR of 23 mm fall in 1st hour, hemoglobin of 11.5 gm/dl with normal differential counts. Urinalysis, liver and renal function tests were normal. USG showed multiple lymph nodes in the peri-pancreatic region causing compression of main portal vein and distal splenic vein. The proximal splenic vein was dilated (12 mm) at the hilum with evidence of splenomegaly (14 mm) and multiple collateral vessels at the splenic hilum. She was HBsAg, anti-HCV and HIV negative. Liver biopsy showed normal histology and upper GI endoscopy did not reveal any varices. A CT-guided lymph node needle biopsy showed granulomatous inflammation with Langhan’s giant cells. However, stain for AFB was negative.

She was started on four-drug anti-tubercular therapy, rifampin, isoniazid, ethambutol, and pyrazinamide in appropriate doses, for a period of 12 months. Patient showed good response to therapy as apparent by gain in weight by 4 kg and subsidence of fever over next 4-6 weeks.

An USG of abdomen did not show any change after five months of therapy, however repeat study after completion of therapy showed complete regression of peri-pancreatic lymph nodes and normalization of the spleen size (9.5 cm) and splenic vein (6 mm), though few collaterals were visualised at the splenic hilum.

DISCUSSION

Tuberculosis remains a common infection in renal allograft recipients with an incidence of 8-15%.1,2 In developed countries disseminated and atypical mycobacterial infections are common, however in India typical mycobacterium is the usual offending organism and lung is the commonest site involved.

All types of abdominal tuberculosis: peritoneal, intestinal and lymphnodal have been encountered in these patients. The common lymph nodes involved in abdominal tuberculosis are mesenteric and ileocaecal groups. Enlarged lymph nodes at the porta hepatitis may compress the main
portal vein resulting in extra-hepatic portal hypertension. Recently, it has been reported that all features of portal hypertension may regress with antitubercular therapy. However reports of segmental portal hypertension due to peri-pancreatic lymphadenitis are rare. Other rare mechanisms of portal hypertension in tuberculosis are hepatic tuberculosis causing sinusoidal compression and hepatic outflow obstruction due to tubercular constrictive pericarditis. Duration of treatment has remained controversial. Through CDC recommends the duration of treatment to be no different from normal population but most of transplant physicians give treatment for longer period varying from 9-24 months. One has to keep in mind that in patients receiving rifampin, dose of cyclosporine has to be increased as per the blood levels since this induces the hepatic cytochrome 450 system and hence lowers the cyclosporin levels. The case report highlights that in a renal transplant recipient, in a setting of segmental portal hypertension, abdominal tuberculosis should be kept in mind.

REFERENCES