Tubercular constrictive pericarditis after renal transplantation

Sir,

Constrictive pericarditis (CP) is reported in 2.4% of renal recipients.[1] A 40-year-old teacher, with presumed chronic interstitial nephritis underwent transplantation in 2004 (donor-6 antigen matched brother). Immunosuppression was mycophenolate mofetil (MMF), cyclosporine and steroids till 2012; MMF and steroids later. There were no rejections; serum creatinine was 0.8 mg/dl.

Two months after a febrile illness she noticed exhaustion, pedal edema and 6 kg weight gain. She had no pallor, was afebrile, BP was 100/70 mmHg. Heart sounds were muffled, jugular venous pressure elevated and there was hepatomegaly. Investigations showed creatinine 1.13 mg/dl, trace proteinuria without active sediments, hypoalbuminemia (2.1 g/dl). Clinical suspicion of CP was confirmed by cardiomegaly on X-ray, pericardial thickening on echocardiogram, 6 mm effusion on computed tomography scan with mediastinal lymphadenopathy.

Pericardiectomy tissue was TB – polymerase chain reaction (PCR) positive; histology showed caseating necrosis [Figure 1]. She received anti-tubercular treatment (ATT) (isoniazid, rifampicin, ethambutol, pyrazinamide); MMF was changed to azathioprine. After 3 months, she was asymptomatic with normal graft and liver functions.

Chronic CP, a sequelae of healing pericarditis, obliterates pericardial cavity, interferes with ventricular filling and cardiac output. TB remains common etiology, especially in developing countries.[2] Clinical features are weakness, cachexia; edema and ascites. Examination findings are feeble apical pulse, muffled heart sounds, distended neck veins, hepatomegaly and jaundice. Electrocardiography displays low voltage complexes, flattened or inverted T-waves and atrial fibrillation. X-ray demonstrates cardiomegaly and pericardial calcification. Echocardiogram shows pericardial thickening and effusion, distended inferior vena cava and hepatic veins, left shift of the ventricular septum during inspiration. Pericardiectomy relieves constriction; operative mortality is 5–10%; histopathology may reveal the etiology.

In patients with chronic kidney disease, uremia or effusion due to under-dialysis, hypoalbuminemia or volume overload contribute to pericarditis. TB remains a differential diagnosis. However, CP is rare.

Tuberculosis occurs in 10–13% of renal recipients.[3] CP is reported in about 2.4%.[1] Calcineurin inhibitors/sirolimus and oppurtunistic infections should be considered as etiologies.[4]

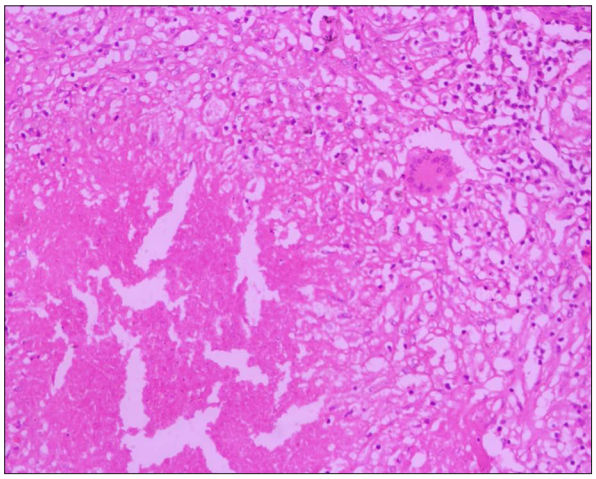

Figure 1: Pericardial tissue showing granuloma with central coagulative necrosis along with langhans' giant cell (x20 H and E stain)

Forty-one cases of CP post renal transplant are reported in the literature. Sever described 34 cases, only one of tubercular etiology.[1] One Presumed chronic glomerulo nephritis, with exudative pleural effusion, treated with 4 weeks of ATT pre-transplant, developed idiopathic CP while on modified ATT 4 months post-transplant.[5] Another case from Sri Lanka had pericardial effusion (fluid: Acid-Fast Bacilli negative) and positive tuberculin test while on dialysis; underwent transplantation after 6 months of ATT and developed CP 6 years post-transplant. Five other cases of CP of unknown etiology are reported in the literature.

Our case was the only CP among 466 renal transplants (2004–2014); 8 (1.7%) had TB. She did not have TB pre-transplant, was not on Cyclosporine, cumulative immunosuppression was minimal and TB was confirmed by histopathology and PCR. CP is extremely rare; can occur at any time post-transplant. High degree of suspicion, pericardiectomy along with ATT can prevent graft dysfunction.

A. Anitha, V. Siddini[1], H. S. Ballal[1]

Department of Nephrology, Manipal Hospital, Salem, Tamil Nadu, [1]Department of Nephrology, Manipal Hospital, Bengaluru, Karnataka, India

Address for correspondence:
Dr. A. Anitha,
903, 21st Main, 38th Cross, 4th T Block, Jayanagar,
Bengaluru - 560 041, Karnataka, India.
E-mail: anitha.a@manipalhospitals.com

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