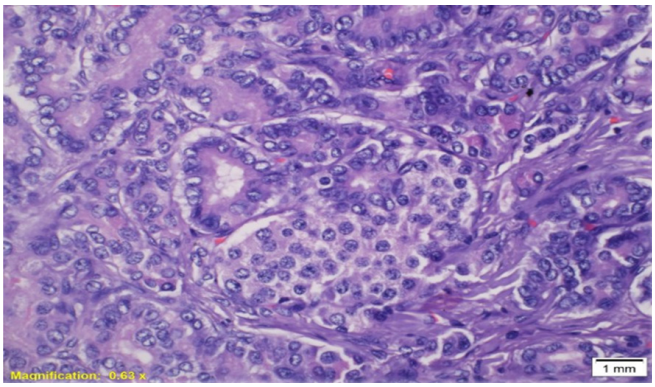

Figure 2: Close proximity of ducts and islet cell cluster forming ductulo–insular complex H&E stain with 40x. H&E: Hematoxylin and Eosin.

uptake. This compensatory mechanism aims to offset declining insulin degradation.

She was negative for anti-insulin antibodies, which was not done in previously reported cases.[1] She had postprandial hypoglycemia, which is typical in Noninsulinoma Pancreatogenous Hypoglycemia Syndrome (NIPHS).

The use of positron emission tomography–computed tomography with Ga-68 EXENDIN, a radiolabeled analog of glucagon-like peptide-1, is an emerging technique for identifying insulin-secreting lesions.[2] In this case, the pancreas showed diffuse increased Ga-68 EXENDIN tracer suggestive of Nesidioblastosis.

The treatment is customized based on severity. Approaches involve dietary adjustments, consuming smaller spaced meals, and avoiding quickly digestible carbs. Medications like diazoxide, octreotide, and acarbose may be used.

Surgical treatment usually involves resecting the insulin-secreting tumor through either enucleation or partial pancreatectomy.[3] The decision regarding the extent of pancreatectomy depends on factors such as size and location of lesion, potential postoperative complications like diabetes mellitus and pancreatic fistula. In instances where diffuse nesidioblastosis is present, subtotal pancreatectomy (80–95%) is typically performed to ensure better management of hypoglycemia compared to conservative pancreatectomy.

Diagnosing adult nesidioblastosis presents unique challenges when coexisting with CKD and diabetes mellitus. The presence of CKD complicates the diagnostic process. Effective management necessitates a comprehensive, multidisciplinary approach, encompassing medical interventions, surgical procedures, or a combination.

Conflicts of interest: There are no conflicts of interest.

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A Rare Case of Tuberculosis Masquerading as Collapsing Glomerulopathy

Abstract

Collapsing glomerulopathy (CG), usually presents with renal dysfunction, hypertension and proteinuria. The etiology is uncertain, yet a number of associations, including many viral infections commonly have been reported. Tuberculosis (TB), one of the most common infections, is not known to cause CG. We report a case of severe renal dysfunction requiring dialysis who had collapsing glomerulopathy on biopsy and evidence of active pulmonary tuberculosis. Anti-tubercular therapy alone resulted in improvement in kidney function.

Keywords: *Antitubercular therapy, Collapsing glomerulopathy, Immune dysregulation, Renal recovery, Tuberculosis*

Introduction

Collapsing glomerulopathy (CG) is a clinicopathologic entity characterized by severe nephrotic syndrome and greater kidney function impairment than other histological variants of Focal segmental Glomerulosclerosis (FSGS) and carries a bad prognosis.[1] HIV infection is a common cause of CG. In non-HIV population, most of the cases of CG are idiopathic, but a number of other disorders, including autoimmune, malignancy, genetic, and drug-induced collapsing glomerulopathies have been reported.[2] Though tuberculosis is very commonly seen in India, there are only a few case reports suggesting tuberculosis as a secondary cause of CG. Here, we report a case of biopsy-proven CG with active pulmonary tuberculosis treated by anti-tubercular therapy.

Case Report

A 45-year-old male, presented with pedal edema and oliguria. He denied dysuria, hematuria, pyuria, fever, cough, SOB, weight and appetite changes. On examination, pallor was detected along with pedal edema, without lymphadenopathy; his blood pressure was 120/70 mmHg. Systemic examination revealed coarse rales bilaterally in lung bases. His investigations showed hemoglobin of 7.0 gm/dl, with leucocytosis and serum creatinine of 3.5 mg/dl with urinalysis showing proteins 4+, 18–20 Rbc, and plenty of pus cells. ANA and Anti ds DNA, ANCA were negative. He was started on dialysis. His chest X-ray showed left upper and middle lobe infiltrates. High resolution computed tomography (HRCT) chest showed patchy consolidation in the left upper lobe, fibrosis in both upper lobes with dilated pulmonary arteries.

On further probing, the patient gave a history of cough on and off which was not fully improving with antibiotics and antitussives. Meanwhile, kidney biopsy, was done and was reported as collapsing FSGS [Figures 1 and 2]. The other causes of FSGS (HIV, HBSAG and HCV, CMV and Parvoviral PCR) were negative. His mantoux was 15 mm; his induced sputum was negative for acid fast bacilli. Bronchoalveolar lavage samples after bronchoscopy were positive for acid fast bacilli. He was started on ATT. One week after starting ATT, his urine output increased to 1.5 liter, and he was discharged with creatinine of 5.0 mg/dl. One month after starting ATT, he had a serum creatinine of 3.2 mg/dl.

Discussion

CG is a recognized variant of FSGS, known to be associated with infections. However, only a few cases associated with pulmonary TB have been reported in the literature.[3,4] Immune dysregulation due to infections in a genetically susceptible individual was causing changes in glomerulus consistent with CG.[5,6] Coventry and Shoemaker reported steroid-resistant nephrotic syndrome in pulmonary TB. In view of absence of the usual associations, infection was the sole known risk factor.[3] Another case of TB-related CG

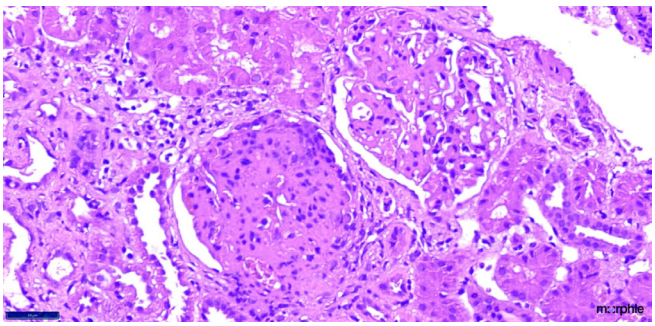

Figure 1: Renal biopsy (40× magnification in H and E staining) depicting two glomeruli; the upper glomerulus is showing segmental sclerosis (9 to 12 o clock position) while the lower glomerulus is showing pseudocrescent with collapse of the glomerular tuft.

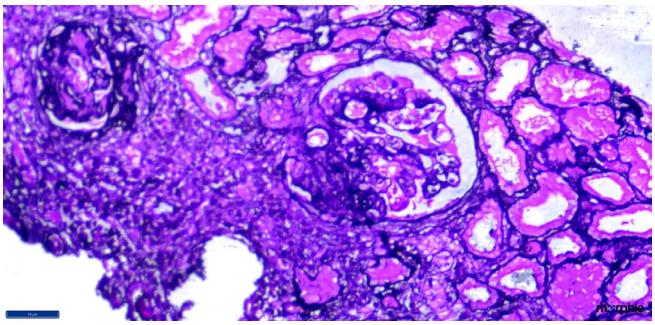

Figure 2: Renal biopsy (40× magnification in silver stain) showing two glomeruli; the one on the right side of the field is showing segmental sclerosis from 6 to 9 o clock position with podocyte hyperplasia and collapsed glomerular tuft.

requiring dialysis for 5 months but with full renal recovery after TB treatment and corticosteroid therapy was reported by Rodrigues *et al*.[4] Currently, there is no specific treatment for CG. However, steroids or immunosuppressive agents have been tried in idiopathic FSGS.[5,6] Our patient responded to ATT, withimproved urine output and improvement in kidney function. However, long-term follow-up is needed to assess the progression of renal disease in this patient.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent.

Conflicts of interest: There are no conflicts of interest.

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Multifocal *Cryptococcus Neoformans* Osteomyelitis in a Kidney Transplant Recipient

Abstract

Cryptococcal infections are notoriously difficult to diagnose and have been associated with high morbidity and mortality. *Cryptococcus neoformans* presenting as osteomyelitis is an unexpected clinical scenario in the transplant ward. A young male who underwent spousal kidney donor transplantation 16 months ago presented with painful and ulcerated soft tissue in upper and lower limbs which were diagnosed as cryptococcus osteomyelitis. He was managed with surgical debridement, liposomal amphotericin B, flucytosine and reduction in maintenance immunosuppression (IS). To our knowledge this is the first reported case of multifocal cryptococcus osteomyelitis in a kidney transplant recipient.

Keywords: *Cryptococcus, Osteomyelitis, Transplantation*

Introduction

Cryptococcus neoformans (CN) is an invasive fungal infection that affects solid organ transplant (SOT) recipients.[1] CN infection in SOT has been described as meningitis and pneumonia, but the entity as osteomyelitis is limited to a few case reports in kidney,[2,3] lung,[4] and liver transplants.[5-7] We report multifocal cryptococcus osteomyelitis (CNO) in a kidney transplant (KT) recipient.

Case Report

A 35-year-old man underwent ABO compatible living spousal donor KT 16 months back with thymoglobulin induction. His baseline serum creatinine was 1.1 mg/dl (steroid 10 mg once daily, tacrolimus @ 0.1 mg/kg with trough levels of 5 ng/mL, and mycophenolic acid in dose of 25 mg/kg/day). He was in continuation phase of anti-tubercular treatment for extra-pulmonary tuberculosis (isoniazid 150 mg, Rifampicin 450 mg, and ethambutol 800 mg once daily). He also had controlled post-transplant diabetes mellitus. The patient first noticed swelling over his right leg, which progressively increased over 15 days and transformed into a painful tense abscess [Figure 1]. Over the next 2 months, he also noticed localized right fore-arm soft tissue swelling. The swelling was painful, fluctuant, and progressively increasing. As his symptoms did not abate, he visited us nearly 2 months after his symptoms first began. On presentation, his vitals, and general and systemic examination were normal. Initial blood investigations were notable for high C-reactive protein of 71 g/dl. His X-ray right forearm [Figure 2a] showed poorly marginated intramedullary lesion seen in the distal aspect of the radius. Magnetic resonance imaging of the right leg showed infective osteomyelitis [Figure 2b-2c]. Pus sample was aspirated from the right arm wound. The gram stain, acid fast bacilli, nocardia stain, fungal stain, TB culture, MTB gene Xpert, and acid fast bacilli were all negative. In view of the infective osteomyelitis, bone curettage, biopsy, external fixation, and bone cementing were done. Pus aspiration and multiplex polymerase test showed CN growth. Bone biopsy sent for fungal culture and histopathology also revealed CN, for which the patient was started on intravenous liposomal amphotericin B (LAMB) in a dose of 200 mg once daily (5 mg/kg/day) and oral flucytosine (5-FLU) 1250 mg every 6 hours. The baseline immunosuppression was reduced by withdrawing the antimetabolite and halving tacrolimus. During the second week an allograft renal biopsy was done for creeping serum creatinine which showed acute tubular necrosis. His tacrolimus level was 4.4 ng/mL and no further changes were made to immunosuppression. After 1 month, the patient's clinical condition had improved, and the swelling over his right forearm and right leg had subsided. The renal allograft function was stable and he was discharged on oral fluconazole 150 mg, which was continued for 6 weeks. At 18 weeks, the patient is doing well, with baseline graft function and baseline immunosuppression restored.