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Case Report

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Disseminated Tuberculosis Following Renal Transplant: A Case Report

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Abstract

Tuberculosis is a great challenge for post-transplant patients. The burden is more complicated in developing countries like Bangladesh. It can cause allograft rejection and graft loss due to delayed diagnosis or loss of efficacy of immunosuppressant drugs following anti-tubercular therapy. The present study documents a case where a patient developed disseminated tuberculosis after receiving a kidney transplant. This article presents the case of a 58-year-old woman who had a renal transplant 10 years back and presented with fever for 2 months. Detailed history and examination shows there is nodular swelling in the left post auricular area along with multiple swelling in the scalp. There was tenderness in the right hypochondriac area. Thorough investigations including a CT scan of the abdomen were done which showed a collection of pus in the subphrenic area. Ultrasound guided aspiration of the pus was done and was sent for microbiological evaluation. FNAC was done from the left post auricular swelling and was sent for microbiological evaluation. Both of the samples were positive for tubercular bacilli. Urine GeneXpert was also positive for AFB. The patient was diagnosed as a case of disseminated tuberculosis and an antitubercular regimen was started. After the treatment patient was clinically improved. The index of suspicion should be high when a post-transplant patient presents with PUO. Early diagnosis may save the graft function.

Keywords: Post-Transplant Tuberculosis; Pyrexia of Unknown Origin; Mycophenolate Mofetil

Introduction

Post-transplant tuberculosis (TB) is notably prevalent in developing countries due to the reactivation of latent infections. The prevalence varies by region: 3.1% to 15% in Asia, 1.5% to 3.5% in the Middle East, 1.7% to 5% in Europe, and approximately 1.5% in the USA [1]. TB in transplant recipients typically arises from either the reactivation of latent infections or the acquisition of new infections [1].

Post-transplant TB can manifest in different organ systems: respiratory involvement in 50% of cases, disseminated TB in 30%, lymph nodes in 5%, skin and soft tissues in 4%, genitourinary system in 4%, intestines in 3%, CNS in 2%, and bones in 1%. Additionally, it presents as Pyrexia of Unknown Origin (PUO) in about 16% of cases [2]. Intra-abdominal abscesses are rare in prevalence. Clinicians should suspect TB in transplant patients presenting with prolonged fever, weight loss, and night sweats, though diagnosis may necessitate invasive procedures.

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Case presentation

A 58-year-old woman presented to us with fever for 2 months, right upper abdominal pain, and multiple swelling in the scalp and left post auricular area for one month. Fever was initially low grade but later it became high grade (the highest recorded temperature was 103°F). It was not associated with chills and rigors and subsided after taking paracetamol. She noticed multiple swellings in the scalp and one swelling in the left post auricular area which was painless. There was a vague discomfort in the right upper abdomen. She has significant weight loss and occasional night sweats. The patient was treated with several courses of different antibiotics but there was no improvement.

The patient had a renal transplant 10 years ago. The donor's kidney was from an unrelated living donor, whose personal information and medical history were unknown. The patient was human leukocyte antigen (HLA)-antibody negative, and her immunological risk status was determined to be low/moderate risk. She was given initial induction therapy with antithymocyte globulin and was started on triple immunosuppressant maintenance therapy with cyclosporine, mycophenolate mofetil (MMF), and prednisolone. She was on antimicrobial prophylaxis with valganciclovir and trimethoprim-sulfamethoxazole. The immediate post-operative period was complicated with urinary tract infections, treated with a short course of intravenous antibiotics, showing a good response. There were no sick contacts but has history of household exposure to pets.

After admission thorough examination was done which revealed tenderness in the right hypochondriac area. There was multiple non-tender swelling in the left post auricular area, and scalp.Her laboratory findings were: Haemoglobin: 11.3 gm/dl, total WBC count was 11.35 K/µL with 92% neutrophil.ESR was 62 mm in 1st hour. CRP was 75 mg/dl. Renal function was normal (S.Creatinine 0.9 mg/dl) , liver function was normal, S. albumin 38 gm/L, blood culture, and urine culture revealed no growth of any pathogenic bacteria. ICT for Malaria and Kalazar was negative. Febrile antigen was also negative.Anti CMV IgG was positive & Anti CMV IgM was negative (Table-1).

Chest X-ray showed mild cardiomegaly.USG of the whole abdomen showed there was a mildly thick-walled irregular cystic lesion, measuring 4.5 ×2.3×3.8 cm.(volume 21.3 cm^3)seen at the right hypochondriac region mostly outside the hepatic capsule. CT Scan of the abdomen showed subcapsular collection in between the right hepatic lobe and anterior parietal wall with adjacent inflammatory changes likely abscess formation(Figure-1). Later pus was aspirated under ultrasound guidance (Figure-2). About 20 ml of thick pus was aspirated and was sent for microbiological evaluation. Z-N stain of the pus shows AFB and MTB was detected in GeneXpert. Ultrasound guided FNAC was done from the postauricular lesion which showed plenty of degenerating polymorphs, histiocytes, and a small number of reactive ductal cells in the background. Urine for GeneXpert was positive for MTB. Sputum was negative for AFB. A diagnosis of disseminated tuberculosis was made and anti-tubercular therapy was initiated with rifampicin (10 mg/kg/day), isoniazid (5 mg/kg/ day), ethambutol (25 mg/kg/day), pyrazinamide (15 mg/ kg/day) and pyridoxine (10 mg/day). After initiation of anti-tubercular therapy fever subsided and her general condition was improved.

Table 1: Laboratory investigations

Detail	Results	Normal Range
White Blood Cells (K/µL)	11.35	4-10
Absolute Neutrophil Count (ANC)%	92%	55-70%
Lymphocytes(K/µL)	0.57	1-3 K/µL
Platelets (K/µL)	314	150-400
Hemoglobin (gm/dL)	11.3	12.0-15.0
ESR (mm/hr)	62	0-19
C-Reactive Protein, CRP (mg/L)	52	0-5
Procalcitonin (ng/ml)	11.5	<0.5
Urea (mg/dL)	19.8	16.60-48.50
Creatinine (mg/dL)	0.9	0.70-1.20
Total Bilirubin (mg/dl)	0.6	0.2-1.0
Albumin (gm/L)	38	35-50
Alanine Aminotransferase (U/L)	38	0-55
Aspartate Aminotransferase (U/L)	20	5-34
Alkaline phosphatase (U/L)	75	46-116
Blood For C/S	Negative	
Urine For C/S	Negative	
ICT for Malaria	Negative	
ICT for Kala-azar	Negative	
Febrile Antigen	Negative	
Anti-CMV IgM	Negative	
Anti-CMV IgG	Positive	

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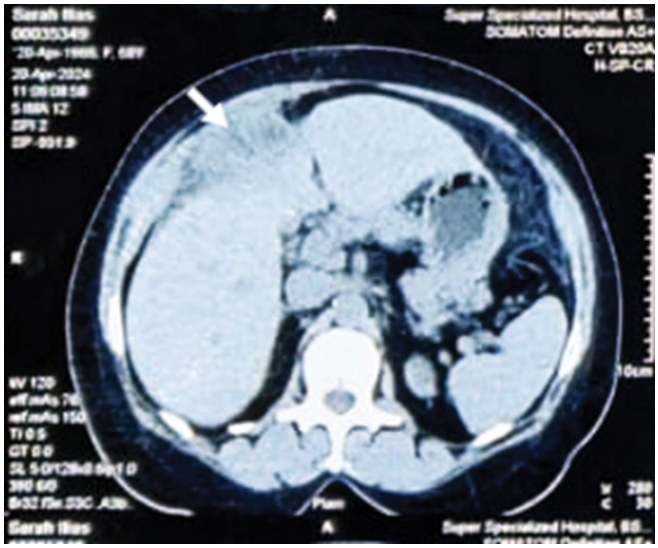

Figure 1: CT abdomen showing right sub-phrenic abscess (arrows)

Figure 2: Ultrasonoguided aspiration of pus from right sub-phrenic abscess (arrows)

Discussion

The risk of tuberculosis (TB) after kidney transplantation is markedly elevated, estimated to be 50-100 times higher than in the general population. A global review indicates that TB typically manifests around 9 months post-transplantation [3]. Among renal allograft recipients, extra-pulmonary TB is common (51.8%), with disseminated TB being the most prevalent (19.3%), followed by pyrexia of unknown origin (15.7%), TB lymphadenopathy (4.8%), and involvement of skin, soft tissue, intestines, CNS, bones, pericardium, or urinary tract [4]. Reactivation of latent infection is the primary cause of TB in these recipients, often exacerbated by immunosuppressive therapy. Delayed diagnosis due to atypical presentations or excessive immunosuppression may lead to disseminated TB.

Classical risk factors include prolonged hemodialysis, pre-transplant diabetes mellitus, use of anti-CD25 monoclonal antibodies for induction immunosuppression, multiple rejection episodes, treatment with antilymphocyte globulin or bolus corticosteroids, chronic liver disease, and prior infections such as pneumocystis pneumonia and nocardiosis. Diagnosis of renal allograft TB utilizes various modalities, with ultrasound playing a significant role due to its superficial nature. Multidetector CT scans are effective in identifying specific TB-related features such as calcifications, renal scars, mass lesions, urothelial thickening, and caliectasis.

In a specific case discussed, active pulmonary TB was absent before transplantation, and the donor also tested negative for acid-fast bacilli in urine and systemic cultures. Thus, the infection was likely de novo due to immunosuppression. The incidence of TB in this case report reflects the high prevalence in regions like Bangladesh.

Disseminated TB in solid organ recipients presents with varied clinical and laboratory findings, necessitating a high index of suspicion for accurate diagnosis. Treatment involves tailored immunosuppression with careful drug monitoring to minimize risk. Prolonged treatment duration and consideration of secondary prophylaxis are essential due to emerging challenges such as drug resistance and atypical mycobacterial infections, particularly in non-responsive patients.

The clinical and laboratory presentations of disseminated TB can be nonspecific and diverse, including hematological abnormalities such as anemia, leukopenia, leukocytosis, monocytosis, agranulocytosis, thrombocytopenia, and pancytopenia [5,6]. Elevated ESR is typical, although specific manifestations may be absent, except for a high ESR, as seen in the described case.

Chest X-rays are pivotal for initial TB detection [7]. In the case discussed, the CXR revealed no significant abnormalities, while abdominal CT scans identified a subdiaphragmatic abscess. The primary diagnosis of TB relies on acid-fast bacilli detection, with sputum smear positivity in less than 20% of miliary TB cases [8]. In this instance, acid-fast bacilli were identified in the post-auricular lymph node fine needle aspiration cytology, urine PCR for MTB, and microbiological evaluation of pus from the subdiaphragmatic abscess, confirming TB through geneXpert, facilitating rapid diagnosis.

Subphrenic abscesses, associated with high mortality and morbidity, historically arose from primary intra-abdominal infections. Modern advances in diagnosis, surgical techniques, and antibiotic use have improved outcomes. Bacterial flora typically include aerobic (e.g., E. coli, Klebsiella, Proteus) and anaerobic organisms (e.g., Bacteroides, Clostridia) [10]. In our case, pus culture confirmed AFB organisms, guiding

effective anti-TB chemotherapy. Given the challenges in culturing extrapulmonary TB, cytological evidence of granulomatous inflammation supported prompt treatment initiation, reducing morbidity and mortality [11,12].

Conclusion

This case report underscores the critical need for maintaining a vigilant clinical approach and conducting comprehensive evaluations to promptly diagnose disseminated tuberculosis during periods of immunosuppression, particularly in regions where tuberculosis is endemic. Additionally, it highlights the importance of considering subphrenic or intra-abdominal abscesses in immunocompromised patients presenting with prolonged fever of unknown origin (PUO), as timely tuberculosis treatment can significantly mitigate both mortality and morbidity risks.

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