

## Common Infection in Unusual Site in a Renal Allograft Recipient

Krithika Muralidhara\*, Renuka Satish and Jayram A

Department of Nephrology, St John's Medical College Hospital, Bangalore, India

\*Corresponding author: Muralidhara K, Assistant Professor, Department of Nephrology, St John's Medical College Hospital, Bangalore, India, Tel: +91 9972389333; E-mail: [krithidm@yahoo.co.in](mailto:krithidm@yahoo.co.in)

Received: March 16, 2022; Accepted: March 24, 2022; Published: April 02, 2022

### Abstract

Infection is a common complication in renal allograft recipient. Due their immunosuppressive state they can develop bacterial, viral, tubercular and fungal infections. This infection can occur any time but more common in first 6 months post-transplant. Here, we report a common infection occurring in an unusual site in a renal allograft recipient.

**Keywords:** *Infection; Immunosuppression; Calcineurin inhibitors (CNI); Renal allograft recipient*

### 1. Introduction

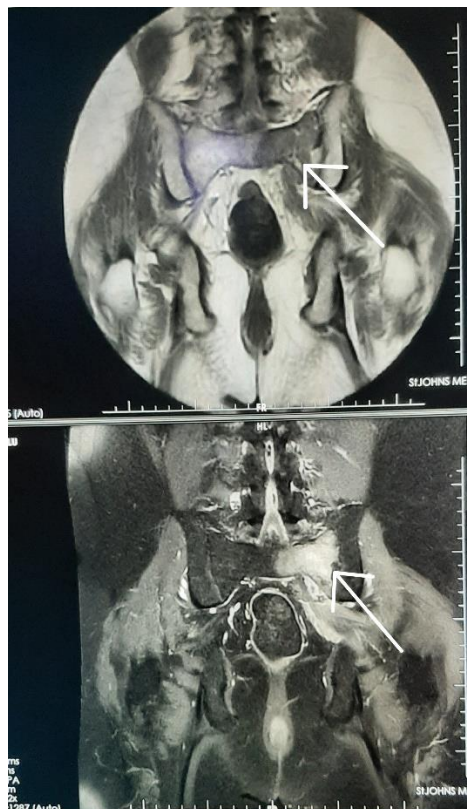
Renal transplant recipients are susceptible to a variety of infections with both common and opportunistic pathogens as a result of immunosuppressive therapy [1]. These infections cause significant morbidity and mortality. Among them tuberculosis is a common infection in transplant patients [2,3]. In renal allograft recipient extra-pulmonary tuberculosis is more common. Skeletal tuberculosis is seen in 1%-5% of cases. The sacroiliac joint is involved in 3%-5% of cases [4]. Sacroiliac joint infections often present as a diagnostic dilemma due to the vague and non-specific clinical presentation. Herein, we report a renal allograft recipient presenting with hip pain and fever, diagnosed with sacroiliac joint tuberculous infection that responded to anti-TB treatment.

### 2. Case Report

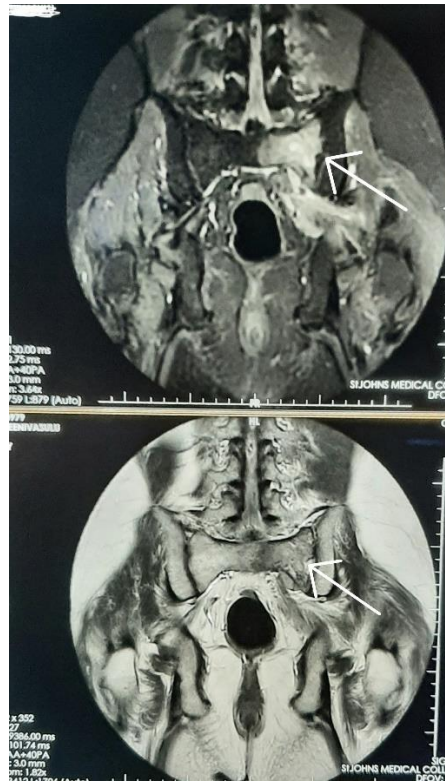
Mr. S 38-year school teacher presented with headache and nausea of 2 months history. On evaluation he was found to be hypertensive with grade II hypertensive retinopathy and pallor. On investigation found have proteinuria active urine sediment, advance renal failure and bilaterally small kidneys on abdominal ultrasound. He was initiated on haemodialysis through right internal jugular access. Two months after starting HD he underwent renal transplantation with his sister being the donor. Post-

transplant he had brisk diuresis and no major issues and was discharged 10<sup>th</sup> post operative day with S. creatinine of 1.0 mg/dl on triple immunosuppression.

He was on regular out-patient follow up, had stable graft function and immunosuppression slowly tapered to maintenance level. Ten months post-transplant he presented with progressive pain in the left hip and lower back with difficulty in walking of 7 days duration. It was associated with low grade fever, but no other constitutional symptoms. On examination, there was localized tenderness, however no restriction of joint movements or neurological deficits. CBC showed haemoglobin 12 g, total count 4700 cell/cu mm with normal differential count. ESR and CRP were 5 and 19 mg/dl respectively. Serum creatinine was 1.2 mg/dl and serum calcium 8.1 mg/dl with normal liver function tests. Initial X-ray of the hip joint was inconclusive. HLA B27 to rule out Ankylosing spondylitis was negative. Further, MRI of the pelvis and spine showed T2 and STIR hyperintensity with low T1 signal intensity in the left sacroiliac joint suggestive of sacroiliitis (as shown in FIG. 1 & 2). Patient was started on empirical antibiotics considering the short duration of symptoms, but he continued to have persistent pain. For definitive diagnosis, CT guided aspiration from the joint lesion was performed. The smear showed numerous acid-fast bacilli & later culture was positive for mycobacterial tuberculosis, thus confirming the diagnosis of tubercular sacroiliitis. Along with pain alleviating therapy, he was started on rifampicin free anti-tubercular regimen substituting it with quinalones. Despite appropriate dosing of medications, he continued to be symptomatic with hip pain requiring readmission and repeat imaging which showed persistent infection and pus collection in the sacroiliac joint. Considering non-response to therapy, rifampicin was added to the regimen. After adding rifampicin, drug level of tacrolimus was monitored regularly and dose of immunosuppressant's was modified accordingly. Two months into therapy, patient has remained afebrile and pain free with stable allograft functions.



**FIG.1. Coronal section of pelvis showing T1 hypointensity in left sacroiliac joint and enhancement on contrast study.**



**FIG. 2. Coronal section of pelvis showing T2 hyperintensity in left sacroiliac joint and confirmation on STIR study.**

### **3. Discussion**

We report the case of a renal allograft recipient who presented with unilateral hip pain 10 months post transplantation with progressive symptoms. Thorough clinical evaluation and appropriate imaging studies with a tissue diagnosis led to the confirmation of tubercular sacroiliitis in our patient.

Transplant recipients are prone to develop a variety of opportunistic infection because of their reduced immunity [1]. Among the various infections, tuberculosis is the most common opportunistic infection in renal transplant recipients. The incidence of TB among transplant recipients is 30 to 50 fold higher than that among the general population [2]. Though, pulmonary TB continues to be the most common form of TB, extra-pulmonary lesions are more frequent in patients following transplantation compared to patients without immunosuppression [3]. Extra-pulmonary TB has been reported in lymph nodes, pleura, gastrointestinal tract, genitourinary tract and bone. Skeletal TB involving the spine, hips, knees, elbows, shoulders and small joints of the hands and feet comprises approximately 3%-5% of extra-pulmonary TB [4]. The likely route for TB sacroiliitis may be hematogenous via the pelvic and paravertebral venous plexus of Batson [5]. Patients generally present with gradually increasing hip pain associated with low grade fever. Unilateral hip pain and free range of motion without neurologic deficit favors a sacroiliac joint disorder rather than lumbar spine disease or hip fracture. Plain radiographs may not show any abnormality in the early stage of sacroiliac tuberculosis. The radiographic changes of blurring and loss of the cortical margins along with pseudo-widening of the joint space appear in the late stage of the disease [6,7]. CT and MRI are valuable in providing an earlier diagnosis of sacroiliitis. CT scan shows clearly the extent of joint destruction and MRI delineates the abscess in the soft tissues [8-10].

However, the limitations of the imaging studies are the inability to distinguish TB from other pyogenic sacroiliitis. A definitive diagnosis is obtained by fine needle aspiration or open biopsy and identification of the pathogen [11,12]. Acid-fast bacilli in direct smears, the growth of the bacilli in the Löwenstein-Jensen culture or the granulomatous lesion identified in the histological specimen will confirm the diagnosis of tuberculosis. The standard of therapy remains anti-tubercular medications for longer duration ( $\geq 6-9$  months). However, the use of rifampicin must be undertaken with caution because of its major interaction with CNI. Dose modification of CNI must be done with frequent monitoring of blood levels [13,14].

In conclusion, this case of a renal allograft recipient depicts a common infection in an unusual site. The diagnosis of post-transplant TB sacroiliitis can be challenging due to vague symptoms and attenuated inflammatory response in the background of immunosuppressant's. A high index of suspicion is warranted to achieve early diagnosis and initiate appropriate treatment.

#### **4. Acknowledgment**

We acknowledge the support by team of radiology.

#### **REFERENCES**

1. Milar JW, Home NW. Tuberculosis immunosuppressed patients. *Lancet*. 1979;1(8127):1176-8.
2. Ho KJ. Galloping caseous pneumonia with miliary dissemination in a renal transplant recipient: emphasis on pretransplant detection and prophylaxis. *Nephron*. 1992;62(3):363-4.
3. Hussam A, Faraj SA, Haider A. Renal allograft tuberculosis. *Ann Saudi Med*. 2002;22(5-6):346-8.
4. Ascher NL, Simmons RL, Marker S, et al. Tuberculous joint disease in transplant patients. *Am J Surg*. 1978;35(6):853-6.
5. Raman R, Dinopoulos H, Giannoudis PV. Management of pyogenic sacroiliitis: an update. *Current Orthopaedics*. 2004;18(4):321-5.
6. Soholt ST. Tuberculosis of the sacro-iliac joint. *J Bone Joint Surg*. 1951;33:119.
7. Delbarre F, Rondier J, Delrieu F, et al. Pyogenic infection of the sacroiliac joint. *J Bone Joint Surg*. 1975;57:819.
8. Keles I, Aydin G, Kitay OL, et al. Tuberculous sacroiliitis: a case report. *Rheumatol Int*. 2004;24:312-4.
9. Kim NH, Lee HM, Yoo JD, et al. Sacroiliac joint tuberculosis. Classification and treatment. *Clin Orthop*. 1999;358:215-22.
10. Vaiopoulos G, Sfikakis PP, Velikas E, et al. Tuberculosis of the sacroiliac joint. *Eur Spine J*. 1997;6(5):330-31.
11. Hendrix RW, Lin PJ, Kane WJ. Simplified aspiration or injection technique for the sacro-iliac joint. *J Bone Joint Surg*. 1982;64(8):1249-52.
12. Miskew DB, Block RA, Witt PF. Aspiration of infected sacro-iliac joints. *J Bone Joint Surg*. 1979;61(7):1071-2.
13. Subramanian A, Dorman S. Mycobacterium tuberculosis in solid organ transplant recipients. *Am J Transplant*. 2009;9(Suppl 4):S57-62.
14. Klote MM, Agodoa LY, Abbott K. Mycobacterium tuberculosis infection incidence in hospitalized renal transplant patients in the United States, 1998-2000. *Am J Transplant*. 2004;4(9):1523-8.