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Case Report

Tuberculous Infection of a Total Hip Arthroplasty in an Immunocompetent Patient, Revealed by Prosthetic Loosening: A Case Report and Literature Review

Elmostafa Benaissa^{1*}, Sara Oucharqui¹, Yassine Benlahlou¹, Adil Maleb², Mariama Chadli¹, Mostafa Elouennass¹

¹Department of Clinical Bacteriology, Mohammed V Military Teaching Hospital, Faculty of Medicine and Pharmacy of Rabat, Mohammed V University, Rabat, Morocco

²Laboratory of Microbiology, Mohammed VI University Hospital, Faculty of Medicine and Pharmacy (University Mohammed the First), Oujda, Morocco

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*Corresponding author: Elmostafa Benaissa

Department of Clinical Bacteriology, Mohammed V Military Teaching Hospital, Faculty of Medicine and Pharmacy of Rabat, Mohammed V University, Rabat, Morocco

Abstract

Prosthetic joint infection caused by *Mycobacterium tuberculosis* is extremely rare. It can mimic aseptic loosening or chronic infection with common bacteria, delaying diagnosis. We report a case of tuberculous infection in a total hip arthroplasty in a 55-year-old immunocompetent patient, revealed by prosthetic loosening. The diagnosis was confirmed by histology, culture, and molecular biology (GeneXpert). This case highlights the need to consider tuberculosis in the differential diagnosis of chronic prosthetic infections, particularly in endemic areas.

Keywords: Prosthetic joint infection, *Mycobacterium tuberculosis*, chronic infection, common bacteria, hip arthroplasty.

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Introduction

Prosthetic joint infection (PJI) is a serious complication of arthroplasty, with major functional and socioeconomic consequences. The incidence of these infections is estimated between 0.3% and 1.9%, depending on the location [1]. Most cases are caused by pyogenic bacteria such as Staphylococcus aureus and coagulase-negative staphylococci [2]. Mycobacterium tuberculosis (MTB), the causative agent of tuberculosis (TB), is a rare but emerging cause of prosthetic infection, high-endemic particularly in countries Osteoarticular tuberculosis accounts for 1-3% of TB cases, and prosthetic involvement remains exceptional [4]. Diagnosis is often delayed due to misleading clinical presentation and the low yield of conventional tests. We report a case of tuberculous infection in a total hip arthroplasty in a 55-year-old immunocompetent patient, revealed by prosthetic loosening.

CASE REPORT

A 55-year-old male patient was admitted for total functional impairment and instability of the left hip. His medical history included a total hip replacement in

2017 following a femoral neck fracture. There was no history of TB exposure or prior TB treatment.

The disease onset dated back to one year before admission, with progressive instability and partial functional impairment of the left hip, accompanied by pain upon movement after a fall on the operated hip. The patient did not seek medical attention. Three months before admission, local swelling and purulent discharge from the lateral hip scar developed, along with general health deterioration. The patient reported no constitutional TB symptoms or respiratory complaints.

On examination, the patient was conscious but in poor general condition (weight = $57 \, \text{kg}$, height = $172 \, \text{cm}$, BMI = 19), with stable hemodynamic and respiratory status. Musculoskeletal examination revealed total functional impairment with instability. The lower limb exhibited significant muscle atrophy, neutral rotation, and approximately 2 cm shortening. The surgical scar showed inflammatory signs without fistula or active discharge. Sensation was intact, with no motor deficit distally, and the posterior tibial pulse was present.

Anteroposterior pelvic radiography showed acetabular implant loosening (Figure 1).



Figure 1: Acetabular implant loosening

CT scan revealed intra-pelvic displacement of the acetabular cup. Chest X-ray was normal. Aseptic loosening of the hip prosthesis was initially suspected, and the patient underwent transcondylar traction. Superficial and deep pus samples were sent for bacteriological analysis, including direct examination, culture, and molecular testing for acid-fast bacilli (AFB). Laboratory findings included a CRP of 43 mg/L and WBC count of 16 G/L. Direct AFB staining (auramine and Ziehl-Neelsen) was negative. GeneXpert MTB/RIF (Cepheid, Sunnyvale, CA, USA) detected M. tuberculosis in deep pus within 2 hours, prompting immediate antitubercular therapy. Two weeks later, liquid culture turned positive, followed by Löwenstein-Jensen culture at 3 weeks.

DISCUSSION

Prosthetic joint infections after total hip arthroplasty occur at a rate below 1% [1]. Common etiologies include coagulase-negative staphylococci, S. aureus, aerobic Gram-negative bacilli, and anaerobes [3]. M. tuberculosis is a rare cause of prosthetic joint infection [4]. TB prosthetic infections typically affect hips or knees and may result from local reactivation or, less commonly, hematogenous spread. Local reactivation can occur up to 42 years after initial infection and may be triggered by trauma or surgery. Reactivation of latent TB (e.g., in lungs, kidneys, or mesenteric lymph nodes) can lead to subsequent prosthetic seeding [5]. Thus, documented TB infection at another site should raise suspicion for tuberculous joint involvement.

Conditions predisposing to TB prosthetic infection include rheumatoid arthritis, corticosteroid therapy, and pulmonary disease [5]. Prosthetic replacement for prior TB-affected joints is a debated risk factor due to potential quiescent infection reactivation [5]. Trauma is a known predisposing factor for skeletal TB, and peri-prosthetic tissue injury may play a key role in reactivation. Mechanical synovial tissue disruption

could potentially destabilize old tuberculous granulomas, leading to reactivation [6].

The most common presenting symptom is pain, while physical findings often include joint swelling and/or fistulous tracts [7]. Radiographs may show lucency at the bone-cement interface, cement fractures, or component migration [4]. Diagnosis relies on tissue culture and histopathology, which may reveal AFB or caseating granulomas. *M. tuberculosis* culture remains diagnostic gold standard [5]. GeneXpert demonstrates high sensitivity and specificity for extrapulmonary samples, making it a rapid and valuable tool.

Other bacteria, particularly *S. aureus*, must be ruled out. Coinfection cannot be excluded, but persistent infection despite bacterial eradication should raise suspicion for TB [7]. The need for implant removal alongside antitubercular therapy remains debated [10]. In one study of 10 cases diagnosed at or within 6 weeks post-arthroplasty, all were successfully treated with antitubercular drugs alone, with favorable outcomes at 9 months–10 years follow-up. However, late-presenting infections (months/years post-surgery) often fail medical therapy, necessitating implant removal [6].

Our patient was diagnosed 4 years postarthroplasty. Initially, the prosthesis was retained, and standard TB therapy was initiated per national guidelines: 2 months of rifampicin, isoniazid, pyrazinamide, and ethambutol, followed by 10 months of rifampicin and isoniazid. At 4 months, clinical improvement was noted. Prophylactic antitubercular therapy has been suggested for patients with prior TB history [5].

CONCLUSION

This case underscores the importance of considering M. tuberculosis in chronic prosthetic infections, particularly in endemic regions. A diagnostic strategy combining histopathology, targeted cultures, and molecular testing is crucial for early diagnosis and appropriate management.

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