A Tuberculous Midpalmar Abscess of the Hand Due to Reactivation of Previous Pulmonary Tuberculosis

Dear Editor,

A 45-year-old renal transplant recipient presented with a 2-week history of progressive painful mid-palmar swelling in his right hand and intermittent low-grade pyrexia. There was no history of trauma. He had been on transplant-related immunosuppression (cyclosporin and prednisolone) for 10 years, and had a significant past history of pulmonary tuberculosis treated 20 years ago without previous reactivation.

Initial physical examination (Fig. 1) revealed oedema of the right hand with central palmar swelling and redness. There was warmth and tenderness of the mid-palm and the middle finger along the flexor sheath from the level of the A1 pulley to the proximal phalanx. The middle finger was not swollen or flexed, but passive extension was painful. Radiographs showed only soft tissue swelling. Suppurative flexor tenosynovitis with midpalmar space extension was provisionally diagnosed. Emergent surgical exploration revealed unhealthy synovium around the middle finger flexors about the A1 pulley, without suppuration of tendon sheath or midpalmar space. This was debrided and sent for histology, bacterial, fungal and mycobacterial cultures and microscopy. Postoperatively, he improved with intravenous antibiotics and was discharged on oral amoxicillin-clavulanate. Tissue bacterial cultures and blood cultures were negative, and fungal and mycobacterial cultures were pending at the time of discharge.

Two weeks later, he developed purulent discharge from the surgical wound along with severe pain and swelling of the hand and digits. The mid-palm was tense, tender, erythematous, and all fingers were flexed and painful on passive extension. He spiked temperatures of 40 degrees Celsius. The total white had risen to 13.50 x 10 (9)/L and erythrocyte sedimentation rate (ESR) was elevated at 86. Histology from the earlier debridement showed a chronic inflammatory infiltrate with Langhan's giant cells. Ziehl-Nielsen staining showed acid-fast bacilli. However, his chest X-ray done for the prior surgery did not show any obvious features of post-primary tuberculosis such as cavitation.

At repeat debridement there was an extensive mid-palmar space abscess, with frank purulence and caseating necrosis of the subcutaneous fat. Necrotic tissue encased the common palmar digital nerves and vessels, superficial arch and flexor tendons. Radical debridement of all necrotic tissue left a significant defect exposing the neurovascular bundles and flexor tendons. A topical negative pressure dressing was used to minimise dressing changes and wound exposure on the ward. *Mycobacterium tuberculosis* infection was confirmed on polymerase chain reaction and mycobacterial cultures (sensitive to streptomycin, rifampicin, isoniazid, ethambutol). A 6-month course of antituberculous chemotherapy comprising isoniazid, rifampicin and pyrazinamide was started in consultation with infectious disease physicians. The infection settled and the wound contracted and granulated rapidly, leaving a superficial defect that was skin-grafted.

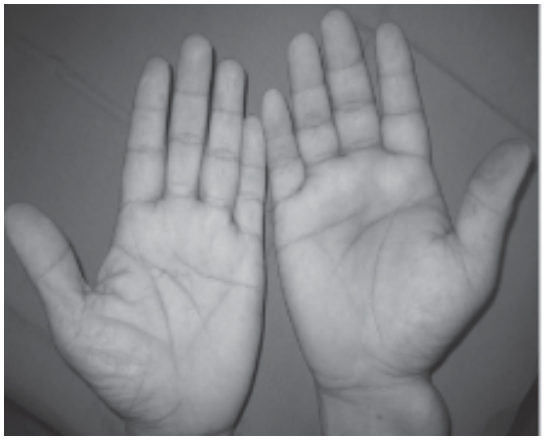

Fig. 1. Right hand with mild palmar swelling in region of third ray before surgery.

Mycobacterial infection of the hand and wrist is uncommon, with atypical mycobacteria being more common than mycobacterium tuberculosis. Both types usually involve tenosynovium or osteoarticular components primarily, occasionally extending into soft tissue.[1] While atypical mycobacterial hand infections may arise in healthy individuals, *mycobacterium tuberculosis* usually affects the immunocompromised [human immunodeficiency virus (HIV), long-term corticosteroid therapy, malnourished unvaccinated children, elderly].[2] Renal allograft recipients have a higher risk of tuberculosis due to long-term immunosuppression. The incidence of tuberculosis in this population ranges from 3.1% to 11.8%, up to 50% of which are extrapulmonary infections with an increased risk of mortality.[3]

Tuberculosis of the hand and wrist usually occurs haematogenously, unlike atypical mycobacterial infections, which arise after innoculation. It may manifest as primary extrapulmonary infection, or as post-primary reactivated

endogenous tuberculosis as in this case.[4] It presents with non-pathognomonic chronic local swelling and pain on motion and is easily confused with non-specific inflammatory synovitis and arthropathy. Occasionally, it may present acutely with features suggesting pyogenic infection. Here, the initial middle finger tenosynovitis subsided rapidly following debridement and antibiotic therapy, suggesting pyogenic infection. Subsequently, a purulent midpalmar abscess and spiking fever developed. This is uncharacteristic of tuberculous hand infection, where localised abscesses usually are indolent "cold" abscesses. Delay of months to years from symptom onset to the correct diagnosis has been reported.[1] Both local steroid injections and systemic oral steroids should be avoided until tuberculosis is excluded in susceptible individuals.

Cases presenting as acute pyogenic infection should be treated emergently. Abscess drainage and radical debridement decreases mycobacterial load, inducing healing and facilitating delivery of antituberculous agents to the infected area. Elevation and rest decreases oedema, allowing early reconstruction and mobilisation. Topical negative pressure dressings are useful if debridement creates a large defect, stimulating wound granulation and contraction to allow a simpler reconstructive procedure such as skin-grafting instead of a flap. Antituberculous chemotherapy should be started empirically once tuberculosis is suspected, and switched to appropriate drugs upon obtaining definitive diagnosis.[1] Combined debridement and chemotherapy for tuberculosis of the hand and wrist is effective leading to decreased recurrence and good recovery of function.[1,4]

Various non-invasive investigations have been suggested but are generally not useful for diagnosing tuberculosis of the hand. White cell count and ESR may be elevated.[1,4] Imaging studies may define the disease extent and help in preoperative planning, but are not specific enough for diagnosing tuberculosis. Plain films may demonstrate arthritis and osteomyelitis, bone or joint destruction, or spina ventosa. Magnetic resonance imaging (MRI) is more useful for soft tissue tuberculosis, while computed tomography (CT) may demonstrate calcification in chronic tuberculous abscesses. Jaovisidha et al[5] were able to diagnose all forms of tuberculous tenosynovitis using a combination of CT and MRI. The Mantoux skin test is affected by many variables and is unreliable for confirming active infection.

Routine tissue biopsy for histopathology, Ziehl-Nielsen staining and mycobacterial cultures should be obtained during synovectomy for chronic swelling or debridement of acute hand infections in at-risk populations. These are the only investigations that will definitively diagnose tuberculosis. In this patient, histopathology and microscopy of tissue samples taken at the first debridement confirmed tuberculosis. Antituberculous chemotherapy was therefore started shortly after the second debridement, despite the unusual presentation suggesting a pyogenic abscess.

The incidence of tuberculosis infection locally has decreased from 300 per 100,000 in the 1960s to plateau at 40.8 per 100,000 in recent years.[6] The trend is similar in other developed countries. However, pulmonary and extrapulmonary tuberculosis, including that of the hand and wrist, may be increasing with increasing worldwide numbers of immunocompromised individuals, an ageing population that is more prone to endogenous tuberculosis reactivation, and the emergence of multi-drug resistant tuberculosis.[6] We recommend a high index of suspicion for tuberculosis when treating pain and swelling of the hand in these populations.

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