Case Report

Mycobacterium tuberculosis Infection of a Tophaceous Pseudogout Nodule

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ABSTRACT. Musculoskeletal infections are uncommon complications of monosodium urate and calcium pyrophosphate dihydrate (CPPD) crystal deposition disease, and frequently involve gram positive and negative organisms. Tumoral calcinosis (tophaceous pseudogout) is a rare manifestation of CPPD deposition disease. We describe a highly unusual case of an infection by *Mycobacterium tuberculosis* (TB) of a tophaceous pseudogout nodule in a patient with endstage renal disease. The highly destructive nature of this case of combined CPPD arthropathy and musculoskeletal TB underscores the urgency of diagnosing this infection in susceptible patients from countries with high prevalence rates of TB infection. (J Rheumatol 2002;29:1093–6)

Key Indexing Terms:
MYCOBACTERIUM TUBERCULOSIS INFECTION
CALCIUM PYROPHOSPHATE DIHYDRATE DEPOSITION DISEASE TOPHUS

Septic arthritis is a recognized complication of crystal deposition disease[1]. In developing countries with high prevalence rates of *Mycobacterium tuberculosis* infection, musculoskeletal tuberculosis (TB) is a serious problem. Patients with compromised immune functions are at higher risk of developing extrapulmonary TB[2].

Tumoral calcinosis or "tophaceous pseudogout" is a rare manifestation of calcium pyrophosphate dihydrate deposition disease (CPPD)[3]. Although there have been 2 reported cases of *M. tuberculosis* infection of gouty arthritis[4,5], mycobacterial infection of a CPPD tophus has not previously been described.

CASE REPORT

A 20-year-old man undergoing hemodialysis for 4 years due to mesangioproliferative glomerulonephritis, with secondary hyperparathyroidism and chronic hepatitis B infection, was admitted after 5 days of crampy abdominal pain and diarrhea. He reported recurrent low grade fever, marked weight loss, and cough productive of white sputum over the last 4 months.

Fourteen months before admission, firm, painless nodules developed over both elbows and metacarpophalangeal (MCP) and proximal interphalangeal (PIP) joints of both hands. Allopurinol and colchicine were started for suspected tophaceous gout. No joint aspiration was done. Despite these treatments, the tophi grew larger during the last 4 months before admission.

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Outpatient medications included nizatidine, sodium bicarbonate, calcium carbonate, erythropoietin, colchicine, celecoxib, allopurinol, and multivitamins.

Examination revealed a pale, emaciated, and underdeveloped young adult with temperature 37.5°C, blood pressure 130/80 mm Hg, pulse rate 106/min, and respiratory rate 28/min. He had coarse bibasilar crackles, a distended abdomen, and hypoactive bowel sounds. Musculoskeletal examination revealed multiple firm, nontender nodules on both elbows, MCP, and PIP. He had softer nodules on the popliteal surfaces of both knees.

Admission laboratory results showed anemia with mild leukocytosis (10,320/mm^3) and left shift (82% segmenters), elevated creatinine, hypoalbuminemia (18 g/l, reference 30–50 g/l), serum calcium of 2.5 mmol/l (reference 2.2–2.6), pyuria, and glucosuria. A recent outpatient serum parathyroid hormone level was normal at 29.5 pg/ml (reference 12–72), down from 150 pg/ml 4 months before admission.

Abdominal radiographs showed fecal retention without obstruction. Chest radiograph showed linear and fine nodular densities in both upper lung fields with small pleural effusions. Sputum was submitted for acid fast bacilli (AFB) culture. Preliminary AFB sputum smears were negative.

The hospitalization was characterized by anorexia, abdominal pain, and recurrent fevers. Three weeks after admission, the left elbow and right 4th MCP tophi became painful, swollen, and fluctuant (Figure 1), with oral temperatures as high as 39.5°C. Purulent fluid (10 cc) was aspirated from the right 4th MCP, and ~0.5 cc of turbid fluid was obtained from the left elbow.

Analysis of the right 4th MCP fluid showed a cell count of 7488 white blood cells/mm^3, with 36% neutrophils, 31% lymphocytes, and 33% monocytes. The gram stains in both MCP and elbow aspirates were negative. Polarized light microscopy of the MCP fluid revealed weakly positively birefringent rods consistent with CPPD and lipid crystals. Monosodium urate crystals were not seen. Investigation for calcium hydroxyapatite crystals was not done because special techniques to detect apatite crystals were not available at our institution. Because of suspected disseminated TB, the MCP fluid was sent for AFB smear, and was found to contain large amounts of AFB positive organisms (Figure 2).

Plain radiography showed periarticular calcifications around the PIP and MCP of both hands, with destruction of the 3rd metacarpal head of the right hand (Figure 3). Comparison with hand radiographs done as an outpatient 4 months earlier confirmed the recent onset of these calcifications and

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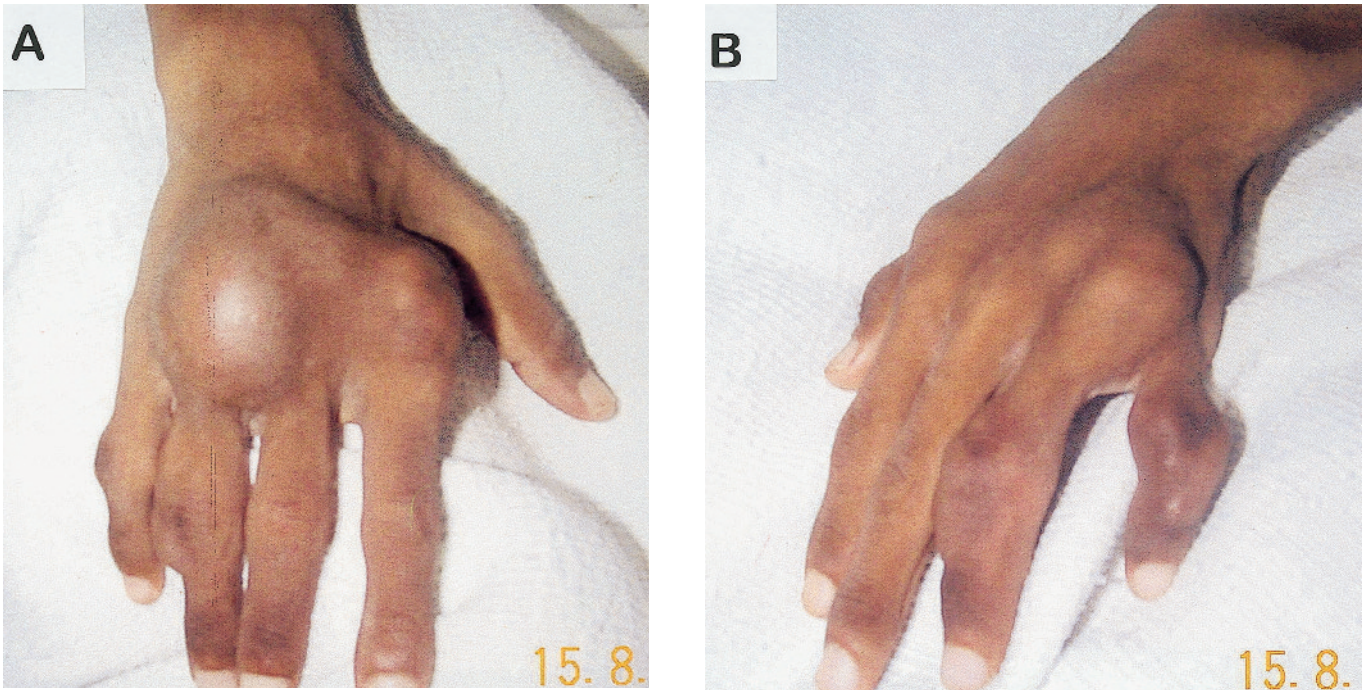

Figure 1. Clinical photographs of the right hand (A) and left hand (B) showing tumoral deposits of CPPD crystals around the PIP and MCP joints. Note the abscess overlying the MCP joints of the right hand.

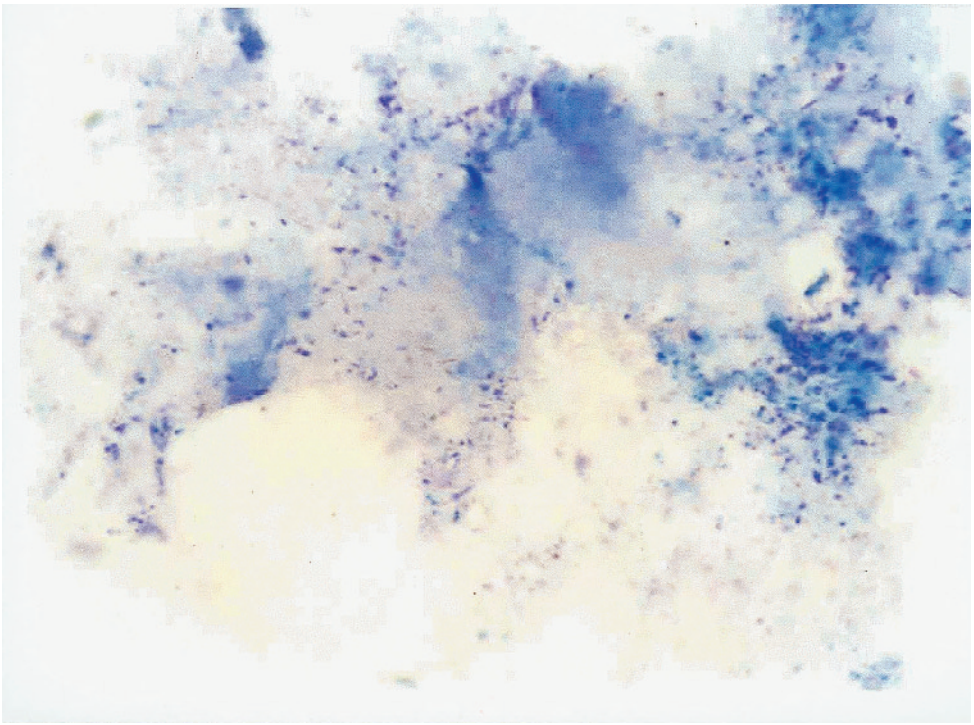

Figure 2. Photomicrograph of fluid aspirate from the right MCP abscess/tophus. Acid-fast bacilli are abundantly visible. Ziehl-Nielsen stain, ×40.

erosions. Radiographs of the knees were not done since the patient was asymptomatic. He was given quadruple antibiotics (isoniazid, ethambutol, rifampicin, pyrazinamide), with initial defervescence, but compliance was erratic due to nausea and vomiting. By this time, the admission sputum cultures were growing *M. tuberculosis*.

The fever and right 4th MCP abscess persisted despite serial needle

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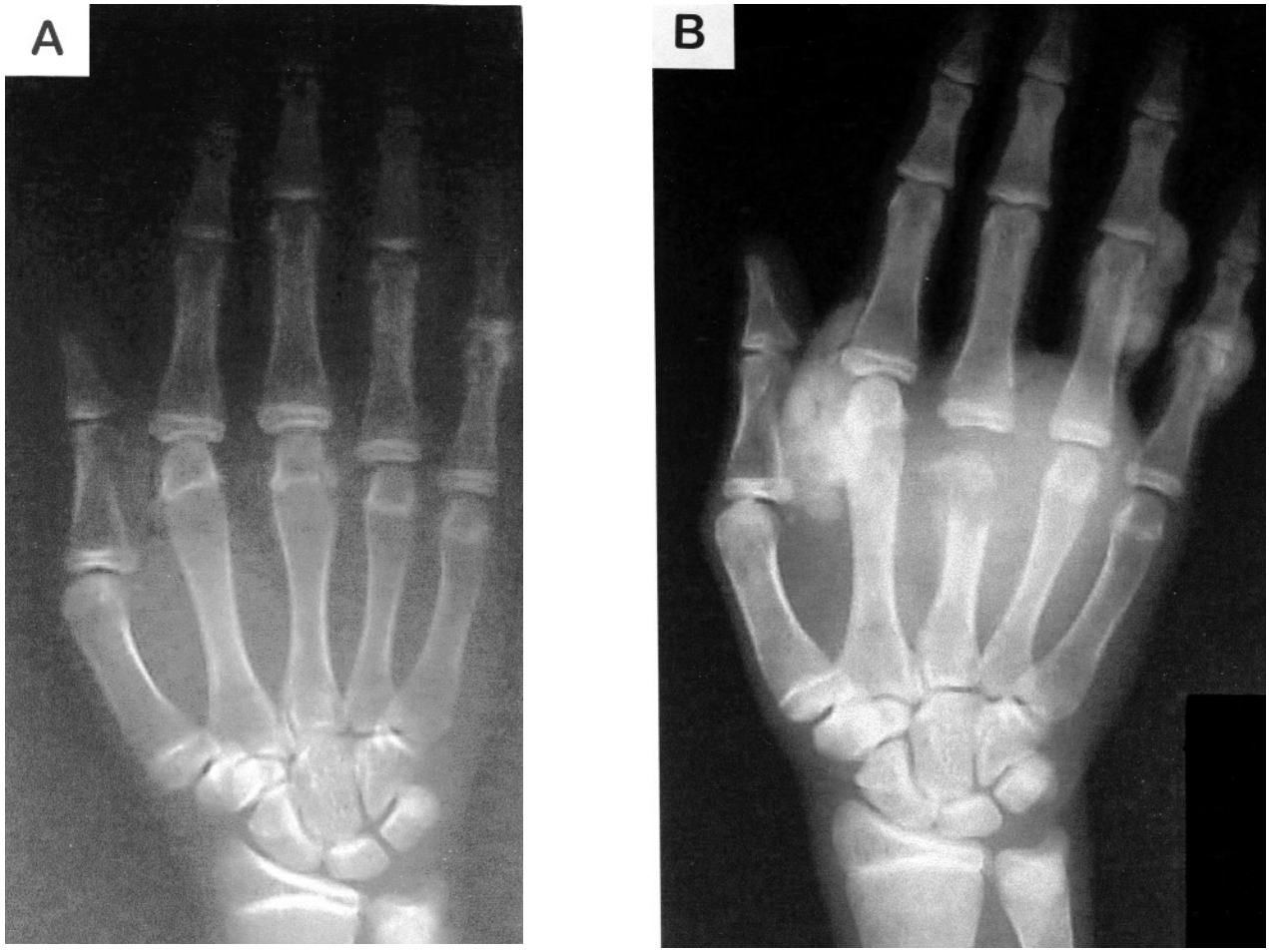

Figure 3. Plain radiograph of the right hand about 8 months after development of tophaceous deposits (A), and a followup radiograph taken during the patient's last admission 4 months later (B). Note rapid progression of periarticular calcifications and destruction of the right 3rd metacarpal head.

aspirations. No other organisms or crystals were found. He underwent surgical debridement of the right 4th MCP tophus, but he died from complications of disseminated TB.

DISCUSSION

This case of superinfection by *M. tuberculosis* of a tophaceous pseudogout nodule highlights the need for vigilance in diagnosing musculoskeletal TB. In 1997, 7.96 million new cases of TB were reported worldwide, with an estimated 3.52 million (44%) being AFB sputum smear positive, suggesting infectivity[6]. Developing countries bear 95% of all TB cases and 98% of all TB related deaths[7]. In 1997, the prevalence of active pulmonary TB in the Philippines was estimated at 42 per 1000 population[8].

In this patient, the presence of tender and fluctuant tophi strongly suggested a coexistent infection. Consequently, the relatively low white blood cell count in the aspirate, low percentage of neutrophils, and negative gram stain and culture could have led to a missed diagnosis of a septic tophus because AFB studies are not routinely done in such cases.

Repeat aspirations of the right 4th MCP tophus failed to reveal other infections or crystals. Destructive changes were only seen in the right 3rd metacarpal bone. Other imaged joints with chronic periarticular calcifications lacked erosions. The radiologic appearance of these periarticular CPPD deposits was consistent with other reviews[3]. Deposits of monosodium urate crystals may calcify, but these are unusual[9]. Although CPPD deposition disease may cause erosions[10], we believe that the destruction of the right 3rd metacarpal head was entirely due to *M. tuberculosis*.

The accelerated CPPD deposition could be explained by several factors. The recent deterioration in nutritional status and hypoalbuminemia could have resulted in a sustained rise in free ionized calcium and greater CPPD deposition. This is consistent with the reversal in serum parathyroid hormone levels from 150 pg/ml 4 months earlier, to a normal level of 29.5 pg/ml at the time of admission.

Both the initial and granulomatous inflammatory responses were probably defective in this patient, resulting in extrapulmonary tuberculous abscess formation. Poor

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nutritional status and chronic renal failure could have been contributory factors.

In summary: (1) the development of acute symptoms (abscess) in addition to preexisting chronic lesions (tophi) should trigger a vigilant search for infection; (2) a high index of suspicion is needed in diagnosing extrapulmonary TB in countries with high prevalence rates of TB infection; and (3) early diagnosis of musculoskeletal TB is critical because of the destructive result of this infection.

REFERENCES

1. Ilahi OA, Swarma U, Hamill RJ. Concomitant crystal and septic arthritis. Orthopedics 1996;19:613-7.
2. Co VM, Grimaldo ER, Rivera AB, et al. Tuberculous abscess: A suppurative response to Mycobacterium tuberculosis infection. JAMA Southeast Asia 1994:600-2.
3. Ishida T, Dorfman HD, Bullough PG. Tophaceous pseudogout (tumoral calcium pyrophosphate dihydrate crystal deposition disease). Hum Pathol 1995;26:587-93.
4. Hoppmann RA, Patrone NA, Rumley R, Burke W. Tuberculous arthritis presenting as tophaceous gout. J Rheumatol 1989; 16:700-2.
5. Lorenzo JP, Csuka ME, Derfus BA, et al. Concurrent gout and Mycobacterium tuberculosis arthritis. J Rheumatol 1997;24:184-6.
6. Dye C, Scheele S, Dolin P, et al. Consensus statement. Global burden of tuberculosis: Estimated incidence, prevalence, and mortality by country. WHO Global Surveillance and Monitoring Project. JAMA 1999;282:677-86.
7. World Health Organization. Global tuberculosis control — WHO report 1999. Geneva: World Health Organization; 1999. WHO/TB/99.259.
8. Tupasi TE, Radhakrishna, Rivera AB, et al. The 1997 Nationwide Tuberculosis Prevalence Survey in the Philippines. Int J Tuberc Lung Dis 1999;3:471-7.
9. Resnick D, Niwayama G. Gouty arthritis. In: Resnick D, Niwayama G, editors. Diagnosis of bone and joint disorders. 2nd ed. Philadelphia: Saunders; 1988:1618-71.
10. Schumacher HR. Crystal-induced arthritis: An overview. Am J Med 1996;100 Suppl 2A:46s-52s.

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