Case Report

Isolated Index Finger Dactylitis with Hilar Adenopathy

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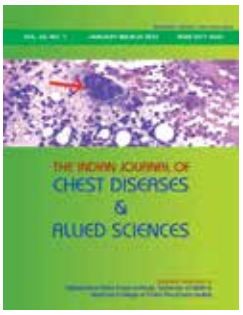

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ABBREVIATIONS USED IN THIS ARTICLE

TB = Tuberculosis

AFB = Acid-fast bacilli

MRI = Magnetic resonance imaging

FNAC = Fine needle aspiration cytopathology

H = Isoniazid

R = Rifampicin

Z = Pyrazinamide

E = Ethambutol

Abstract

Dactylitis due to tuberculosis (TB) is an extremely uncommon manifestation of TB. We report a unique case of isolated index finger dactylitis with right hilar adenopathy due to TB in a 15-year-old immunocompetent student. High index of clinical suspicion, radiological findings and histological confirmation helped in the diagnosis. Anti-tuberculous therapy remains the cornerstone for the management of these cases.

Introduction

Tuberculosis (TB) is the most widespread infectious disease throughout the world; especially in resource-limited countries like India. It commonly affects lungs; but can also involve uncommon sites. Skeletal TB represents 3% to 5% of all TB cases.[1] The involvement of fingers due to TB is an exceptionally uncommon phenomenon. The non-specific clinical features, insidious course and pauci-bacillary nature of the disease often mimics inflammatory arthritis resulting in delayed diagnosis. Histological confirmation by aspiration or biopsy of the affected finger is the sheet anchor for the diagnosis of TB dactylitis. The role of anti-tuberculous therapy is the main therapeutic armamentarium for the management.

Case Report

An adolescent student of high school presented with progressively increasing painful swelling of the right index finger and cough with minimal mucoid expectoration for the last three months. There was no history of fever, weight loss or loss of appetite, breathlessness and drug allergy. He denied history of trauma to finger. Patient was non-diabetic. He had no history of close contact with any TB patient. His family history was unremarkable.

On general physical examination, there were no enlarged lymph nodes. The examination of his hand showed a painful swollen finger at the level of the proximal inter-phalangeal joint of right index finger without inflammatory

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signs or ulcerations (Figure 1A). There was partial restriction of movements at that joint. Examination of respiratory system revealed no adventitious sound. There were no other significant systemic findings.

Routine blood haematological and biochemical tests were within normal limits. Serology for human immunodeficiency virus I and II was non-reactive. Radiograph of the right hand revealed lytic lesion at the base of the proximal phalanx of the index finger (Figure 1B). Chest radiograph (postero-anterior view) showed right hilar adenopathy (Figure 1C). Sputum smear examination for acid-fast bacilli (AFB) was negative. Mantoux test was strongly positive with ulceration. Ultrasound abdomen was unremarkable. Magnetic resonance imaging (MRI) of the hand showed expansile lytic lesion, tenosynovitis of flexor tendon of proximal phalanx, synovitis of metacarpo-pharangeal joint (heterogeneous signal intensity) with caseous necrosis and marrow oedema (hyperintense signal) involving proximal phalanx on T2 weighted images (Figure 1D).

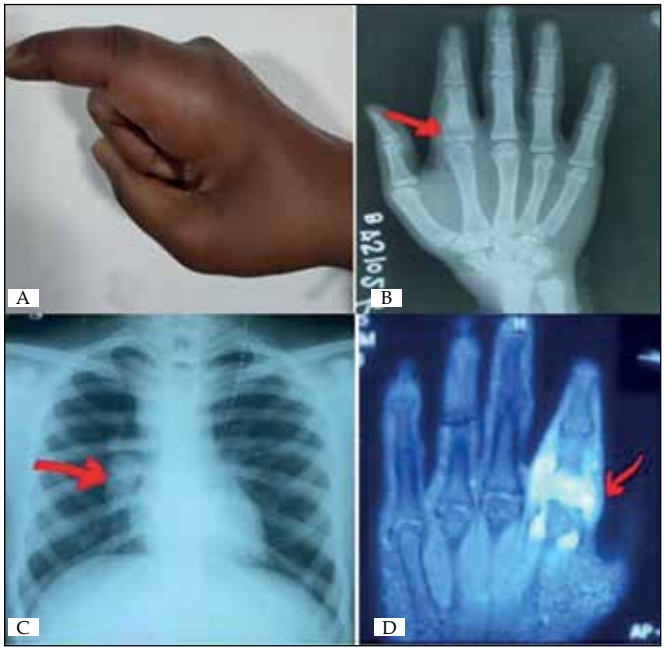

Figure 1. (A) Photograph of the hand showing swollen index finger of the right hand; (B) radiograph of the right hand showing lytic lesion at the base of the proximal phalanx of index finger; (C) chest radiograph (postero-anterior view) showing right hilar adenopathy and (D) magnetic resonance imaging of the right hand showing expansile lytic lesion, synovitis of metacarpo-pharangeal joint with marrow oedema and caseous necrosis involving proximal phalanx.

Fine needle aspiration cytopathology (FNAC) of finger swelling revealed tiny epitheloid cell granuloma with necrosis (Figure 2), compatible with TB. As FNAC from peripheral site was compatible with TB, further evaluation with bronchoscopy was not done. On the basis of clinical, radiological and cytopathological findings, a diagnosis of tubercular dactylitis of the right index finger with the right hilar adenopathy was made. Patient was started on anti-tuberculous therapy comprising of isoniazid (H), rifampicin (R), pyrazinamide (Z) and ethambutol (E) for initial intensive phase for two months followed by HRE for next four months.

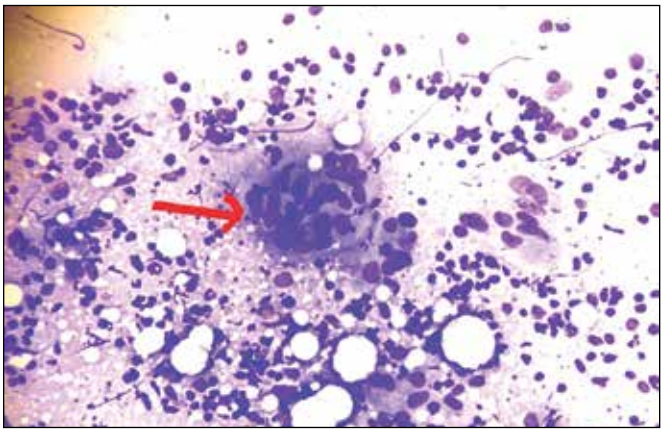

Figure 2. Fine needle aspiration cytopathology from the finger swelling showing epitheloid cell granuloma with necrosis (Haematoxylin and Eosin x 200).

Patient improved clinically as well as radiologically. On follow-up, finger swelling reduced (Figure 3A), with improvement in the movements at index finger. Subsequently, radiograph of the hand (Figure 3B) and chest (Figure 3C) also showed improvement.

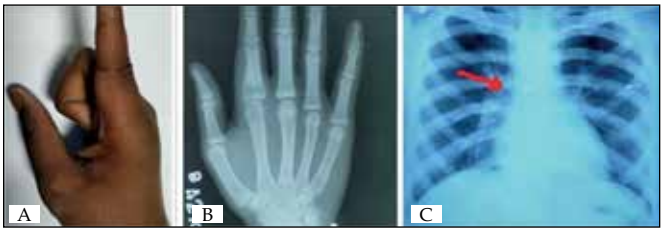

Figure 3. (A) Follow-up photograph of the hand after anti-tuberculosis therapy showing improvement in finger swelling; (B) radiograph of the right hand taken after treatment showing improvement and (C) chest radiograph (postero-anterior view) after the treatment showing resolution of the right hilar adenopathy.

Discussion

Dactylitis due to TB, also known as spinaventosa, is a very rare form of extra-pulmonary TB affecting the small bones of the hands or the foot. The first anatomical description of spinaventosa was in 1803 by Boyer. Tubercular aetiology of spinaventosa was first proven by Nelaton in 1837.

Eighty-five percent cases of tubercular dactylitis occur in children less than six years of age.[2] It has been

postulated that the haematopoietic marrow in the small bones among pediatric age-group offers an ideal substrate for haematogeneous bacterial implants. The occurrence of tubercular dactylitis in adolescent and adults is extremely rare and limited to scattered case reports only.[3] Tubercular dactylitisis reported three times more among males as compared to females.

Immunodeficiency, malnutrition and poor socio-economic status have been reported as the common predisposing factors for tubercular dactylitis.[4] Often, there is prior history of trauma; but our patient denied any history of trauma. The exact mechanism of tubercular dactylitis is not fully understood. However, the proposed mechanisms include: (i) haematogenous dissemination after pulmonary TB and (ii) direct inoculation.[5]

The clinical course of the disease is insidious with non-specific symptoms resulting in delayed diagnosis. Usually, patients present with progressively increasing finger swelling with minimal or no pain. Joint mobility is usually preserved. The most frequently proximal phalanx of the second and the third fingers are affected.[6] In our patient, there was progressively increasing painful swelling and restriction of movement of the right index finger with the right hilar adenopathy.

The differential diagnosis for tubercular dactylitis are inflammatory arthritis, septic arthritis, osteoid osteoma, chondroma, pyogenic osteomyelitis, sarcoidosis, fungal infections and other granulomatous infections. In inflammatory arthritis, cartilaginous followed by subchondral bony erosions occur. Pyogenic osteomyelitis is differentiated from tubercular by the absence of periosteal reaction and sequestrum. There are florid signs of inflammation, such as high grade fever in cases of septic arthritis. Sarcoidosis affects multiple organ systems with "lacy" lytic like intra-osseous abnormalities. Enchondromas are benign asymptomatic tumours, typically seen as well defined central lucent expansile lesions without periosteal reaction or cortical destruction. Fungal infections have minimal or absent periosteal reaction with multiple granule draining sinuses.

The diagnosis of tubercular dactylitis requires high index of clinical suspicion, especially in TB endemic countries like India. The Mantoux intradermal tuberculin test is usually positive; but negative Mantoux does not exclude TB. In our case, Mantoux test was strongly positive and ulcerative.

The radiological findings in tubercular dactylitis are non-specific in nature and often there are osteolytic lesions without periosteal reaction. The absence of periosteal reaction characterises TB osteitis.[7] MRI of the affected finger shows tenosynovitis, synovitis, marrow oedema and osteolytic lesions. It may also show the typical finding of caseous necrosis on contrast study.

Histopathology typically shows epitheloid cell granuloma and caseous necrosis, as was observed in our case. The Gold standard for the diagnosis of TB is the demonstration of *Mycobacterium tuberculosis* on culture. However, as this is a pauci-bacillary disease, AFB cannot always be identified. The anti-tuberculosis therapy remains the cornerstone for the management of tubercular dactylitis. Surgical indications are limited to biopsy to confirm the diagnosis.

Our case of TB dactylitis was unique in many ways: (i) adolescent age, (ii) no evidence of immunodeficiency, (iii) no history of trauma prior to onset, (iv) presence of another site of TB (right hilar adenopathy) and (v) MRI of the fingers showing findings of tenosynovitis, marrow oedema with typical caseous necrosis.

In conclusion, TB dactylitis is an extremely rare form of extra-pulmonary TB. Although it commonly affects children less than six years of age; but can occur in adolescents and adults as well. There is often diagnostic delay due to non-specific symptoms and clinico-radiological discordance. The diagnosis often requires histopathological confirmation. High index of suspicion, especially in TB endemic countries is essential in early diagnosis and prompt treatment for a better outcome.

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