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Cutaneous Tuberculosis Mimicking Cellulitis in an Immunosuppressed Patient

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ABSTRACT

A 28-year-old lady suffering from systemic lupus erythomatosus (SLE) with diffuse proliferative glomerulonephritis (DPGN) and who was on oral cyclophosphamide and prednisolone presented with left lower limb 'cellulitis'. The 'cellulitis' of the left lower limb failed to respond to usual antibiotics which prompted evaluation of the clinical diagnosis. The diagnosis is made based on the presence of granulomas, multinucleated giant cells and acid fast bacilli on the skin biopsy.

Keywords: cutaneous tuberculosis, cellulitis, immunosuppressed

INTRODUCTION

Cutaneous tuberculosis (TB) is a relatively rare clinical entity in Western countries but relatively common in the Far East (approximately 0.4%)[1]. It may manifest itself in many different forms depending on the differences in the number and virulence of the bacilli, route of infection, age of the patient, previous history of exposure or infection with mycobacterium tuberculosis (Myco TB) and the immunity and the hypersensitivity reaction of the host. The cutaneous lesions may range from papules, nodules, plaques, ulcers, verrucous lesions, papillomatous tumour, vegetative reaction or cicatricial infiltrates.

The most common cutaneous TB encountered are lupus vulgaris[2,3]. However, Wong et al found a higher incidence of tuberculosis verrucosa cutis as compared to lupus vulgaris in his study population[1]. The classification of cutaneous TB remains unsatisfactory as described by most of the authors.

This report describes a case of cutaneous TB of unclassified subgroup presenting as left lower limb cellulitis in an immunosuppressed patient.

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CASE REPORT

A 28-year-old lady presented with acute onset of pain in the left leg and redness associated with fever for three days prior to admission on 1/2/1995. There were no preceding trauma and other systemic symptoms.

She was first diagnosed to have SLE in July 1992 based on clinical presentations and serology results and was treated with prednisolone. She had a clinical relapse with nephrotic syndrome in October 1994. The renal biopsy was consistent with DPGN. She was then started on oral cyclophosphamide 100 mg and prednisolone 60 mg daily. She was on cyclophosphamide for three months and prednisolone 40 mg when she presented with the current problem.

She had no symptoms suggestive of active SLE nor any previous history of pulmonary TB

or recent contact with tuberculous infected patients.

On clinical examination, she was febrile, pale, cushingoid facies but no pedal oedema was visible. The main finding was confined to the left lower limb. Her left leg was warm, tender and inflammed. The erythematous area extended diffusely from the dorsum of the left foot to the left leg though the margin was not raised. Nodules, sinus and lymphadenopathy were absent. Examination of the other systems yielded normal results.

Initial investigations revealed that she had anaemia (haemoglobin of 7g/dL), normal white cell count (5700/dL), thrombocytosis (platelet count of 511,000/dL) and mild renal impairment (urea 9.7mmol/L), creatinine 145 umol/L). Her liver function was normal except for hypoalbuminaemia of 19g/L. The erythrocytes sedimentation rate and c-reactive protein were markedly raised (> 140 mm/1st hour and 29.2 mg/dL respectively). Her urine analysis showed proteinuria of 1+ and some red and white blood cells. The serology marker for SLE was not suggestive of active disease. She had normal complements level, ANF titre of 1:80 and dsDNA negative (as compared with strongly positive titre in October 1994). Her blood culture was negative and chest radiography was normal.

She was empirically started on crystalline penicillin and cloxacillin. Cyclophosphamide was withheld and prednisolone was initially maintained at the same dose then gradually reduced. Antibiotic treatment was changed from penicillin and cloxacillin to ceftazidine and unasyn because of poor clinical response. A lesion on her left lower limb worsened and extended further to the medial aspect of the left thigh with uneven surfaces suggestive of abscess formation. Ultrasonography of the left lower limb showed some areas of fluid collection in the left thigh and leg. An incision and drainage was attempted revealing only fluid collection. Antibiotic treatment was changed to ciprofloxacin; wound culture was normal.

However, her left lower limb cellulitis did not resolve with ciprofloxacin. A skin biopsy of the involved skin two weeks after the incision and drainage revealed presence of granulomas deep in the dermis and the subcutaneous tissue. It composed of epitheloid cells, multinucleated giant cells and a few neutrophils. There was flattening of the epidermis with infiltration of the nerve fibres and blood vessels by the mononuclear cells. Ziehl Neelson stain showed presence of acid fast bacilli. Histologic findings were consistent with cutaneous TB (Fig 1).

The final diagnosis is SLE, clinically inactive with cutaneous TB.

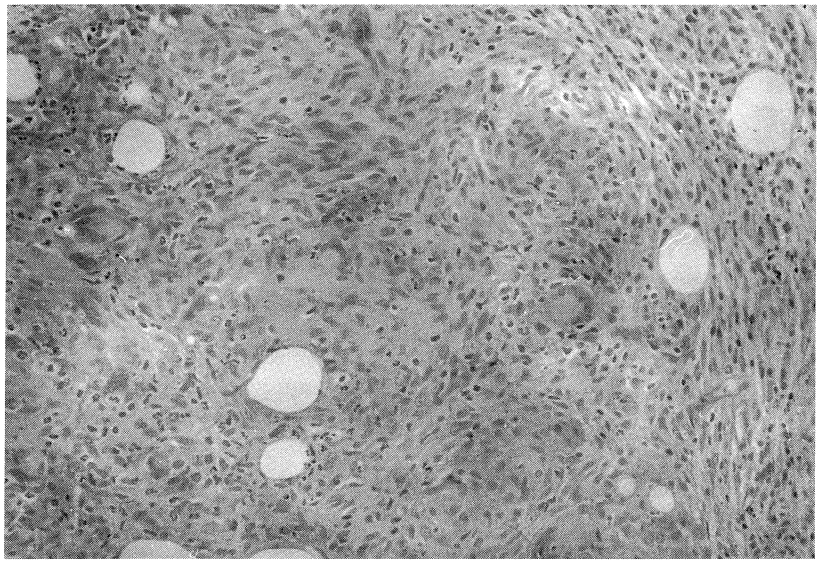

Fig 1 – Histology finding consistent with cutaneous TB.

DISCUSSION

This case represents an unusual form of 'cellulitis' occurring in an immunosuppressed patient who did not respond to antibiotics treatment. This raised the possibility of mycobacteria, atypical mycobacteria and various form of fungal infection[7].

The diagnosis of cutaneous TB requires a high index of suspicion with a correlation in the histologic and culture results and a vigilant search for other possible sites of tuberculous infection. Cutaneous TB lesions may comprise of a multitude of skin changes. Although agreed by most of the authors, there is no satisfactory existing classification of cutaneous TB[1-4]. Generally, two main types of cutaneous TB are identified: 1) true bacterial invasion of the skin and 2) tuberculid or hypersensitivity reaction associated with a primary focus elsewhere[2-4,7]. True cutaneous TB consists of primary chancre, lupus vulgaris, tuberculous verrucosa cutis, scrofuloderma, tuberculosis cutis orificialis and tuberculous cutis miliaris disseminates[2,4,7].

This patient's skin lesion did not fit into any of the above form of cutaneous TB. This 'cellulitis' form of cutaneous TB is probably due to the bacterial invasion of the skin which has not been described in the literature. The rapid clinical progression of the skin changes in her lower limb is an unusual feature of cutaneous TB indicating her poor cellular immune response either to the immunosuppressive therapy or her underlying SLE[9]. There have been reported cases of cutaneous TB with rapid progression in patients with SLE on corticosteroid[8].

With recent advances in modern chemotherapy, TB can be treated with a six-month regimen consisting of isoniazid, rifampicin, pyrazinamide and ethambutol for the first two months follows by four months of isoniazid and rifampicin for immunocompetent patients. However, immunocompromised patients including those on immunosuppressive therapy will require nine months of antituberculous treatment[6]. Our patient was started on the four-drug regimen for two months followed by isoniazid and rifampicin. She responded well to the antituberculosis treatment. At the end of the second month of antituberculosis treatment, her left leg lesion had almost healed completely.

In summary, cutaneous TB can present atypically in patients who are immunocompromised, on immunosuppressive therapy and those with underlying SLE.

REFERENCES

1. Wong KO, Lee KP, Chiu SF. Tuberculosis in Hong Kong. Br J Derm 1968; 80:424-9.
2. Brown TS, Anderson RH, Burnett JW. Cutaneous tuberculosis. J Am Acad Dermatol 1982; 6:101-6.
3. Ramesh V, Misra RS, Jain RK. Secondary tuberculosis of the skin: clinical features and problems in laboratory diagnosis. Int J Dermatol 1987; 26:578-81.
4. Chen CH, Tsai JJ, Shih JF, Perng RP. Tuberculous subcutaneous abscesses developing during chemotherapy for pulmonary tuberculosis. Scand J Infect Dis 1993; 25: 149-52.
5. Wortman Paul D. Pulmonary and cutaneous tuberculosis. J Am Acad of Dermatol 1992; 27(3):459-60.
6. American Thoracic Society and the Centres for Disease Control. Treatment of tuberculosis and tuberculosis infection in adults and children. Am J Resp in Crit Care Med 1994; 149:1359-74.
7. Samuel LM, Harry JH eds. Diseases of the mononuclear phagocytic system: Tuberculosis of the skin. In: Dermatology 3rd ed. 1992:1089-100.
8. Pablos JL, Poveda MJ, Ciruelo E, Palerque E, Alonso J, Mateo I. Cutaneous infection with rapidly growing mycobacteria in patients with systemic rheumatic disease. Clin Exp Rheumatol 1994; 12(5):535-7.
9. American Thoracic Society and the Centres for Disease Control. Diagnostic standards and classification of tuberculosis. Am Rev Respir Dis 1990; 142:725-35.