

NODULAR SKIN TUBERCULOSIS WITH LYMPHATIC SPREAD – A CASE REPORT

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ABSTRACT

An unusual case of tuberculosis paronychia with skin infection of the big toe was recently seen in a patient returning from Kalimantan. This was complicated by inguinal lymphadenitis and tuberculosis abscess formation. The diagnosis was made on culture of the pus from the abscess and upon biopsy and histological examination of the skin lesion from the toe. The patient responded to surgical treatment and chemotherapy with ethambutol, rifampicin and isoniazid.

Keywords: tuberculosis, paronychia, lymphatic spread, lymphadenitis, abscess formation

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CASE REPORT

The patient, a 33-year-old Indian male, first presented to Toa Payoh Hospital in January 1992. He had returned from Kalimantan, Indonesia 3 weeks earlier, and had developed an infection of the right toe upon return. This was in the form of paronychia which discharged pus, along with formation of a skin nodule on the big toe. The infection failed to resolve with potassium permanganate wash and oral antibiotics. Four days after the start of the toe infection, the patient also noted pain and swelling in the right inguinal region. He was given ampicillin and cloxacillin for 6 days at the outpatient clinic, but the inguinal swelling worsened, and he developed fever on and off. There was no cough, nor recent loss of weight or appetite. Other than a past history of torn left meniscus, there was no medical history of note.

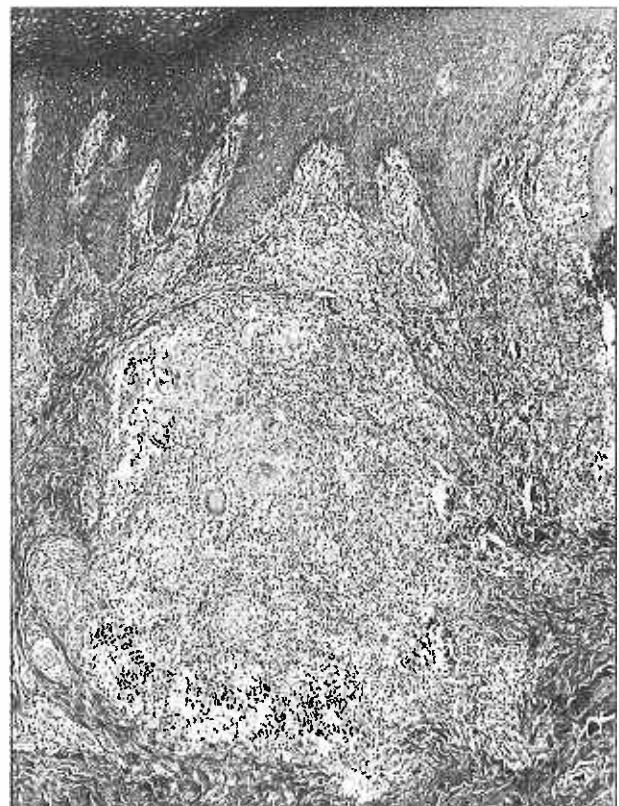
Clinically, no other sites of lymphadenopathy were found. Cardiopulmonary examination was normal. There was no hepatosplenomegaly. The total white cell count was 12,400 (polymorphs: 78%, lymphocytes: 16%); fasting and 2 hour post-prandial blood sugar levels were normal. Urine microscopy revealed no pyuria. Blood culture showed no bacterial growth. A chest X-ray revealed no infective changes. Mantoux test was done but was complicated by suspected skin reaction to cleaning agent.

He was given a course of co-trimoxazole in the hospital and his symptoms improved. However upon follow-up in the outpatient clinic, it was found that the right inguinal nodes had become fluctuant. In late March 1992, incision of his groin

abscess was done. Pus and necrotic material were drained and a portion of the abscess wall was sent for histology. This showed acute-on-chronic inflammation with an area of caseous necrosis, and the presence of multinucleated giant cells. Stains for acid-fast bacilli were negative, but culture of the pus grew *Mycobacterium tuberculosis* sensitive to streptomycin, isoniazid, rifampicin and ethambutol. Chemotherapy was started with the last 3 drugs 4 months after presentation.

During follow-up, attention was drawn to the skin nodule over the right big toe, which still persisted. In June 1992, he had excision biopsy of the nodule. This was reported as showing chronic inflammation of the dermis. A granuloma was seen, and there was a central aggregate of polymorphs rimmed by epithelioid histiocytes and occasional Langhan's type giant cells. Ziehl-Nielsen stain was negative. The appearance was consistent with tuberculosis. (Fig 1)

Fig 1 – The dermis shows epithelioid granulomas with lymphocytes and occasional giant cells. The epidermis shows hyperplasia. (Original magnification x 40)



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Final Diagnosis: Tuberculosis infection of the skin of the right big toe with later development of right inguinal tuberculosis lymphadenitis.

DISCUSSION

Cutaneous tuberculosis is usually caused by *Mycobacterium tuberculosis*, *Mycobacterium bovis* and rarely, *Bacillus Calmette-Guerin*. The portals of entry for *Mycobacterium tuberculosis* are usually the lung, and in decreasing frequency, the tonsils and the intestine. Rarely is the portal of entry the skin or the mucous membranes. Lymphatic spread to the regional lymph nodes and then the blood stream occurs rapidly in the latter instance.

In the classification of cutaneous tuberculosis (Table I) used by Kakakhel^(1,2), inoculation tuberculosis takes three forms, namely, tuberculosis chancre, warty tuberculosis (nodular tuberculosis, tuberculosis verrucosa cutis) and lupus vulgaris. Nodular tuberculosis is the most common form of skin tuberculosis in Asia, and it occurs from the inoculation of the organisms into the skin of a person who has moderate-to-high immunity from previous infection or immunisation⁽³⁾. While it tends to occur on the hands in Europeans, the pattern in Asians consists of involvement of the knees, ankles and buttocks⁽³⁾. Inoculation usually occurs at the sites of minor abrasions or wounds, as illustrated in our patient.

Seghal et al⁽⁴⁾ describes the initial lesion as an asymptomatic small papule or papulopustule with an inflammatory areola that develops at the site of inoculation. It later becomes hyperkeratotic, warty and develops into a verrucous plaque with a horny surface with deep clefts and fissures. The plaque is usually firm, but later areas of softness may occur, with pus and keratinous material being expressed from the fissures. Spontaneous involution may occur. More often, atrophic scars result. Other forms that may occur include fungating granulomas, tumour-like forms^(5,6), wart-like forms, psoriform plaques and keloidal forms. Seghal and Kakakhel contend that regional lymphadenitis is rare and is often pyococcal. However, we managed to culture *Mycobacterium tuberculosis* successfully from our patient's inguinal lymph node abscess.

The histologic picture⁽⁴⁾ consists of hyperkeratosis, hypergranulosis, acanthosis and papillomas overlying an acute inflammatory infiltrate in the epidermis. Tuberculoid granulomas with moderate amount of caseation necrosis and few tubercle bacilli are seen in the mid-dermis. Marked fibrosis occurs consistently.

The demonstration of tubercle bacilli in the smear and/or tissue section confirms the diagnosis but a limitation, as shown in Seghal's study, is the failure to detect the bacilli in 87% of patients with lupus vulgaris and in about 50% with other variants⁽⁷⁾. Besides acid fast staining, fluorescent staining with auramine or rhodamine is also available and more sensitive. Most authors report difficulty in culturing the *mycobacterium* or demonstrating the tubercle bacilli on histology. Immunologic diagnosis may be established by the tuberculin test and ELISA assays for antibody to *M. tuberculosis* antigen (Table II).

Recent regimes for cutaneous tuberculosis are outlined in Table III. Currently, short course regimes of 6 to 9 months duration are favoured. Surgical excisions of these small lesions are useful adjuvants to chemotherapy.

Table I – Classification of cutaneous tuberculosis

Type Of Tuberculosis	Clinical Appearance
Inoculation tuberculosis (Exogenous source)	Tuberculosis chancre Warty tuberculosis Lupus vulgaris (some)
Secondary tuberculosis (Endogenous source)	Contiguous spread (scrofuloderma) Auto-inoculation (orofacial tuberculosis)
Hematogenous tuberculosis	Acute miliary tuberculosis Lupus vulgaris (some) Tuberculosis gumma
Eruptive tuberculosis	Lichen scrofulosorum Papular or papulonecrosis tuberculosis

Table II – Criteria for diagnosis of cutaneous tuberculosis

Criteria	Methods of Diagnosis
Absolute criteria	– culture – guinea pig inoculation
Relative criteria	– history and signs – presence of active proven tuberculosis elsewhere in the body – presence of acid-fast bacilli in the lesion – histopathology – positive reaction to tuberculin – effect of specific therapy

Table III – Chemotherapy regimes for skin tuberculosis

Chemotherapy Regimes	Schedules
Conventional continuous chemotherapy	<u>Initial 3 months</u> Three drugs – isoniazid, streptomycin, para-amino salicylic acid <u>Next 18 months</u> Two drugs – isoniazid, para-amino salicylic acid
Short course continuous chemotherapy	1) <u>Daily for 6 months</u> Isoniazid, rifampicin, (add daily for first two months, ethambutol and pyrazinamide)
	2) <u>Daily for 6 months</u> Isoniazid, rifampicin, (add daily for first two months, streptomycin and pyrazinamide)
	3) <u>Daily for 9 months</u> Isoniazid, rifampicin, (add daily for first two months, ethambutol)

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