

A Case of Atypical Lupus Vulgaris Developing at a Skin Graft Site

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Lupus vulgaris is most prevalent on exposed parts, especially the face but can also develop on extremities. Lupus vulgaris originates from tuberculosis elsewhere in the body by hematogenous, lymphatic, or contiguous spread.

A 19-year-old male patient came to our department. The patient had had many recurrent oozing and verrucous plaques and crusts on the left foot for one year. A skin biopsy from the lesion on the left dorsum of the foot showed scattered well defined granulomas consisting of the epithelioid cell clusters with Langerhans and foreign body type giant cells in the mid dermis. Caseation necrosis was slight. There were no bacilli on AFB staining. The multi test CMI for tuberculin was highly positive. A chest X-ray did not show any abnormal findings. The presence of *Mycobacterium tuberculosis* DNA was demonstrated by polymerase chain reactions (PCR) for detection of mycobacterial DNA from a routinely prepared paraffin-embedded skin specimen. Herein we report a very atypical case of lupus vulgaris confirmed by PCR.

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Key Words : Atypical lupus vulgaris, PCR Skin graft

Lupus vulgaris, which is a progressive form of postprimary tuberculosis in patients with a moderate to high degree of immunity and tuberculin sensitivity, is the most common, most serious and most variable type of cutaneous and mucous membrane tuberculosis¹. The characteristic lesion is a reddish-brown plaque with peripheral nodules that are deeply embedded and are about 1 mm in size and yellowish in color². This may also appear clinically as ulcers, nodules, hypertrophic verrucous or vegetative plaques, sclerotic masses, papillomatous overgrowths and edematous thickenings¹. It involves the head and neck most frequently^{3,4}. Here we report a case of atypical lupus vulgaris appearing clinically as a papillomatous overgrowth on a skin graft site.

REPORT OF A CASE

A 19-year-old male patient visited our department because of many oozing and verrucous plaques and crusts with a recurrent tendency on the left dorsum of foot. This had been present for one year. There were no subjective symptoms except an itching sensation. 10 years ago, he had received an injury with deep laceration on this left ankle, heel and lower leg after a traffic accident and he had an STSG (split thickness skin graft). After one year, the erythematous and eczematous papules developed on the skin graft site and was treated privately. 3 years ago, the erythematous papules on the left foot spread slowly and confluent each other. At the time of his visit to our department, exudative erythematous papillomatous plaques were seen on the left dorsum of the foot. The lesion was covered with thick, partly bleeding yellowish crusts to the condition of minor trauma (Fig. 1). His family history was non-contributory and he

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Fig. 1. Localized multiple erythematous papillomatous plaques & erosions with exudate on the left dorsum and sole of the left foot.

had no history of pulmonary tuberculosis.

The results of laboratory tests including CBC, urinalysis, liver function test, serum electrolytes, fasting blood sugar, BUN/creatinine and VDRL were negative and within normal limits. The multi test CMI for tuberculin was highly positive. A chest X-ray was insignificant and direct KOH examination and fungal culture of materials from the skin lesion revealed negative findings.

A skin biopsy from the lesion on the left dorsum of the foot showed scattered well defined granulomas consisting of epithelioid cell clusters with Langerhans and foreign body type giant cells in the mid dermis. Caseation necrosis was slight (Fig. 2). There were no

Fig. 2. Histologically, the mid dermis shows a solitary well demarcated round granulomatous lesion. The peripheral part of the lesion is surrounded with lymphocytes, epithelioid cells & multinucleated giant cells. (H&E stain, A \times 40, B \times 200).

bacilli on AFB staining. Mycobacterium tuberculosis DNA was demonstrated by polymerase chain reactions (PCR) for detection of mycobacterial DNA from routinely prepared paraffin-embedded skin specimens (Fig. 3). For two step-nested PCR, we used the two set of primers, P1 5'-CGT-GAG-GGC-ATC-GAG-GTG-GC-3', P2 5'-GCG-TAG-GCG-TCG-GTG-ACA-AA-3', P3 5'-GAA-CGG-CTG-ATG-ACC-AAA-CT-3', P4 5'-ACG-TAG-GCG-AAC-CCT-GCC-CA-3'. Rounds of amplification consisted of a denaturation step at 94 °C for 20 sec, an annealing step at 65

Fig. 3. PCR detection of *M. tuberculosis* DNA in tissue from the patient.

Lane 1,2 : tissue samples from patient
Lane 3 : 100 bp size marker
Lane 4 : negative control
Lane 5 : positive control

°C for 20 sec and an extension step at 72 °C for 45 sec. After 40 cycles, the samples (μ l) were used for the other PCR with the same condition, and then analyzed by electrophoresis.

He was treated with isoniazid 300 mg/day, rifampicin 600 mg/day and ethambutol 800mg/day for 5 months. At present, the skin lesions are markedly improved (Fig. 4).

DISCUSSION

Cutaneous tuberculosis has a wide range of clinical manifestations, varying from warty or granulomatous nodules, papulonecrotic lesions, to ulceration or abscess⁵. Lupus vulgaris is the most common type of cutaneous tuberculosis¹. It is characterized by groups of reddish brown nodules which, when blanched by diascopic pressure, have a pale brownish yellow or "apple-jelly" color. They tend to heal slowly in one area and progress in another. The nodules are minute, translucent and embedded deeply and diffusely in the infiltrated dermis, forming plaques by the coalescence and development of new lupus nodules⁴. This disease is destructive, frequently causes ulceration, and on involution, leaves deforming scars as it slowly spreads peripherally over the years. Lupus vulgaris is

Fig. 4. After treatment with INH, rifampicin, EMB for about 5 months, thick erythematous plaques are decreased in its sizes and exudates are subsided on the dorsum and sole of left foot.

most prevalent on exposed parts, especially the face but can develop on extremities³. The course of lupus vulgaris is so slow that a patch of the disease may remain limited to a small area for several decades, while showing signs of activity. A lupus patch may start in childhood and persist throughout a lifetime. During this period it may slowly spread, or new patches may develop in other regions. In some instances the granulations become papillomatous, vegetative, fungoid or thickly crusted⁴. However, like our case, lupus vulgaris developing at a skin graft site showing exudative erythematous papillomatous plaques is very rare⁶.

For diagnosis, a skin biopsy and AFB staining is needed. However, in some AFB staining of skin

tissue, no bacilli can be shown. In this case, PCR is a useful diagnostic plan^{7,9}.

Histopathologically, the infiltrate, which is diffuse, is made up of tubercles or tuberculoid granulomatous inflammation consisting of epithelioid and giant cells, usually of the Langhans type, and lymphocytes, plasma cells, and occasionally, polymorphonuclear leukocytes. Caseation necrosis within the tubercle is slight and may be absent. The tubercle bacilli is difficult to demonstrate with special stains. Also, attempts to culture the organism are usually unsuccessful^{3,4}. In our case, a skin biopsy from the lesion on the big toe showed scattered tuberculoid granulomas composed of epithelioid cell clusters with Langerhans and foreign body type giant cells in the dermis. Caseation necrosis was slight. There were no bacilli in the AFB stain. The cultures of tuberculosis bacilli were negative.

Therefore, for the purpose of exact and rapid diagnosis, PCR was done, and *Mycobacterium tuberculosis* DNA was detected in the tissue of the skin lesion^{1,10}.

When we carried out our initial examination, clinically we thought squamous cell carcinoma, verrucous carcinoma or blastomycosis might be present. However, we made a diagnosis through histological findings and PCR. We think that especially, in our case which showed atypical skin lesions, the detection of *Mycobacterium tuberculosis* DNA using PCR can give much help for the early diagnosis and treatment of lupus vulgaris^{8,9}.

Isoniazid in combination with rifampicin is the initial treatment of choice. The isoniazid dosage is 300mg a day. Rifampin is a standard concurrent treatment for pulmonary tuberculosis and is worthwhile giving in cutaneous tuberculosis as well. Though nine months treatment is standard for most cases of pulmonary tuberculosis, clinical recovery plus a reasonable margin is probably enough for lupus vulgaris¹¹. Streptomycin, though potentially effective, is best avoided because of the risk of ototoxicity. Ethambutol is rarely used¹². Our case was treated with isoniazid 300 mg/day, rifampicin 600 mg/day, and ethambutol 800mg/day for 5 months. After treatment, thick erythematous plaques had decreased in size and exudates had

subsided on the dorsum and sole of the left foot.

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