

A Case of an Atypical Mycobacterial Ulcer Following an Intramuscular Injection

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Citation

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Abstract

Buruli ulcer is a chronic, indolent, necrotizing disease of the skin and soft tissue. Buruli ulcer is the third most common mycobacterial disease of immunocompetent hosts, after tuberculosis and leprosy, and is caused by toxin-producing mycobacteria named *Mycobacterium ulcerans*. Over the last 2 decades, a re-emergence of cases has occurred, leading to the 1998 World Health Organization (WHO) Buruli Ulcer Initiative and the Fifty-Seventh World Health Assembly Resolution on Buruli Ulcer, which have stimulated ongoing research into diagnosis, pathogenesis, and effective treatment.

CASE REPORT

A 16-year-old male came with the complaint of a non-healing ulcer over the left arm in the deltoid region for three months. Three months ago he had got an injury over his foot for which he had visited a local doctor. The doctor administered him a dose of tetanus toxoid by intramuscular route in the deltoid muscle.

However, two days after this, he noticed a swelling at the injection site. Initially, it was small, but over 2-3 days it attained the size of about 0.5cm in diameter. He was diagnosed as a case of injection site abscess and the abscess was drained at a private clinic. The culture-sensitivity report of the pus was not available to us.

Later, the patient developed a non-healing ulcer over the site. There was no history of persistent trauma and there was no loss of sensation over the affected part. He did not give any previous history of tuberculosis or leprosy and he is a non-smoker and non-alcoholic.

On examination, there was a single oval ulcer of about 0.5 x 0.3 x 0.1cm over the left arm in the deltoid region. There was purulent discharge and slough with unhealthy pale granulation tissue. The edges of the ulcer were undermined and the margin was indurated. The base was the subcutaneous fat. Left-sided axillary lymph nodes were palpable. No other stigmata of tuberculosis were visible.

Figure 1

Fig 1: Atypical mycobacterial ulcer with undermined edges



On investigation, his ESR was raised. The rest of the blood investigations were within normal limits. He tested negative for HIV [I and II] viruses.

The imprint cytology report showed suppurative granulomatous inflammation with possibility of atypical mycobacteria. The histopathology report confirmed the presence of an atypical mycobacterial granuloma.

The patient was started on a combination of clarithromycin, rifampicin and ciprofloxacin orally and is responding well to the treatment.

DISCUSSION

Buruli ulcer was first described by Sir Albert Cook in patients from Buruli County in Uganda, and the causative organism was isolated in 1948 by MacCallum in the Bairnsdale region of Victoria, Australia.

M. ulcerans are slow-growing mycobacteria that affect the skin and the mucous membranes. The organism produces a soluble polyketide toxin called mycolactone. Mycolactone has both immunosuppressive properties and cytotoxic properties, which explains the lack of host symptoms, such as fever, malaise, or adenopathy. [1,2] Mycolactone is responsible for the extensive tissue necrosis seen in Buruli ulcers.

Genetic susceptibility is a possibility, associated with the SLC11A1 (NRAMP1) D543 polymorphism. [3]

The incubation period ranges from a few weeks to months.

Lesions usually begin as a single, painless, occasionally pruritic, dermal papule or subcutaneous nodule.

Suppuration and ulceration occur within 1-2 months. Approximately 90% of lesions occur on the limbs, with 60% occurring on the lower limbs.

In the preulcerative stage, Buruli ulcers manifest initially as firm, non-tender, subcutaneous nodules 1-2cm in diameter. The ulcerative stage occurs days to weeks later with the formation of an ulcer with undermined edges.

Ulcerations are generally painless unless complicated by secondary infection. The Buruli ulcers may destroy nerves, appendages, and blood vessels and may invade bone. A few studies have shown relatively high frequencies of bone involvement (15% of patients). Metastatic lesions may occur in skin, soft tissue, or bone via spread through the vasculature or lymphatics.

Healing is a slow process that often results in cosmetically disfiguring scars and functional disabilities.

Although the exact mode of transmission is unknown, *M. ulcerans* most likely causes infection through contamination of a traumatic wound [4].

The medical management of Buruli ulcers is an active area of research to determine the most effective combination and duration of treatment with antimicrobials. Treatment has been shown to promote healing of smaller lesions and as an adjunct to surgical management to decrease recurrence. The best outcomes occur when treatment is initiated in smaller lesions (<5cm).

In 2004, the WHO recommended a treatment protocol that divided lesions into 3 categories. The WHO recommends that all categories receive a course of rifampicin and streptomycin. Recurrence rates after antibiotic treatment are reported to be 2-3%. [5,6]

In 2007, the Australian Victorian Department of Human Services recommended the combination of rifampicin and clarithromycin or ciprofloxacin or moxifloxacin for 3 months. In severe disease, oral rifampicin with intravenous amikacin is the treatment of choice. Oral medications should be used for 12 weeks, and intravenous amikacin should be used for 4 weeks

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