


**Cutaneous Mycobacterium avium Complex Infection at an Intramuscular Injection Site in a Patient with AIDS**

*Mycobacterium avium* complex (MAC) infection occurs commonly in patients with advanced HIV infection. Patients with MAC infection usually present with disseminated disease, possibly acquired from organisms in the gastrointestinal or pulmonary tracts [1]. There are few case reports of cutaneous manifestations of MAC infection, and most of these have occurred in patients with disseminated disease [2], although infection at an iv injection site in a drug user has been documented [3]. We describe the presentation and treatment of cutaneous MAC infection that occurred at an im injection site in a patient with AIDS.

A 29-year-old African-American woman was admitted to the hospital for evaluation of an ulcerative lesion on her left thigh. Two months before this admission, she was hospitalized for biopsy of a brain mass. Examination of biopsy specimens revealed cerebral toxoplasmosis secondary to AIDS. During that hospitalization, she received an im injection of codeine in the left anterior thigh. She was discharged from the hospital with prescriptions for sulfadiazine, pyrimethamine, zidovudine, lamivudine, and saquinavir. She did not receive prophylaxis for MAC infection since her CD4 cell counts were still pending. Three weeks after discharge, a firm, painful, erythematous papule developed at the previous injection site. This papule enlarged and eventually drained a purulent material. The patient did not have a fever or any other constitutional symptoms.

On admission, the patient’s temperature was 98.7°F; a physical examination was significant for a 2-cm well-circumscribed ulcer on her left thigh that was draining a serous fluid. The ulcer was tender, and there was a 1-cm area of surrounding induration (figure 1a). She did not have inguinal lymphadenopathy or hepatosplenomegaly. Her WBC count was 3,800/mm$^3$ with a normal differential. The results of blood biochemistry tests did not reveal any electrolyte, renal, or hepatic abnormalities. Radiographs of her left femur did not reveal osteomyelitis. An ultrasonogram of the ulcer revealed a depth of 9 mm with no evidence of myositis. Therapy with cefonicid and probenecid was started.

A gram stain of the ulcer drainage showed no organisms, and no fungal elements were found on a potassium hydroxide preparation. An acid-fast stain of the drainage revealed acid-fast bacilli. Examination of a punch biopsy specimen showed acute necrosis, minimal inflammation, and rare acid-fast organisms. Blood was not drawn for culture. Therapy with clarithromycin, ciprofloxacin, and ethambutol was started. Her CD4 cell count was determined to be 38/mm$^3$. Six weeks later, the lesion was healing and there was no drainage. Cultures of the drainage yielded MAC. Five months after treatment with the clarithromycin-based regimen, the lesion had completely healed (figure 1b).

Cutaneous MAC infection in patients with AIDS is usually a complication of disseminated disease. The lesions can vary from papules, pustules, nodules, and soft-tissue abscesses to ulceration [2]. Nonulcerative lesions can progress to ulceration with drainage, as occurred in our case. Localized infection without apparent disseminated disease has also been reported. Cutaneous ulcerations have occurred at the sites of underlying MAC-associated lymphadenitis [4]. Subcutaneous abscesses and ulcers due to localized MAC infection have also been described [5]. It has been suggested that unrecognized local trauma to the skin could account for these cases [2].

Transcutaneous inoculation of atypical mycobacteria leading to localized infection has been recognized since the 1930s [6]. These cases occurred in nonimmunocompromised hosts who received injections of a variety of pharmaceutical preparations contaminated with mycobacteria. In contrast with our case, all the previous cases were not due to MAC. Since we did not perform blood cultures for MAC in our patient’s case, it is possible that the inoculation may have initiated a local exacerbation of a clinically unrecognized condition.

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**Figure 1.** Ulcer on the left thigh of an AIDS patient with cutaneous *Mycobacterium avium* complex infection at an im injection site. A: Before treatment. B: Five months after treatment.
Severe Recalcitrant Erythematous Desquamating Disorder Associated with Fatal Recurrent Toxic Shock Syndrome in a Patient without AIDS

Since the first description of toxic shock syndrome (TSS) [1], a variety of clinical manifestations of TSS have been reported [2–4]. A TSS-like syndrome was described in patients with AIDS; this syndrome, which was characterized by recurrent and recalcitrant erythema, extensive cutaneous desquamation, hypotension, and fever, was designated recalcitrant erythematous desquamating (RED) disorder [4]. To our knowledge, RED disorder has not been described in patients without AIDS. We describe herein a patient without AIDS who presented with TSS and who had five recurrent episodes of TSS and RED disorder.

A 63-year-old male with a history of alcoholic liver disease was admitted to the hospital on 20 October 1994 with fever, an erythematous macular rash, desquamation, hypotension, myalgias, confusion, and diarrhea (table 1). His symptoms met the TSS case definition [3], and his blood cultures remained negative throughout this admission. A skin biopsy was supportive of the diagnosis of TSS. Antibodies to TSS toxin 1 (TSST-1) and enterotoxin B (MRL Reference Laboratory, Cypress, CA) were present. Therapy with antibiotics was started (table 1). Although cultures of blood samples obtained during the second admission yielded methicillin-resistant Staphylococcus aureus (MRSA), vancomycin therapy was stopped because of the possibility of vancomycin-induced “red man syndrome.” Therapy with clindamycin was started, and the patient’s erythema was healing at the time of discharge.

The patient was readmitted to the hospital three more times (table 1), and each time he had diffuse erythema with desquamation of the hands and feet. Several blood cultures yielded MRSA during all three admissions. During the third and fifth admissions, the MRSA strains cultured from blood were tested for the presence of TSST-1 and enterotoxin B. TSST-1 and enterotoxin B were not detectable (MRL Reference Laboratory). During the last two hospital admissions, he complained of shoulder pain in addition to his other symptoms. An MRI scan of the shoulders showed bilateral osteomyelitis and fluid collections in joint capsules and bursae. Culture of a bursal aspirate yielded MRSA. Despite bilateral drainage of the fluid collections in the shoulders and the administration of antibiotics (table 1), the patient was persistently infected with MRSA and developed gram-negative and fungal sepsis; he died on 4 July 1995.

According to the case definition of TSS, the patient described herein had three episodes of TSS and two episodes of RED disorder [4], which was followed by a fatal episode of TSS (table 1) [3]. Secondary infection of both shoulders may have caused the recurrent TSS and RED disorder. RED disorder has been described in AIDS patients, but our patient did not meet the clinical case definition for AIDS (he was not tested for antibodies to HIV).