

First Case of *Mycobacterium ulcerans* Disease (Buruli Ulcer) Following a Human Bite

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***Mycobacterium ulcerans* disease (MUD) is rapidly reemerging in many countries, especially in West African countries. Antecedent trauma has often been related to the lesions that characterize this frequently crippling disease. We report here the first case of MUD that followed a human bite at the site where the lesion later occurred.**

Human bites are common among children and occur most frequently on an upper extremity. More than 40 species of bacteria have been isolated from wounds caused by human bites. Other specific infections and diseases associated with human bites include herpes simplex, cytomegalovirus, hepatitis B and C, HIV, syphilis, tuberculosis, actinomycosis, and tetanus [1]. This is the first report of *Mycobacterium ulcerans* disease (MUD; also known as “Buruli ulcer”) related to a human bite.

Case report. A 13-year-old girl from Bonou (Ouémé Department, Benin) presented with an extensive induration on the entire right forearm and the dorsum of the right hand. There was only slight ulceration, and the lesion was typical of the plaque form of early disseminated MUD. The patient was otherwise healthy, with normal results of routine laboratory tests. Approximately 30 days previously, a playmate had bitten the patient on the same forearm. The lesion associated with MUD was excised from the elbow to the hand within 24 h after admission, and a skin graft was performed 2 weeks later. The

patient was discharged in good condition 35 days after admission.

Laboratory analyses of samples of the skin surface and the subcutis from the excised lesion were performed. Direct smears were strongly positive for acid-fast bacilli (AFB). *M. ulcerans* was detected by PCR and was grown on culture. Histopathologic analysis revealed contiguous coagulation necrosis of the subcutis and fascia and massive numbers of extracellular AFB in the deep necrotic areas of the subcutis (figure 1). Thus, the results of all known tests for MUD were confirmatory [2–4].

Discussion. MUD is the third most common mycobacterial infection in humans, after tuberculosis and leprosy. The disease is highly endemic in geographic foci in West Africa, and, to a lesser extent, in other parts of the world [2]. MUD is very common in the patient’s area of residence and is a frequent cause of physical deformity and disability [5]. Because of the frequent serious sequelae, such as crippling cicatricial contracture and osteomyelitis requiring amputation, MUD has profound socioeconomic implications in many West African countries [6].

The etiologic agent of MUD, *M. ulcerans*, is an environmental mycobacterium associated with bodies of stagnant water, such as swamps and lakes. Person-to-person transmission of MUD is extremely rare. Although the modes of transmission are incompletely understood, there are multiple reports that patients have had antecedent trauma at the site where the lesion later occurred. Traumas may be as slight as a hypodermic injection or as serious as a gunshot or a landmine wound [7]. In one report, severe MUD with osteomyelitis followed a snakebite [8]. Circumstantial evidence suggests that trauma introduces the etiologic agent into the skin and the subcutaneous tissue from the contaminated surface of the skin. The skin surface may be contaminated with *M. ulcerans* by direct contact with water or other material (e.g., plants or mud) from swamps or ponds. Insects (e.g., waterbugs) that normally dwell in the muddy bottoms of these ecological locations often leave the water and fly to nearby sites. The waterbugs thus are capable of contaminating human skin with *M. ulcerans* either by contact or by biting [9]. Although it is possible that, in the case reported here, the playmate’s mouth was contaminated with *M. ulcerans*, this is considered highly unlikely. We cannot rule out, however, that the trauma due to the bite activated a latent focus of *M. ulcerans* organisms at the bite site. We have occasionally noted this phenomenon in individuals who originally resided in an area where MUD was endemic and developed MUD at a body site where trauma occurred several years after leaving the area

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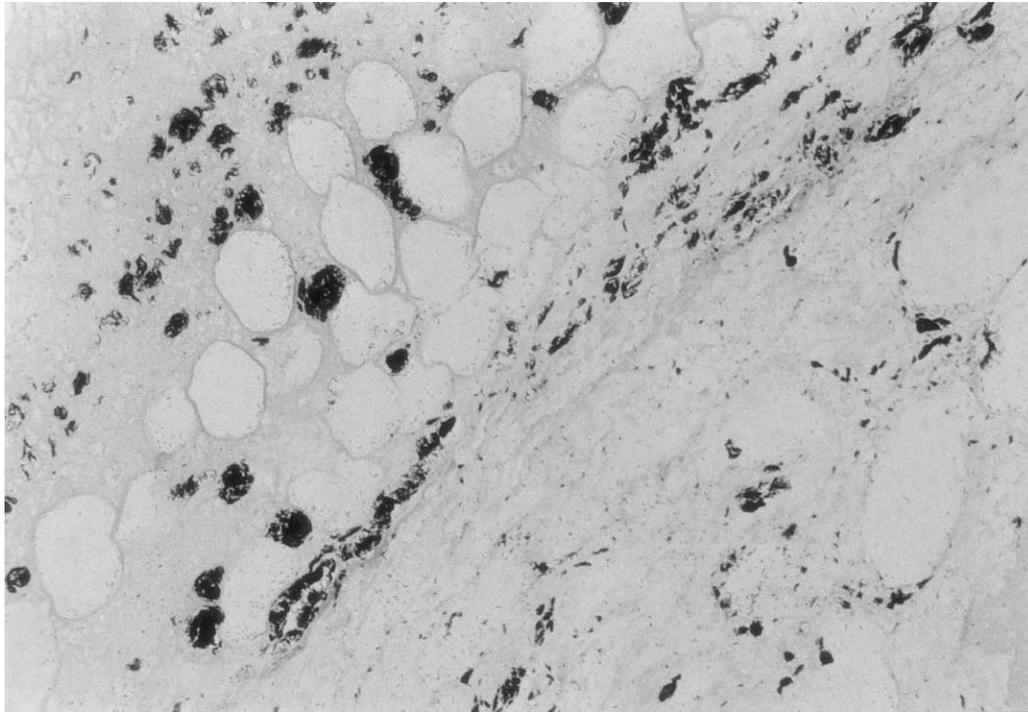


Figure 1. Panniculus and fascia from deep in the lesion. Massive coagulation necrosis and “ghosts” of dead fat cells in the panniculus are visible (left), as well as necrotic fascia (right). Note the numerous clusters of *Mycobacterium ulcerans* with an apparent tendency to spread along the fascial border. Ziehl-Neelsen stain (original magnification, $\times 225$).

of endemicity. In our opinion, the playmate’s bite introduced the etiologic agent into the patient’s skin and subcutaneous tissue from an area of the skin surface that was superficially contaminated with *M. ulcerans*.

MUD is occasionally seen in individuals who have only briefly visited countries where it is endemic (e.g., tourists) [10]. A history of local trauma at the site of chronic cutaneous lesions should alert the physician to the possibility of MUD in patients who have been in areas of endemicity.

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