Tropical infectious ulcers

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INTRODUCTION
The list of ulcers originating in the tropics is extensive. In this comprehensive overview the most common causes of ulcers in the tropics are discussed. Viral causes of ulceration are not discussed in this overview.

BACTERIAL INFECTIONS

Tropical ulcers
Tropical ulcers are often overdiagnosed. They were common in Africa and known as Veldt sores. A true tropical ulcer is a chronic phagogenic ulcer, possibly caused by a necrotic reaction induced by anaerobic bacteria. Anaerobic fusobacteria (F. ulcerans) together with Treponema vincenti are thought to be the dominant responsible organisms. Noma or cancrum oris may have a similar pathogenesis. Treatment: local wound care, and, if needed, antibiotics.

Ulcerating pyodermas
Pyoderma covers several clinically distinct entities of skin lesions that are caused by Staphylococcus aureus and/or β-hemolytic streptococci group A. Pyoderma is a common cause of (purulent) ulcerative skin lesions in the tropics.

Pyoderma is most commonly seen as a secondary infection in skin lesions caused by environmental insults to the skin, such as insect bites, abrasions and atopic dermatitis.1

Treatment: it can be treated by, for instance, gentian violet; in severe cases antibiotics are needed.

Mycobacterial infections
After tuberculosis and leprosy, M. ulcerans is the most common mycobacterial disease and the most important cause of cutaneous ulceration.

Mycobacterium ulcerans
M. ulcerans may cause a chronic ulcer known as Buruli ulcer. It is characterized by a painless nodule, papule, plaque or oedema, evolving into a painless ulcer with undermined edges.2 Trauma by vegetation appears to be essential for the introduction of M. ulcerans in the skin. There may be a role for insects in the transmission of the infection. HIV infection seems to be no risk factor for Buruli ulcer, and also HIV infection does not appear to influence the treatment outcome of Buruli ulcer.

The largest number of patients is found in Africa. After 1980 foci emerged in West Africa particularly in distinct regions of Benin, Ivory Coast and Ghana. It is also present elsewhere, in particular in Australia (Bairnsdale ulcer).

The majority of cases are children below the age of 15, and lesions are mostly localized on extremities (legs more than arms). (Fig 1) The major endemic foci are localized in wetlands, especially those with slow-flowing or stagnant water in tropical and subtropical countries. It appears that natural or man-made changes in water management influence the outbreak of Buruli ulcer.

Treatment: surgery (when necessary), combined with chemotherapy (a combination of Rifampicin and Streptomycin for 8 weeks as a first line).
**Mycobacterium ulcerans**

Exogenous inoculation of *M. tuberculosis* may cause ulceration, also called tuberculous chancre. The lesion starts 2 to 4 weeks after inoculation with a smooth papule or nodule, which enlarges in the course of several weeks to a plaque which then ulcerates, showing undermined edges. Lesions are painless. After 3 to 8 weeks non-tender regional lymphadenopathy develops which may suppurate to form a “cold” abscess, which then may spontaneously drain with sinus tract formation. This process in general heals spontaneously with atrophic scarring in 3 to 12 months.

Scrofuloderma is a common type of cutaneous tuberculosis in which ulceration may occur due to contiguous spread from an active infection at a deeper site such as a lymph node. The lesions are mostly localized over the lymph glands in the neck.

Ulceration may also occur in lupus vulgaris, which is caused by reactivation of the disease in patients with a high degree of cell-mediated immunity after earlier haematogenous dissemination. The lesions start with brown-red papules, which in the classical form extend to plaques with peripheral activity with an irregular border and central healing with atrophic scar formation and depigmentation. (Fig 2). The clinical picture can be variable. The most common localization is the face, with the nose, cheeks, mouth and earlobes as preferential sites. In Asia and Africa lesions on legs and buttocks are common. Tuberculous gumma or metastatic tuberculous ulcer is caused by haematogenous dissemination from a primary focus, during periods of lowered resistance with bacillaemia. The lesion starts with a subcutaneous nodule or a fluctuant swelling. The overlying skin breaks down, resulting in an undermined ulcer with sinus formation.

Treatment: according to WHO guidelines for TB.
Cutaneous diphtheria

Cutaneous diphtheria is an infectious bacterial disease caused by *Corynebacterium diphtheriae*. It is transmitted from person to person through close physical and respiratory contact. A powerful toxin is responsible for the tissue damage. It primarily affects the upper respiratory tract, which may lead to breathing difficulties or even death. The organism may be harboured in the nasopharynx, skin, and other sites of asymptomatic carriers. *C. diphtheriae* is often found in pre-existing ulcers like ecthyma or as superinfection of eczema. In immunized individuals systemic toxic complications such as myocarditis and neuritis are rare. The clinical aspect depends on presentation as a primary infection or as a secondary infection. The characteristic lesion of a primary lesion ranges from a pustule to a chronic, non healing ulcer with a punched out appearance, a slightly undermined margin and a grey adherent pseudo-membrane covering the ulcer. During the first 2 weeks it is painful, later the lesion becomes painless. In many cases lesions are less distinctive. Secondary infection in any pre-existing wound and superinfection of eczematized skin lesions is common and often overlooked. Cutaneous diphtheria may persist for 6 to 12 weeks.4

Treatment: antibiotics (erythromycin and penicillin) and on indication vaccination and antitoxin.

Cutaneous leishmaniasis

Cutaneous leishmaniasis (CL) is caused by parasites belonging to the genus *Leishmania*. The parasites are transmitted to humans through the bite of the female phlebotomine sand fly that has fed on an infected mammal.

The disease occurs throughout the tropical and subtropical regions. Old world CL is found in the Mediterranean, northern Africa, the sub-Saharan, the Middle East, and south west Asia. New World CL extends from southern Texas to the highlands of northern Argentina. In the past decades, there has been a definite increase in the incidence of CL. This is due to several factors such as rural to urban migration, development of new agro-industrial projects, movement of army troops into endemic regions, and the termination of insecticide spraying. HIV infection does not seem to increase the risk of CL infection, but may influence treatment response. A lesion may start with a papule or nodule, which develops into an ulcer with or without a scab. (Fig 3) The lesions are usually painless.5
Treatment: depending on the species involved, systemic/intralesional pentavalent antimony, intramuscular pentamidine, oral ketoconazol or itraconazole, or miltefosine.

Subcutaneous mycoses
Subcutaneous mycoses are characterized by a heterogeneous group of infections that often result from direct penetration of the fungus into the dermis and subcutaneous tissue through traumatic injury. The most common subcutaneous mycoses which may show ulceration are sporotrichosis, chromo(blasto)mycosis and mycetoma.⁶

Sporotrichosis
The causative organism of sporotrichosis is *Sporothrix schenckii*, a dimorphic fungus occurring in nature. Infection results via traumatic inoculation through the skin of material which contains the fungus, in particular wood splinters or thorns. Most cases at present are reported in South and Central America.

The incubation period is probably a few weeks. It is mostly encountered as a lymphocutaneous or sporotrichoid form or less common as a localized or fixed cutaneous form. The primary cutaneous lesions may appear as papular, nodular, or pustular lesion that develops either in a superficial ulcer or verrucous plaque. The lymphocutaneous form shows, during progression of the disease, multiple subcutaneous nodules that are formed along the course of local draining lymphatics. (Fig 4)

Treatment: kalium iodide or an azole, terbinafine or amphotericine.
**Chromo(blasto)mycosis**
Chromoblastomycosis is a chronic (sub)cutaneous mycotic disease that occurs more frequently in tropical and subtropical areas and is caused by several darkly pigmented fungi. The disease is most prevalent in tropical and sub-tropical America and Africa. Lesions occur typically on the foot or leg. After inoculation of the fungus through the skin slowly growing scaly wartlike nodules develop, ulceration may occur.

Treatment: cryotherapy, heat, azoles, amphotericine or terbinafine.

**Mycetoma**
Mycetoma is discussed on page 11 in MT, Bulletin of the Netherlands Society for Tropical Medicine and International Health.

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**References**


