Mycobacteria
Infections with Nontuberculosis Mycobacteria

Among the nontuberculosis, nonleprosy mycobacterial infections of the skin, those caused by *M. marinum* are the most common among nonimmunosuppressed people. Unlike *M. tuberculosis*, which is transmitted from person to person, nontuberculosis mycobacteria are abundant in nature, in soil and water, and contact is frequent in most zones of the world.
These skin infections may be acquired by direct inoculation into the skin or by hematogenous spread from a visceral source.
The histopathologic picture in nontuberculosis mycobacterioses is just as variable as the clinical picture.

*Infection with Mycobacterium Kansasii*
M. *kansasii* is usually a lymph node and pulmonary infection, and skin lesions are unusual. Implantation causes a chronic cutaneous infection, with hematogenous dissemination to skin. The skin lesions are acute abscesses with large numbers of acid-fast bacilli.

*Infection with Mycobacterium Avium-intracellulare*
M. *avium* complex infection is a common cause of cervical lymph node mycobacteriosis in normal children and of pulmonary disease in patients with immunodeficiency. Severely immunocompromised patients, such as those with HIV infection, have a very high prevalence of disseminated *M. avium-intracellulare* bacteremia, and many show one or more cutaneous papules and nodules. Steroid therapy also predisposes to skin lesions. Necrotizing granulomas with necrosis, and a spindle cell transformation of macrophages, forming a histoid-like lesion (as in leprosy), can occur.
Infection with *Mycobacterium Marinum*
Infections with *M. marinum* can be contracted through minor abrasions incurred while bathing in swimming pools or in ocean or lake water or while cleaning home aquariums. Infected swimming pools have caused epidemics, the largest of which affected 290 persons. The period of incubation usually is about 3 weeks but may be longer.

Clinically, most of the lesions caused by *M. marinum* are solitary and consist of indurated, dusky red, hyperkeratotic papules, nodules, or plaques. Superficial lesions persist in some patients for many years. A few HIV-associated cases have been reported. Fatal disseminated *M. marinum* infection is rare.
Histopathology.
Early lesions no more than 2 or 3 months old show a nonspecific inflammatory infiltrate composed of neutrophils, ... of the granulomas. The epidermis often shows marked hyperkeratosis with an acute inflammatory infiltrate and ulceration. Acid-fast bacilli usually can be identified in histologic sections of early lesions that show a nonspecific infiltrate.
Differential Diagnosis

The granulomatous reaction produced by M. marinum is similar to that observed in tuberculosis verrucosa cutis or lupus vulgaris. The pattern of pseudoepitheliomatous hyperplasia and acanthosis need to be examined alongside Ziehl-Neelsen stains. For definitive identification, culture may be necessary.
Buruli Ulcer (*Mycobacterium ulcerans*)

Buruli ulceration, an infection caused by a nontuberculous mycobacterium, *M. ulcerans*, is endemic in West and Central Africa.
Central Africa, Central America, and South Australia. The organism is now identified in nature, near inland waters.

*Histopathology*
The infection begins as a subcutaneous nodule exhibiting "ghost" ischemic-type
Pathogenesis

The widespread necrosis of subcutaneous tissue is caused by a toxin secreted by M. ulcerans, a pathogenesis unique to this species of mycobacteria. It is polyketide mycolactone, and strain and geographical factors affect its production. This toxin causes necrosis when inoculated into guinea pig skin and into macrophages in tissue culture. Like M. marinum, M. ulcerans shows optimal cultural growth at 30° to 33°C.
PCR technology has been used to track the distribution of M. ulcerans in Buruli ulcer lesions and to compare the mycobacterial burden with histopathologic changes. While peaks of cell-mediated immunity, development of satellite lesions by contiguous spreading was not completely prevented.
Healing of Buruli ulcer coincides with development of delayed hypersensitivity to the mycobacterium, possibly contingent with the presence of granulomas. When granulomas were present, significantly higher expression of IFN-gamma was seen as well as lower bacillary counts.