A 33-year-old man with a medical history of tenosynovitis of the third finger of his left hand, which had been treated successfully with a local infiltration of glucocorticoids approximately one year previously, was admitted for evaluation of a cutaneous nodule on the same hand. The nodule first appeared 2 months prior to presentation; it had developed rapidly after the patient had injured his palm while travelling through Morocco and cleaned the wound in a river. Physical examination revealed a well-defined, reddish, painful nodule, 2×2 cm, on the palm of his left hand (Fig. 1). No other cutaneous lesions, regional lymphadenopathies or accompanying systemic symptoms were observed.

*What is your diagnosis?* See next page for answer.
A skin biopsy revealed hyperplastic epidermis, a dense inflammatory infiltrate extending to the subcutis, development of granulation tissue and histiocytes in a granulomatous disposition, sometimes with a central focus of necrosis was found (Fig. 2). Ziehl–Neelsen staining did not reveal any bacilli. Acid-fast bacilli, identified as *M. kansasii*, were isolated in the specimen cultured on Lowenstein–Jensen agar. Complete blood tests, HIV testing and chest X-rays were normal. Subsequently, the whole lesion was excised, and a triple anti-tuberculous drug therapy was established for a total course of one year, with no further recurrence of disease.

*M. kansasii* is a slow-growing, atypical mycobacteria that was first described in 1953 by Buhler & Pollak (1). It is included in the photochromogenic group of mycobacteria (Runyon group 1), which produce pigment when grown in light at 37ºC (2). *M. kansasii* is also known as the “yellow bacteria” because of its producing beta-carotenoid pigment when exposed to light (1). It usually inhabits water supplies, swimming pools and sewage (3), and is mostly known for causing granulomatous pulmonary infections in patients with underlying lung diseases, such as chronic obstructive pulmonary disease and pneumoconiosis (4). It can also be the cause of localized infections, such as myositis, arthritis and lymphadenopathy. Disseminated disease caused by these bacteria is less common and is primarily seen in immunocompromised patients.

Cutaneous *M. kansasii* infection has only rarely been described in the literature (4, 5). Skin and soft tissue infections usually result from penetrating injuries or disseminated disease, mostly affecting patients with an underlying immunological disorder (1). When appearing in immunocompetent patients, the mode of infection is exogenous inoculation following cutaneous trauma (5, 6). A review of cutaneous *M. kansasii* infection by Breathnach et al. (6) found that in 93% of patients only a limb was affected, supporting the role of skin trauma in acquiring the organism. We suspect that the injury our patient suffered in Morocco might have been the route of entry for *M. kansasii* into the skin.

The clinical appearance of cutaneous *M. kansasii* infection is heterogeneous and has been reported as pustules, nodules, ulcers, sporotrichoid pattern, verrucous lesions, panniculitis, rhinophyma-like lesions and erythematous plaques (1, 5). Differential diagnosis should include other bacterial infections, deep mycosis, cutaneous tumours and lupus profundus, among others (7, 8).

The morphological expression of mycobacterial infection is wide. In immunocompetent patients granulomas and epidermal hyperkeratosis are usually observed, while in immunocompromised patients show more components of an acute inflammatory response, with necrosis, and infiltrating neutrophils and monocytes (7).

A positive culture remains the gold standard for diagnosis of a mycobacterial infection. Since the concentration of organisms may be low, Ziehl–Neelsen staining is often falsely negative, and multiple cultures are recommended (7). Nevertheless, mycobacterial DNA identification using PCR is considered the most sensitive and specific test available to date (8).

Cutaneous *M. kansasii* infection can be treated with the same regimen recommended for pulmonary disease in adults; at least one year of multidrug treatment with isoniazid, rifampicin and ethambutol, excluding pyrazinamide, to which the bacteria is resistant (9).

REFERENCES