A 35-year-old physician presented with an asymptomatic lesion on his middle finger. The lesion had appeared during a holiday in Australia and had persisted for several weeks. The patient attributed the lesion to an insect bite. On his right middle finger, there was a solitary, well-defined, 15 × 15 mm, infiltrated brownish-red plaque with discrete scaling (Fig. 1a). The remaining integument was completely inconspicuous and the lymph nodes were not swollen on palpation. The patient did not mention any underlying diseases. Based on the patient’s statements and the clinical findings, a granulomatous foreign body reaction, cutaneous leishmaniasis, ringworm (tinea), sarcoidosis, atypical mycobacteriosis and eczema were considered as differential diagnoses. A skin biopsy was performed. Histology revealed a psoriasiform epithelial hyperplasia with a pronounced lichenoid inflammatory response with interface dermatitis and a deep granulomatous component with many plasma cells (Fig. 1b).

What is your diagnosis? See next page for answer.

Fig. 1. (a) Initial presentation. A solitary brownish-red plaque with discrete scaling on the middle finger. (b) Epithelial hyperplasia with interface dermatitis and many plasma cells (HE; original magnification ×100).

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**Diagnosis: Primary syphilitic lesion on finger**

Syphilis was suspected because of the plasma cell infiltration. Immunohistochemical analysis with an anti-*Treponema pallidum* antibody revealed the presence of numerous spirochetes in the epidermis and at the dermoepidermal junction (Fig. 2).

Extragenital lesions of primary syphilis are rare (approximately 5%) (1–4). However, it is a well-recognized symptom of syphilis (5, 6). In previous reports, cases of extragenital syphilis have been described in physicians who had become infected during clinical examination of infected patients. The unusual occurrence of extragenital syphilis and the variable clinical picture frequently result in a false or delayed diagnosis (5, 7).

The patient was confronted with the diagnosis and stated that he had numerous high-risk homosexual contacts in Australia, some with digito-anal sexual intercourse. He denied any previous syphilis infection. We extended our diagnostic spectrum and additionally performed syphilis, HIV and hepatitis serologies. The results were as follows: *T. pallidum* particle aggl. (TPPA) 1:327680, cardiolipin reaction Venereal Disease Research Laboratory test (VDRL) 1:2, FTA-ABS IgG/IgM (IFT) positive, anti-*Treponema pallidum* IgG (IB) positive, anti-*T. pallidum* IgM (IB) negative, and hepatitis- and HIV serologies negative. We treated the patient 3 times with 2.4 million IU benzathine penicillin i.m., in weekly intervals. The lesion healed within 3 weeks after initiation of therapy. Syphilis and HIV serologies were checked after 3, 6 and 12 months, during which the specific *T. pallidum* immunoglobulin G (IgG) antibodies decreased by more than 2 titre steps. The cardiolipin reaction VDRL remained 1:2. The HIV and hepatitis serologies remained negative.

In this case, a number of specific features hindered the diagnosis. The patient did not exhibit the classical chancre, but instead a non-ulcerated plaque on his finger, and he did not provide his full sexual history. As a consequence, the diagnosis was ultimately made by histological examination. The serological findings were also unusual: anti-*T. pallidum* IgM was negative at the time of diagnosis and the VDRL titre was only weakly reactive, but on the other hand the TPPA test was highly positive. For primary syphilis, one would expect a positive anti-*T. pallidum* IgM and a definitely reactive VDRL screening test. Our patient might already have had a syphilis infection in the past, therefore re-infection would not result in a persistent increase in IgM-titre. Thus, we assume that the IgM window was missed and that healing had already begun. However, this again raises doubts about the patient’s anamnestic statements. Although histopathology is not generally needed for the diagnosis of syphilis, it can be helpful in selected cases with atypical presentations or laboratory findings, as presented in our case.

**REFERENCES**


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**Fig. 2. Immunohistochemical analysis. Presence of *Treponema pallidum* in the epidermis and at the dermoepidermal junction (Immunohistochemistry with anti-*Treponema pallidum* antibody; original magnification ×200).**