genitals have been described as likely due to simultaneous transmission or autoinoculation (7).

In general, the morphology of extragenital chancres parallels those on the genitals, although variations occur (3). Cutaneous primary syphilitic lesions can be atypical, often widespread, deeply infiltrated, ulcerated or with impetiginoid or ectimatoid features (1, 7-9). The clinical pattern of dactilitis, periungual paronychia and panaritium has been reported on the fingers (7, 10).

The unusual case of extragenital chancre and the variable clinical appearance of chancre on the fingers frequently result in incorrect or delayed diagnosis, because syphilis is rarely suspected in such cases.

Differential diagnosis includes, at first, both clinically and by laboratory procedures, primary complexes of primary cutaneous tuberculosis, cat-scratch disease, tularemia and sporotrichosis. However, primary syphilitic lesions of the fingers must also be distinguished from cutaneous leishmaniasis and atypical mycobacterial skin infections, which sometimes develop a primary complex, as well as staphylococcal lymphangitis and foreign body granulomas (2, 3, 5). Furthermore, viral diseases, lymphomas and cancers with nodal metastasis must also be excluded.

The patient's history may evoke suspicion. The presence of typical lymphadenopathy, as in our report, is helpful when considering the possibility of syphilis. In fact, a papuloulcerative lesion with regional lymphadenopathy should raise one's suspicion of primary syphilis. Where epitrochlear or axillary adenitis are painless, serological tests for syphilis are indicated (5).

In conclusion, chancre of the fingers can be diagnosed only when the clinician maintains a high index of suspicion of syphilis, "the great simulator".

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Type 2 Segmental Cutaneous Leiomyomatosis

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Sir,

We read with interest the report on "zosteriform and disseminated lesions in cutaneous leiomyoma" by Agarwalla et al. (1). We would like to suggest categorizing this case as a type 2 segmental manifestation of cutaneous leiomyomatosis (2). Agarwalla et al. noted a few leiomyomas on their patient's back, and from the title of their report we conclude that these lesions were disseminated, reflecting heterozygosity for the gene of this autosomal dominant trait. By contrast, the agminated leiomyomas on the left side of the chest would reflect an area of skin in which the corresponding wild-type allele has been lost at an early developmental stage, which is why a pronounced involvement is superimposed on the ordinary non-segmental phenotype (3-5). Apparently, early loss of heterozygosity occurs rather frequently in cutaneous leiomyomatosis. At least 9 cases suggesting a type 2 segmental manifestation have previously been documented (2, 5, 6).

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Editors comment: Agarwalla et al. have been given the opportunity to respond to this letter.