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Primary Human Immunodeficiency Virus Infection Mimicking Syphilis

Colleagues—Primary exposure to the human immunodeficiency virus (HIV) may lead to an acute infectious disease with skin involvement [1–4]. In the first review, in 1985 [1], a rash of a mononucleosis type predominantly located on the trunk was present in 50% of patients and seemed characteristic although nonspecific. We report a case with unusual dermatologic manifestations that were initially mistaken for syphilis.

In September 1990, a 39-year-old homosexual man was referred to us with the diagnosis of secondary syphilis. He presented with a 7-day history of three genital ulcers. A high fever began 5 days later, associated with arthralgias, odynophagia, and a nonpruritic skin rash. Further inquiry disclosed unprotected orogenital sex with an unknown partner 17 and 32 days before.

Physical examination revealed a patient in good general condition, febrile at 39.5°C, with a widespread symmetric purplish papular eruption in a linear distribution on his face, trunk, and proximal part of the four limbs and palms. Numerous painful oval-shaped ulcers up to 1 cm in diameter were seen on the oropharyngeal mucosa, including the tongue. On the penis, an oblong, nonindurated, painful chancre of 2.5 × 1 cm with a clean base was surrounded by two similar smaller ulcerations. Serous exudate was not obtained when pressure was applied. The scrotal area was also covered by many shallow oval-shaped erosions. Generalized lymphadenopathy was present, including the antecubital and epitrochlear areas. No hepatomegaly was noted, but a slightly enlarged spleen was felt. The rest of the clinical examination was unremarkable.

Findings from laboratory evaluation revealed normal values for sedimentation rate, hemoglobin, white blood count, and biochemical profile. Platelet count was 119,000/mm^3.

Repeated darkfield examinations for *Treponema pallidum*, direct examination and culture for *Haemophilus ducreyi*, and immunofluorescence staining for herpesviruses (simplex and zoster) of the genital ulcer's base were all negative. Serum VDRL test, fluorescent treponemal antibody test, and *Treponema pallidum* hemagglutination assay done on several occasions remained unreactive. Serologic evaluations for cytomegalovirus, *Toxoplasma gondii*, Epstein-Barr virus, rubella, and hepatitis A (IgM) and hepatitis B (antigen) were negative. On the day of referral, search for anti-HIV antibody by screening ELISA and immunoblotting tests was unsuccessful. In contrast, HIV p24 antigen (HIVAG-1; Abbott, North Chicago) was detected in the serum at 39 ng/l. Lymphocyte count was 1113/mm^3, with a CD4 count of 323/mm^3 and CD8 count of 545/mm^3.

Major features of histologic examination were edema and a perivascular and perifollicular lymphocytic infiltrate in the dermis. There was minimal basal and suprabasal spongiosis, with lymphocytic exocytosis and rare necrotic keratinocytes in the epidermis. On electron microscopy, cytoplasmic reticulotubular structures were present in most lymphocytes and histiocytes and in rare keratinocytes and Langerhans cells. There was no evidence of viral particles either assembling or budding.

The patient's illness resolved spontaneously within 2 weeks. Return to normal body temperature occurred on day 3 and reso-

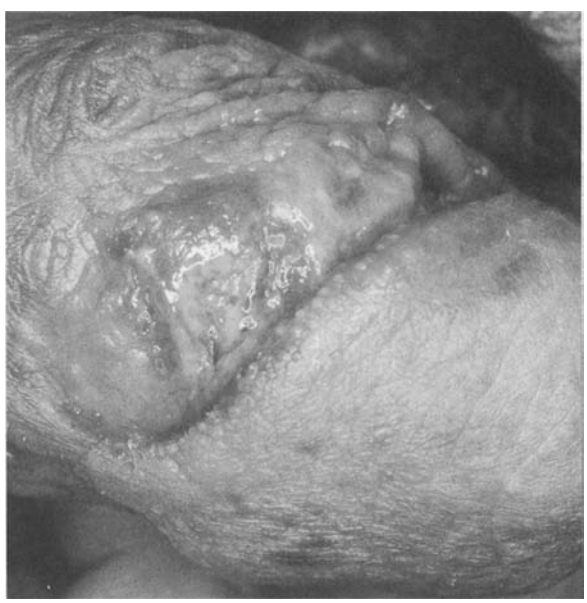

Figure 1. Genital ulcers in a patient with human immunodeficiency virus infection.

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The Journal of Infectious Diseases 1991;164:615–6

0022–1899/91/6403–0036$01.00

lution of the skin rash on day 7 and of the erosive lesions on day 15. HIV antibodies appeared on day 7 and coincided with the disappearance of p24 antigenemia on day 8.

Previous reports have shown that an exanthem, located predominantly on the face and trunk, is probably the most common skin manifestation of primary HIV infection [1–4]. Several reports have mentioned oral, esophageal, or genital erosive lesions [3, 5]. In very rare cases [6–9], concurrent oral and genital ulcers have been documented, but they have often been considered a minor diagnostic feature because of their limited extent.

Owing to the presence of general symptoms associated with mucosal erosive lesions and a skin rash, all suggestive of syphilis, the initial clinical picture was misleading [10]. The incubation period, morphology, and location of the penile chancre (figure 1) were all compatible with primary syphilis, although several features such as the multiplicity of lesions and the absence of induration made this diagnosis unlikely. The papular eruption, which involved the palms as well, could easily have been mistaken for secondary syphilis, particularly as contamination seemed to have occurred via the orogenital route, unusual for HIV. The diagnosis of primary HIV infection in our case was, however, confirmed by successive serologic investigations.

This case reminds us that primary HIV infection must be included in the differential diagnosis of genital ulcers, particularly if associated with a skin rash and oral lesions. The use of p24 antigen is useful in this instance. Because of the clinical similarities with other sexually transmitted diseases such as herpes, syphilis, or *H. ducreyi* infection, these should be ruled out as well.

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Unusually High Level of a Tumor-Associated Antigen in the Serum of Human Immunodeficiency Virus–Seropositive Individuals

Colleagues—A 90,000-Da molecular mass lipoprotein (90K) recognized by monoclonal antibody Sp-2 [1] has recently been identified in the sera of patients with breast or ovarian cancer or other malignant diseases [2–4]. The 90K, which is now being biochemically characterized, has an NH_2-terminal amino acid sequence that does not resemble any protein for which sequence information is available (unpublished data). In preparation for a multicenter study on 90K as a prognostic indicator in a broad range of malignancies, we have obtained serum samples from three patients with human immunodeficiency virus (HIV)–related Kaposi's sarcoma. We found that these sera had the highest 90K-immunoreactive activity ever recorded in our laboratory. This observation prompted us to investigate the role of this antigen in HIV infection.

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The Journal of Infectious Diseases 1991;164:616–7

0022–1899/91/6403–0037$01.00

Sera were obtained from 104 anti-HIV–positive subjects: 20 were symptom-free and without decreases in $CD4^+$ lymphocyte number, 27 were also asymptomatic but had decreases in $CD4^+$ lymphocytes (<500/μl), 11 had lymphadenopathy syndrome, 30 had AIDS-related complex, and 16 had frank AIDS. Control sera were obtained from 62 anti-HIV–negative blood donors

Table 1. Levels of 90-kDa protein (90K) in human immunodeficiency virus (HIV)–seronegative and -seropositive subjects.

Group	No.	90K (units/ml), mean ± SD	No. (%) with 90K >1.7 units/ml
Anti-HIV-negative	62	0.87 ± 0.56	3 (5)
Anti-HIV-positive	104	2.65 ± 1.54	73 (70)
Asymptomatic, $CD4^+ > 500/\mu l$	20	1.70 ± 0.81	8 (40)
Asymptomatic, $CD4^+ < 500/\mu l$	27	2.12 ± 1.01	16 (59)
LAS	11	1.96 ± 1.16	7 (64)
ARC	30	3.65 ± 1.83	28 (93)
AIDS	16	3.31 ± 1.18	14 (87.5)

NOTE. LAS = lymphadenopathy syndrome; ARC = AIDS-related complex. All means for anti-HIV-positive subjects and subgroups were significantly greater than those for anti-HIV-negative subjects ($P < .0001$, analysis of variance).