Follicular Accentuation of Leukocytoclastic Vasculitis in an HIV-Infected Patient

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Human immunodeficiency virus (HIV) not only brought new diseases but changed the face of long-known ones. Leukocytoclastic vasculitis (LV) tends to show a homogeneous cutaneous clinical picture. A predominantly perifollicular location of the palpable purpura can be seen in HIV-infected patients.

We have recently seen a 36-year-old female intravenous drug abuser, who had been found to be seronegative for HIV and hepatitis B virus (HBV) 6 months before. She presented with a striking perifollicular palpable purpura affecting symmetrically the lower extremities (fig. 1), fever, lymphadenopathy and polyarthritis. Biopsy showed LV. Perifollicular distribution was also evident on histology (fig. 2). Days after, she complained of anorexia and developed jaundice, while the cutaneous lesions persisted with perifollicular distribution and scarce wheals appeared. Serologic tests were indicative of HIV infection and HBV acute hepatitis. The C3 level was 40 mg/dl (normal: 70-160 mg/dl), C4 18 mg/dl (normal: 20-40 mg/dl) and CH50 82 U/ml (normal: 150-250 U/ml). Cryo-globulins were negative. The CD4 cell count was 2,463 ×106 cells/l, the CD4/CD8 ratio 0.96. She showed no gingival bleeding, hyperkeratotic papules or hair alterations suggestive of scurvy. Histologic signs of cytomegalovirus infection were not present.

Although LV often starts with a follicular pattern, it rarely persists as a follicular eruption, as it did in our patient. HIV-infected patients are prone to suffer from numerous follicle-associated diseases [1-3] and to
show follicular accentuation of disease [4], including vasculitis [4, 5].

We think that several mechanisms could explain these findings, but the most feasible hypothesis is that HIV-infected patients have a subclinical vascular damage in the follicle that facilitates immune complex deposition: Weimer and Sahn [4] suggested that ‘perhaps the HIV infection lowers total body vitamin C stores enough to produce follicular accentuation of some skin diseases’, but other possible mechanisms, such as subclinical infections, should not be forgotten. Although the pathogenesis remains unclear, the important fact is that perifollicular LV should make the clinician suspect HIV infection.

References
Papular-Purpuric Gloves-and-Socks Syndrome Related to Cytomegalovirus Infection

We read with interest the article by Feldmann et al. entitled ‘Papular-purpuric “gloves and socks” syndrome: Not only parvovirus B19’ [1]. The papular-purpuric gloves-and-socks syndrome (PPGSS) has been considered by some authors as a manifestation of parvovirus B19 infection in adults [2, 3]. However, the lack of evidence of primary infection due to this virus in other cases suggests that other agents might be involved [1]. We report a patient who developed an acute dermatosis consistent with PPGSS associated with serologic tests indicating active cytomegalovirus (CMV) infection.

Case Report

We observed a 38-year-old white man with a 72-hour history of acral pruriginous erythema. He had serum positivity for hepatitis B surface antigen, without biological evidence of hepatitis. On physical examination, his hands and feet were symmetrically edematous and erythematos. Twenty-four hours later, the patient complained of malaise and sore throat. Fever up to 38 °C was present. Hundreds of confluent, erythema-topurpuric, flat papules, 1-3 mm in diameter, were seen at that time on his hands and feet, with sharp margins on the wrists and ankles. Isolated purpuric lesions could also be observed on the forearms and legs. Enanthema, with some painful erosions, 1 -4 mm in diameter, on the lower lip were present, accompanied by laterocervical and axillary lymph node enlargement. Laboratory investigation showed: leukocyte count $6.33 \times 10^7$ with a differential count of 63% segmented neutrophils, 6% unsegmented neutrophils, 12% lymphocytes, 15% monocytes and 3% eosinophils. The platelet count was $128 \times 10^7$. IgM anti-CMV antibodies were detected by ELISA and total antibodies by the latex technique. Other laboratory data were within normal limits or negative, including red blood cell count, coagulation tests, urinalysis, chest X-ray film, urine and blood cultures, rheumatoid factor and serologic tests for lues, Salmonella, Brucella, Rickettsia, toxoplasmosis, Epstein-Barr virus, ECHO virus and parvovirus B19. Histopathologic study showed a dense inflammatory mononuclear perivascular lymphocytic infiltrate on the papillary dermis without evidence of vasculitis.

During the next 3 days, a symptomatic and clinical improvement was accompanied by a transitory maculopapular generalized exanthema. The purpuric lesions cleared gradually without scarring during the next 10 days.

Four months later, total antibodies to CMV examined by the latex technique were still positive, whereas CMV-specific IgM were undetectable.

Discussion

PPGSS was first reported as a distinctive entity by Harms et al. [4] in 1990. It consists of acral pruritic erythema and edema rapidly followed by petechial purpura in a charac-