Leukocytoclastic Vasculitis in a Patient with HHV-6 Infection

Sir,

Cutaneous involvement in adults infected with human herpes virus 6 (HHV-6) is rare. Exanethematous reactions in immunosuppressed transplant patients and 1 case of reactive haemophagocytic syndrome have been reported (1, 2). Recently we described a case of erythema elevatum diutinum, a rare form of chronic cutaneous vasculitis, envisaging the aetiological role of HHV-6 (3). We describe here the case of an immunocompetent adult with leukocytoclastic vasculitis and propose that it is caused by HHV-6.

A 54-year-old woman presented with 1-month history of non-itchy papulo-purpuric lesions on her legs that had undergone several episodes of remission and exacerbation. The patient complained of fatigue, diffuse myalgia and arthralgia on her knees, ankles, pelvis and scapulae. Her medical history was unremarkable and she denied taking any drug before the eruption.

On examination she exhibited papular-purpuric lesions with sparse petechiae on the lower legs (Fig. 1). There were neither hepatosplenomegaly nor palpable lymph nodes. A biopsy specimen showed endothelial swelling with deposits of fibrin within and around the blood vessels and a cellular infiltrate consisting mainly of neutrophils with nuclear dust compatible with a diagnosis of leukocytoclastic vasculitis. Direct immunofluorescence revealed deposits of IgM and C3 in the blood vessels of the papillary dermis. Laboratory investigations showed normal values, except for the blood level of alanine aminotransferase 87 U/l (normal values 0 – 40 U/l) and aspartate aminotransferase 56 U/l (normal values 0 – 37 U/l). Chest X-rays and abdominal ecography were normal.

The disease slowly improved and the lesions gradually faded over a period of 2 months, leaving pigmented macules. Aminotransferase levels became normal within 2 months. Deflazacort was tapered and stopped after 45 days. One month later, the anti-HHV-6 IgG titre was 1/320 and anti-HHV-6 IgM 1/40. At present, the patient is in good health and free from lesions.

In early childhood, HHV-6 causes exanethem subitum (4). In adults, instead, the clinical spectrum of diseases associated with HHV-6 is still largely undefined (5). Like other herpes viruses, HHV-6 can produce latent infection and occasionally reactivation in immunocompromised hosts, causing bone marrow suppression, interstitial pneumonitis and encephalitis (1). In immunocompetent adults, its aetiological role has been suggested in several conditions, including mononucleosis-like syndrome, hepatitis and Kikuchi’s lymphadenitis (5). Anti-HHV-6 IgM can be detected only in primary infections and reactivation states (6). On the contrary, anti-HHV-6 IgG are present in 80 – 90% of adults, but tend to disappear over time. In adults, therefore, their increase in titres may be considered indicative of reactivation (7).

Our patient had a rising titre of anti-HHV-6 IgG and a decreasing titre of anti-HHV-6 IgM. In the absence of active Epstein-Barr and cytomegalovirus infections causing serological cross-reactions, her cutaneous vasculitis (and possibly her liver involvement) may be ascribed to either a primary HHV-6 infection or an endogenous reactivation.

The rarity of HHV-6 primary infections in adulthood and its usual severe course both suggest that an endogenous reactivation is the most likely possibility. In conclusion, we recommend examining for HHV-6 infection in any patient presenting with otherwise unexplainable leukocytoclastic vasculitis.

REFERENCES

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