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LETTER TO THE EDITOR

ABSENCE OF HHV-8 INFECTION IN 37 PATIENTS WITH PERIPHERAL LYMPHEDEMA

Hengge et al recently suggested that human herpesvirus 8 (HHV-8) by infecting various cell types in the lymph node, account for lymphadenopathy and secondary lymphedema in patients with Kaposi sarcoma (KS) (1). Out of curiosity, we decided to test the blood sera for HHV-8 titers from patients with peripheral lymphedema but without KS.

Thirty-seven in-hospital patients with peripheral lymphedema were prospectively included over a period of two months in the Dermatology and Lymphology Department at the Trousseau University Hospital. None had congenital or acquired immunosuppression, or evidence of cutaneous KS. The reasons for hospital admission were either planned physiotherapy treatment or acute infection of a lymphedematous limb. Except for one patient, who was Portuguese, each patient was of French origin. Thirty-five of the subjects were women. Mean age was 62 years. Mean number of lymphedematous limbs was 1.6 (median = 2). Lymphedema was present in the upper limbs (13 patients) and lower limbs (46 patients), respectively. One patient was at stage II, 28 at stage III, and 8 at stage IV (2) of lymphedema. Lymphedema was primary in 18 patients and secondary in 19 patients and 12/19 of the secondary lymphedemas were related to treatment for breast cancer. Recurrent limb infection was the major cause of worsening lymphedema in 17/37 patients. Seroprevalence for HHV-8 was assessed using a latent indirect immunofluorescence assay (IFA) as previously described (3). Using this technique, we had previously detected antibodies to HHV-8 in 100% of patients with classical KS and in 80% of patients with AIDS-associated KS (3,4). None of the 37 patients with peripheral lymphedema in this study was HHV-8 positive (0/37, IC_{95} = [0.08]).

The patients in this study had long-standing lymphedema of various causes and severity as is often the case for subjects treated in a tertiary hospital. The absence of seroprevalence of HHV-8 in these 37 patients with peripheral lymphedema was not significantly different from that of the general population in France, estimated at 2% (4). These findings suggest that HHV-8 plays no part in the pathophysiology of peripheral lymphedema without coexistent KS.

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Editors' Note:

Whereas this study shows that without concomitant Kaposi sarcoma (KS with or without AIDS), peripheral lymphedema is not related to HHV-8 virus (the putative cause of KS), the corollary issue is also relevant, namely, what is the basis for lymphedema with KS. The implication of the article by Hengge et al (1) is that lymphedema arises from lymph nodal blockage but radiotracer studies (lymphangioscintigraphy) of the upper and lower extremities in KS/AIDS patients (2-4) demonstrates bizarre patterns more consistent with diffuse lymphatic endothelial damage, plugged lymphatics, and diffuse angioparalysis of lymphatic collectors as opposed to simple restriction to lymph transport through infected regional lymph nodes (2-4).

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