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Letter to the Editor

Herpes Zoster Ophthalmicus in a Patient with Systemic Lupus Erythematosus and Antiphospholipid Syndrome

Sistemik Lupus Eritematöz ve Antifosfolipid Sendromlu Bir Hastada Herpes Zoster Oftalmikus

Nilton Salles Rosa NETO,[1] Luciana Feitosa MUNIZ,[1] Luciana Parente COSTA,[1] Manoel Tavares Neves JÚNIOR,[1] Licia Maria Henrique da MOTA,[2] Jozélio Freire de CARVALHO[1]

[1]*Department of Rheumatology, Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, São Paulo, Brazil;*
[2]*Department of Rheumatology, Hospital Universitário de Brasília, Faculdade de Medicina, Universidade de Brasília, Brasília, Brazil*

The varicella-zoster virus (VZV) is responsible for two infectious syndromes. Primo-infection generally occurs in infancy and manifests as varicella (chicken pox). Subsequent reactivation of the latent virus in the sensorial ganglia results in herpes zoster (HZ).[1] The incidence of HZ in the general population ranges from 1.2-4.8 cases per 1000 people in the United States,[2] and the incidence in SLE patients ranges from 3.2-43%,[3-6] 11% of which may be in a disseminated form.[2] Among immunocompetent individuals, 10-25% may have herpes zoster ophthalmicus (HZO).[1,6] There are few reports in the English language literature regarding patients with antiphospholipid syndrome (APS) who presented with VZV-related infections.[7,8] Herein, we report the case of a patient with SLE and APS who presented with HZO.

A 25-year-old female was diagnosed with SLE in 2000. She also had prior cutaneous, hematological, articular, and serosal involvement along with antinuclear and anti-double stranded deoxyribonucleic acid (anti-dsDNA) antibodies. In 2005, the patient was diagnosed with APS after an episode of thrombophlebitis of the left saphenous vein that was associated with high titers of anticardiolipin immunoglobulin M (aCl IgM) on two occasions (92 and 52 MPL) that were 12 weeks apart. The patient was on warfarin, chloroquine diphosphate 250 mg/day, prednisone 5 mg/day, and cyclosporine 100 mg/day. There was no clinical evidence of disease activity, although there was evidence of inflammatory activity and consumption of complement. The patient presented with hemicranial cephalalgia that was refractory to analgesics but had no neurological deficits. After four days, vesicular lesions appeared in the region of the opthalmic branch of the right trigeminal nerve that were consistent with a diagnosis of HZO. Ocular involvement was verified by the evaluation of a specialist. At that time, the patient's leukocytes were 4430/mm^3, and the lymphocytes were 1000/mm^3. The patient was treated with intravenous acyclovir along with clindamycin due to a secondary infection. The amount of prednisone was increased, and carbamazepine was introduced. The patient recovered without sequelae and did not present with any new thrombotic phenomena after two years of outpatient follow-up.

There is a wide array of lesions that may appear in patients with HZO. The sclera and the episclera may be involved in addition to the cornea, and damaging forms of keratitis (e.g., punctata epithelial, dendritic, nummular, or neurotrophic) may appear. Other complications that have been reported are anterior

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Correspondence: Jozélio Freire de Carvalho, M.D. Department of Rheumatology, Hospital das Clínicas da Faculdade de Medicina da Universidade de São Paulo, São Paulo, Brazil. Tel: 55-11-3061-7492 e-mail: jotafc@gmail.com

uveitis, glaucoma, acute retinal necrosis, cataracts, optic neuritis, and paralysis of the oculomotor muscles. Some cases present with blepharitis and conjunctivis along with secondary eyelid infections that are usually caused by *Staphylococcus aureus*.[6] It is also important to note the possibility of severe neurological complications, such as encephalitis and prolonged contralateral hemiplegia.[1]

The frequency of HZ infections in patients with lupus is elevated; however, it has not been previously reported in patients with APS. Rheumatologists should be attentive to the possibility of this occurring with the aim of promoting early and adequate treatment. In addition, it is necessary to take extreme care when monitoring patients, especially those expressing APA or those diagnosed with APS, after infection due to the risk of potential new thrombotic phenomena which might occur.

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