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Case report

Varicella zoster virus-associated neuroretinitis

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ABSTRACT

Varicella zoster virus-associated neuroretinitis is rare. We report a patient who presented with blurred vision of the left eye and extraocular movement pain. A fundoscopic examination revealed disc edema, hyperemia, and macular edema. The impression was neuroretinitis. Intravenous methylprednisolone pulse therapy was administered. However, visual recovery was incomplete with optical coherence tomography (OCT) imaging showing photoreceptor layer disruption. The laboratory data were rechecked and demonstrated a high varicella zoster virus immunoglobulin G titer. Varicella zoster virus-associated neuroretinitis was suspected and oral acyclovir was prescribed. His visual acuity improved to 0.9 after 2 weeks of treatment, and OCT showed photoreceptor layer restoration. Spectrum-domain OCT provides useful information when evaluating the disease course of neuroretinitis.

1. Introduction

Most patients with varicella zoster virus (VZV) posterior segment involvement present with acute retinal necrosis or optic neuritis. Varicella zoster virus is not usually considered a causative agent of neuroretinitis, and therefore diagnostic tests are often not performed. We report the clinical findings of an unusual case of VZV-related neuroretinitis in a healthy adult.

2. Case report

A 28-year-old male with no systemic diseases presented with blurred vision of his left eye with extraocular movement pain for 3 days. Ophthalmic examinations revealed that his best corrected visual acuity (BCVA) was 0.9 OD and 0.1 OS. The anterior segment was unremarkable, and the relative afferent pupillary defect of the left eye was positive. A fundoscopic examination revealed disc edema, hyperemia, and macular edema in his left eye (Fig. 1A). Spectrum-domain optical coherence tomography (OCT) demonstrated severe macular edema (Fig. 2A). He did not have a fever, trauma, or a history of animal contact. The laboratory data showed the patient was immunocompetent with a normal white blood cell count and differential count. The serologic test revealed that syphilis-rapid plasma reagin was nonreactive, *Bartonella henselae* immunoglobulin (Ig) G was negative, and toxoplasma IgM antibody was negative. Because of the patient's immunocompetent state, we did not check for human immunodeficiency virus. The impression was neuroretinitis. He was admitted and received intravenous methylprednisolone pulse therapy at 500 mg twice daily for 3 days. After discharge, he continued taking oral prednisolone with gradual tapering off. The disc and macular edema improved; however, some lipid exudate deposits were present on the macula area (Fig. 1B). The OCT image revealed photoreceptor layer disruption (Fig. 2B). His BCVA gradually increased to 0.7 and remained stable for 1 month. We rechecked his history and found he had vesicles around his trunk during this episode of neuroretinitis. Chickenpox was confirmed by a dermatologist. He had no history of varicella disease or vaccination. The laboratory data were rechecked and showed a high VZV IgG titer of 7075.91 mIU/mL, but was negative for herpes simplex virus (HSV)-1 and HSV-2 IgM. Varicella zoster virus-associated neuroretinitis was suspected and oral acyclovir was prescribed. His BCVA improved to 0.9 after 2 weeks of treatment, and the lipid exudate on the macula area gradually decreased (Fig. 1C). Photoreceptor layer disruption also improved on OCT imaging (Fig. 2C).

Conflicts of interest: None of the authors has any conflicts of interest to declare.

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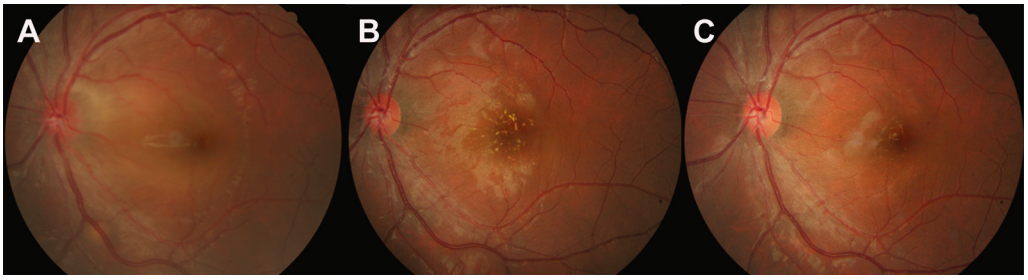

Fig. 1. Fundus photography. (A) Initial presentation shows disc edema, hyperemia, and macular edema. (B) Two months later, the disc and macular edema have improved with some lipid exudates on the macula area. (C) Three months later, the lipid exudates on the macula area have decreased.

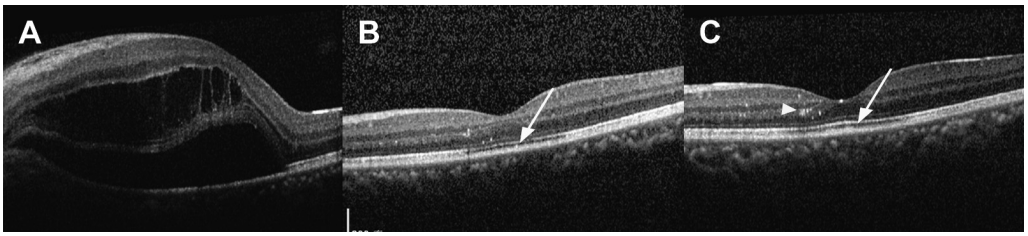

Fig. 2. Spectral-domain optical coherence tomography. (A) Initial presentation shows severe macular edema. (B) Two months later, the macular edema has improved, but photoreceptor layer is disrupted (arrow). (C) Three months later, the photoreceptor layer disruption has improved (arrow). Some residual exudates are present (arrowhead).

3. Discussion

Cases of neuroretinitis have been reported in association with a wide variety of infectious agents.[1] When more common etiologies such as *B. henselae* and *Toxocara canis* have been excluded, other rare pathogens should be considered. Varicella zoster virus-associated isolated neuroretinitis is extremely rare. Only one case report in a 9-year-old child has been reported in the literature.[2] The typical posterior segment involvement of VZV presents as acute retinal necrosis.[3] There are also some case reports of VZV optic neuritis after herpes zoster ophthalmicus.[4–6] Cases of VZV posterior segment infection have been reported in AIDS patients,[7–9] although it is uncommon in healthy adults.

The most sensitive method for confirming a diagnosis of varicella is the polymerase chain reaction (PCR) test, which detects VZV in skin lesions. Positive serum IgM suggests a primary infection, reinfection, or reactivation of latent VZV. A four-fold rise in IgG in paired serum samples in the acute and convalescent phases has an excellent specificity for varicella, but this method is not as sensitive as PCR of skin lesions.[10] Polymerase chain reaction and IgM and IgG titers in the acute stage of our patient at the initial presentation were unfortunately not performed. Based on concurrent neuroretinitis and varicella, and a high IgG titer in the late phase, we presumed that the patient's neuroretinitis was associated with the VZV infection.

In the present case, the patient's BCVA remained stable, although fundoscopic examinations revealed that the disc and macular edema gradually improved. The photoreceptor layer disruption on spectrum-domain OCT explained the condition. After undergoing combined treatment with oral steroids and antiviral agents, the disrupted layer was restored and his visual acuity (VA) increased.

Wong et al[11] reported that patients with VZV-induced acute retinal necrosis had a visual loss of 0.4 logMAR. In their report, adjunctive intravitreal foscarnet treatment reduced the risk of rhegmatogenous retinal detachment, compared to patients who received intravenous acyclovir treatment only.[11] Moorthy et al[8] demonstrated that VZV retinitis in immunocompromised patients had poor VA outcomes with an initial and final median VA of 0.5 and hand movements, respectively. Of 39 eyes examined, 19 (49%) eyes had no light perception at the last follow-up. The patients treated with a combination of intravenous ganciclovir and foscarnet therapy or ganciclovir alone had a significantly better final VA, compared to patients treated with acyclovir or foscarnet alone.[8] Visual outcomes are reportedly poor when steroids are used as monotherapy for optic neuritis in herpes zoster ophthalmicus.[12] Recent case reports have shown better visual outcomes when systemic acyclovir and steroids are prescribed.[5,6] MacKinnon et al[2] reported a case of VZV neuroretinitis in a 9-year-old child. The patient had blurred vision of the right eye with a VA of 0.01, after an episode of varicella. In addition, the right disc was hyperemic with peripapillary swelling and hemorrhaging, and the macular area was pale and edematous. Intravenous acyclovir and methylprednisolone were administered; however his final VA did not improve beyond 0.8. The right disc was pale and a yellow lipid deposit was present at the macula.

Our patient received systemic steroids in the acute phase, and his VA improved from 0.1 to 0.7. After oral acyclovir treatment in the late phase, his VA further increased to 0.9. However, we could not tell whether the good prognosis resulted from the therapy or was the natural course of the disease. The patient's VA moderately improved without antiviral drugs in the early stage. Further case collections are needed to clarify the best treatment for VZV neuroretinitis. In conclusion, VZV-associated neuroretinitis is rare. Spectrum-domain OCT provides useful information when evaluating the disease course of neuroretinitis.

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