CASE REPORT

Optic Neuritis in Herpes Zoster Ophthalmicus: A Rare Manifestation of a Common Malady

Chia-Chee Chew[1,2], Nurul Ain Masnon[1,2,3], Liza Sharmini Ahmad Tajudin[1,2], Wan Hazabbah Wan Hitam[1,2]

[1] Department of Ophthalmology and Visual Science, School of Medical Sciences, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia.
[2] Ophthalmology Clinic, Hospital USM, Universiti Sains Malaysia, 16150 Kubang Kerian, Kelantan, Malaysia.
[3] Ophthalmology Clinic, Hospital Kuala Lumpur, 50586 Kuala Lumpur, Wilayah Persekutuan Kuala Lumpur, Malaysia

ABSTRACT

Herpes zoster optic neuropathy (HZON) is a rare manifestation of herpes zoster ophthalmicus (HZO). It is a potentially blinding condition. We report a case of an immunocompetent patient with HZON following keratouveitis during the course of treatment. A 33-year-old gentleman presented with left eye (LE) worsening of visual acuity (6/9 reduced to 6/24) despite on treatment for HZO keratouveitis. It was associated with pain on ocular movement and central scotoma. He was on systemic acyclovir and topical corticosteroids prior to current complaint. Fundoscopy showed left optic disc swelling with impaired optic nerve functions. Diagnosis of left optic neuritis secondary to HZO was established in view of close temporal relationship with occurrence of cutaneous herpes zoster. Systemic corticosteroids was commenced. The patient had obtained good visual outcome at two months. Early referral for ophthalmology assessment is crucial to establish diagnosis of HZON and prompt initiation of treatment may preserve vision.

Keywords: Optic neuritis, Keratouveitis, Herpes zoster ophthalmicus, Corticosteroids

Corresponding Author:
Wan Hazabbah Wan Hitam, MSurg (Ophthal)
Email: hazabbah@usm.my
Tel: +609-767 6362

INTRODUCTION

Ocular complications of HZO may lead to substantial and variable of visual impairment. HZO has a wide spectrum of manifestations that involve both anterior and posterior segment of the eye. HZON is considered as a rare presentation and it has been reported to occur in 0.4% of eye with HZO (1). We report a case of HZO with keratouveitis, complicated with optic neuritis in an immunocompetent patient.

CASE REPORT

A healthy 33-year-old gentleman, presented with LE deterioration of vision three days after receiving treatment for HZO keratouveitis. It was associated with pain on ocular movement. He initially developed erythematous skin lesion over the left forehead. It subsequently evolved into vesicles one week prior onset of ocular symptoms. The vesicles extended to the left scalp region, left upper lid and also left-sided of the nose. It was associated with excruciating pain. He was then developed LE redness and photophobia. He was diagnosed with left HZO keratouveitis as there was evidence of circumcorneal injection, keratic precipitate, endothelial striae and inflammatory cells of 2+ in the LE anterior chamber. He was started on oral acyclovir 800mg five times a day and topical prednisolone acetate 1% four times a day for LE. He denied of having double vision, hearing problems or neurological symptoms. There was no positive history to suggest high risk behaviour or infection.

On examination, he was alert and afebrile. Multiple hyperpigmented scars were seen over the distribution of left trigeminal nerve (Hutchinson's sign) (Fig.1). The right eye (RE) best corrected visual acuity (BCVA) was 6/6, while the LE BCVA was 6/24 (dropped from 6/9 in the previous visit). There was positive left afferent pupillary defect (RAPD) and the LE optic nerve functions were reduced. There was reduction in light sensitivity and red desaturation to 80%, colour vision was also affected. The corneal sensation on the LE was noted to be decreased. The LE anterior segment examination revealed slight improvement of keratouveitis. The lens was clear and intraocular pressure was 16 mmHg.

Fundoscopy demonstrated generalised hyperaemic disc swelling with tortuous vein (Fig. 2A). Humphrey visual field showed an enlarged blind spot with caecocentral scotoma. Examination of RE was unremarkable. Erythrocyte sedimentation rate (ESR) was raised at 42 mm/hr. Fasting blood glucose was 5.6 mmol/l and hemoglobin A1c was 4.5%. Total white cell and differential counts

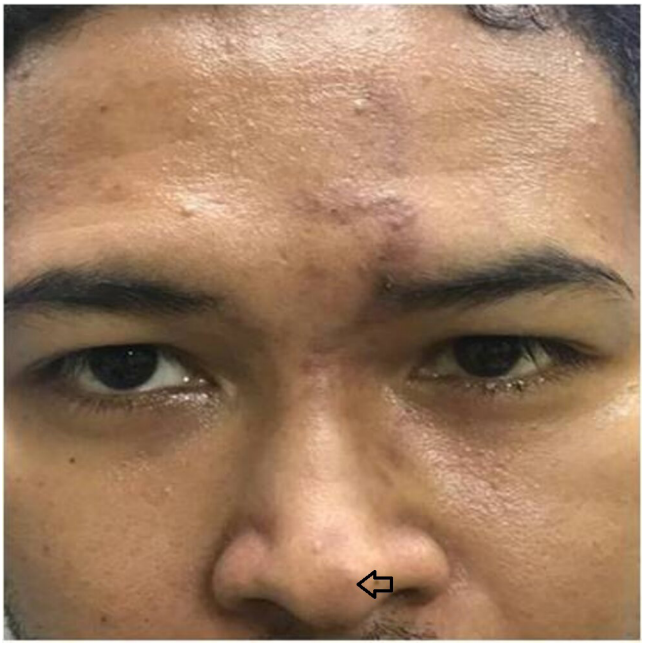

Figure 1: Photo showing mutiple hyperpigmented scars distributed over left forehead and involving tip of nose (positive Hutchinson's sign)

were normal. Investigations for infectious disease such as tuberculosis, syphilis, toxoplasmosis, bartonella and cytomegalovirus were negative.

He was diagnosed with left optic neuritis secondary to HZO complicated with keratouveitis. Oral acyclovir was continued for six weeks. Oral prednisolone 1mg/kg was started for two weeks and continued with tapering dose for another four weeks. Topical prednisolone acetate was tapered off in six weeks. His condition was improved with treatment. On followed up at six weeks, visual acuity had improved to 6/9 with clear cornea and quiet anterior segment. The swelling of the optic disc reduced. At 10 weeks, his visual acuity improved to 6/6 with complete resolution of keratouveitis and optic neuritis (Fig. 2B).

DISCUSSION

Herpes zoster ophthalmicus (HZO) is an infectious disease that commonly involves ophthalmic branch of the trigeminal nerve. It may lead to wide spectrum of sight threatening complications (2). Risk factors for reactivation of varicella zoster virus include older age and immunocompromised status such as HIV infection, lymphoma, leukemia, immunosuppressive therapy and diabetes mellitus. In the present case, our patient had none of these risk factors and was extensively investigated.

Ocular involvement in HZO follows the onset of rash over one to four weeks. It is relatively common, accounting for 10-25% of the total cases. However, optic neuritis associated with HZO is a rare and unusual ocular complication, only reported involved 0.4% of eyes with

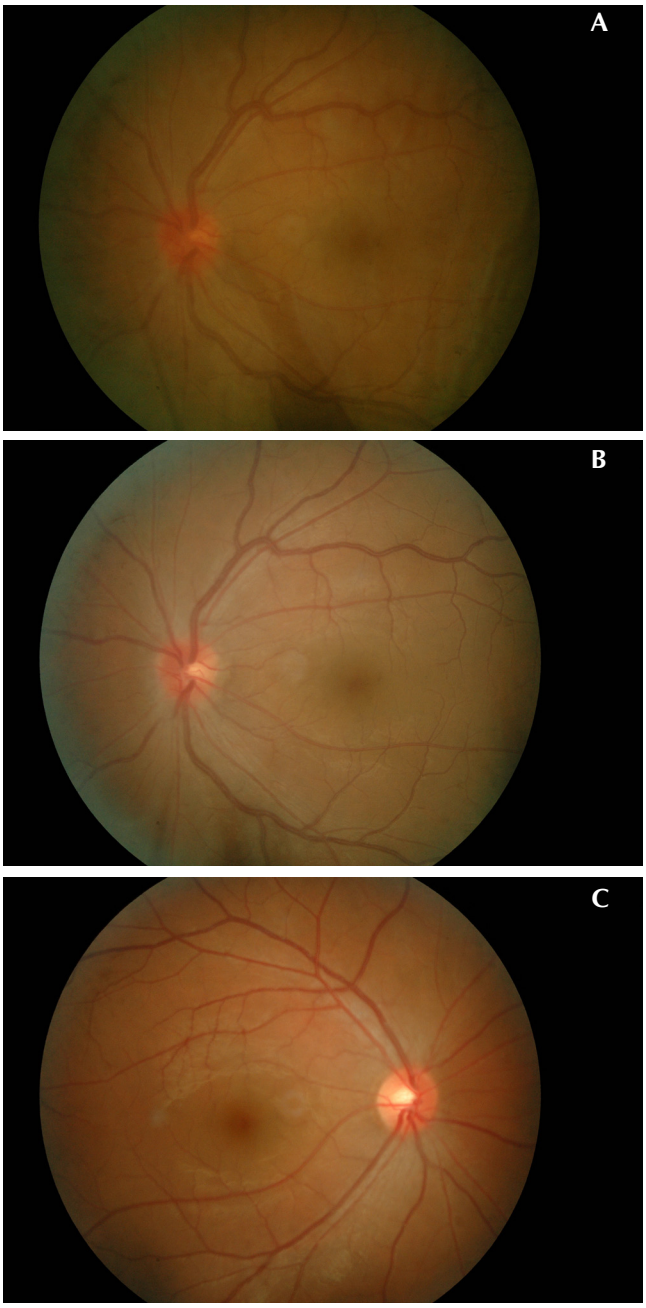

Figure 2: (A) Fundus photo of left eye at presentation showing hyperaemic swelling disc and tortuous vein. (B) At ten weeks follow up, the left eye optic disc swelling was completely resolved. (C) Normal fundus photo of right eye.

HZO (1). The time to developing optic neuritis varies from weeks to months after onset of the characteristic rash (3). Our patient developed optic neuritis ten days after the rash onset, which is comparable to mean 14.1 days previously reported (3). HZON may present either in anterior or retrobulbar form . Our patient presented with anterior form of HZON with reduced vision, pain on ocular movement and central scotoma. The diagnosis of HZON was made in view of there is close temporal relationship of optic neuritis after the development of cutaneous herpes zoster. Ancillary laboratory testing was done as a complementary test to diagnose HZON, particularly to exclude other possible cause of optic

neuropathy and immunocompromised status.

The exact mechanism of optic neuritis following HZO is unclear. Three possible mechanisms had been proposed and it is difficult to differentiate from each other. Virus may spread via the cavernous sinus and superior orbital fissure to the orbit causing optic nerve injury. Secondly, local extension into meninges and brain tissue, which may lead to meningoencephalitis and in turn damage the optic nerve. Another possible mechanism of optic nerve damage is generalised ocular ischemia caused by extensive inflammation of posterior ciliary arteries and nerves.

There was no consensus on the duration treatment for HZON. The duration of treatment in previous reports ranged from ten days to two month (3). Combination of systemic acyclovir in addition to corticosteroids is widely accepted in treating HZON and associated with good outcome (3). Acyclovir is commonly used either in oral or intravenous route. Corticosteroids are used in different routes including topical, oral and intravenous depends on severity of HZON. Visual outcome had been reported poor when corticosteroids are used without antiviral therapy in the setting of HZON (4). In the present case, our patient was started on systemic acyclovir and topical corticosteroids due to presence of keratouveitis prior to the onset of HZON. Despite aggressive systemic acyclovir, he progressed to HZON three days after initiation of antiviral treatment and topical corticosteroids.

He was continued on oral acyclovir 800 mg five times a day for six weeks. Oral prednisolone 1mg/kg was started for two weeks and continued on tapering dose for four weeks. Patient's visual acuity had improved to 6/6, optic disc swelling had subsided and normalised optic nerve function at 10 weeks. There were no side effects in the patient's treatment regime. The visual prognosis of HZON varies (3). Severe visual acuity impairment upon presentation of optic neuritis has been associated with poor visual outcome (5). The visual outcome of our patient was good and there were no sequelae after a course of treatment with anti-viral and corticosteroids.

CONCLUSION

Secondary optic neuritis is a rare complication of HZO. It may present at initial presentation or during the course of treatment. Early ophthalmology referral and assessment is crucial for treatment initiation. The use of antiviral treatment and corticosteroids may lead to better visual outcome.

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