Case report

Herpes zoster ophthalmicus with retrobulbar neuritis

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Abstract

Background: Retrobulbar neuritis in Herpes Zoster Ophthalmicus (HZO) has been reported very rarely. **Objective**: To report a very rare case of HZO with retrobulbar neuritis with detailed clinical features and treatment responses. **Case**: A fifty-eight-year old male presented with Herpes zoster-retrobulbarneuritis in the left eye. It was characterized by decreased visual acuity, mid dilated pupil with sluggish reaction, normal optic disc and central scotoma in Humphrey visual field. Visual acuity improved with systemic Acyclovir and steroids. **Conclusion**: This is a rare case of HZO associated with retrobulbar neuritis. Prompt treatment with systemic antiviral and steroid improve the visual outcome.

Keywords: Herpes zoster ophthalmicus, retrobulbar neuritis, visual field

Introduction

Herpes Zoster is caused by the reactivation of the varicella zoster virus (VZV), zoster usually presents as a painful dermatomal rash. In addition to skin or mucosal involvement, VZV reactivation commonly affects the ophthalmic division of the trigeminal nerve and subsequently the eye. This manifestation is termed herpes zoster ophthalmicus. The most common ocular complications of HZO are conjunctivitis, keratitis and uveitis but others include: edema of the eyelid, ocular hypertension, trabeculitis, pseudodendritis, keratouveitis, scleritis, retinal vasculitis, central retinal artery occlusion, choroiditis, acute retinal necrosis and optic neuritis (Liesegang TJ, 1985).Throughout the world, the incidence rate of herpes zoster every year ranges from 1.2 to 3.4 cases per 1,000 healthy individuals, increasing to 3.9–11.8 per year per 1,000 individuals among those older than 65 years (Donahue JG et al, 1995). Immunosupression, malignancies, chemotherapy, tuberculosis, trauma and aging greatly increase patient's risk for reactivation of the latent Varicella. Here we present a rare case of HZO with retrobulbar neuritis and treatment responses.

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Case report

A 58-year-old male attended the emergency with rashes and swelling in left side of the face of three days duration. Initially the rashes appeared on the upper lids later extended towards the forehead. It was associated with severe pain and left sided face swelling that the patient was not able to open his lefteye. There was no history of fever, malaise, common cold prior to presentations of symptoms. He had no history of trauma,weight loss, chronic diarrhea or skin rashes during childhood. There was no history suggestive of any systemic disease or chronic illness in the past.

On examination vesicular rashes were present on left sided forehead and eyelid with severe

swelling of face and neck. It was erythematous and tender. Ocular examination of right eye was normal. On the day of admission,left eye examination was not possible as the eyelids were opposed to each other and was not able to retract due to severe swelling (Figure 1). It was associated with left sided neck swelling. He was admitted to the hospital and oral Acyclovir 800mg five times a day and Ointment Acyclovir 3% five times a day were started and continued for 14 days. Dermatologist and ENT consultations were done too. Ointment steroid with antibiotic was advised to apply over the rashes. Intravenous Ceftriaxone 1gmxBD and Metronidazole 500 mgx TDS were started for left sided neck swelling by ENT surgeon. On the second day of admission, swelling was slightly decreased so that the ocular examination of left eye was possible. Unaided visual acuity of his left eye was 6/36 and was no improvement with Pinhole. Extraocular movements were in full range with no diplopia. Hutchinson's sign was absent. Slit lamp examination of left eye revealed circumciliary congestion of conjunctiva, fine small keratic precipitates were seen on inferior corneal endothelium with decreased corneal sensation, anterior chamber cells grade II was noted, pupil was mid-dilated about 4mm in size with sluggish reaction, iris pigment deposits were seen in anterior lens capsule (Figure 2). Fundus examination was normal in both eye(Figure 3). Intraocular pressures were normal bilaterally. Colour vision was normal. Investigations of hemoglobin, total count, differential count, ESR, RBS, Na/K, urea, creatinine, HIV I and IIwere within normal limits. Humphrey Visual field (HFA 24-2) revealed central scotoma in left eye whereas normal in right eye (Figure 4). A diagnosis of herpes Zoster ophthalmicus with retrobulbar neuritis was made. He was started on oral Prednisolone 60mg once a day along with antacids for 7 days on tapering dose for one month. Topical steroid andtropicamidewerestarted as there were signs of inflammation. He was discharged on fifteenth day as the symptoms of pain,swelling, rashes had decreased significantly and improvement in vision was noted(Figure 5). But the anisocoria persisted as before. The patient was seen at 8 months follow up and his VA was improved to 6/6. Repeat HFA 24-2 was done which was normal (Figure 6).

Figure:1 showing vesicular rashes on the left sided forehead with severe lid edema.

Figure 2: Showing LE circumciliary conjunctival congestion,mid-dilated pupil about 4 mm.

Figure 3: Fundus photograph showing BE normal fundus

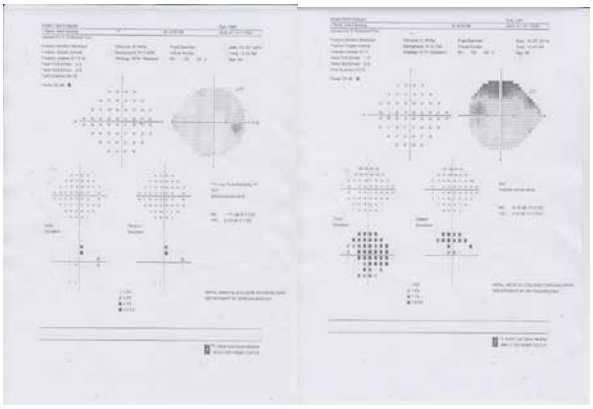

Figure: 4 showing left eye central scotoma on HFA 24-2.

Figure 5: Showing decrease rashes and swelling

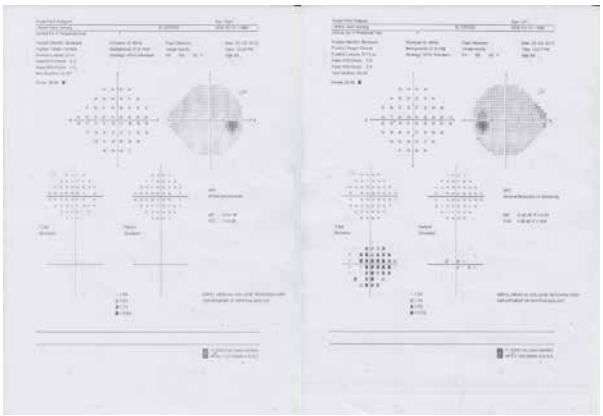

Figure 6: Normal HFA on follow-up

Discussion

Varicella-zoster virus manifests itself as two distinct syndromes in humans. During primary infection, the virus causes chicken pox. After the initial infection, VZV remains latent in the dorsal root ganglia of sensory neurons then reappear as herpes zoster later in the life. Risks for reactivation include any decline in the T-cell mediated immune response including that caused by normal aging, HIV/AIDS, and immunosuppressive medications. In the present case he was of late fifty but neither HIV positive nor on any immunosuppressive medications. As aging starts, develop decreased cell-mediated immunity and it is by this mechanism that the virus is allowed to become active after having been held quiescent for many years (Hope-Simpson RE, 1965). HZO is reactivation of VZV in the ophthalmic division of the trigeminal nerve (V_1), and accounts for 10–25% of all herpes zoster cases (Ragozzino MW et al, 1982). Involvement of nasocilliary nerve (Hutchinson's sign) indicate 100% development of eye pathology (Zaal MJ et al, 2003). Approximately one third of those without nasocilliary involvement will eventually develop eye manifestations (Harding SP et al, 1987). In this case also there was no involvement of nasociliary nerve but ocular manifestations were present. HZO may lead to severe pain and a wide spectrum of sight-threatening complications affecting all ocular and orbital tissues (Culbertson WW et al, 1986). About 60 percent of patients have varying degrees of dermatomal pain in the distribution of the ophthalmic nerve (Cobo M et al,1987). Our patient also presented to emergency with severe pain,rashes and swelling in the left side of the face. Involvement of optic nerve is very rare sequelae of HZO. It may present as papillitis, optic neuritis,retrobulbar neuritis or optic nerve infarction (Gunduz K et al, 1994). The exact mechanism of involvement of optic nerve in not known. However, the optic nerve may be involved by these mechanisms.

1) Direct extension of the virus through the cavernous sinus to nerves, muscles and the optic nerve(Gunduz K et al, 1994).

2) Local extension into the meninges and brain tissue may lead to mild meningoencephalitis, which in turn, may damage the optic nerve (Gunduz K et al, 1994; Scharf Y et al, 1987).

3) Generalized ocular ischemia caused by inflammation (Gunduz K et al, 1994).

In the current case,retrobulbar neuritis was demonstrated by decreased Visual acuity, mid dilated pupil with sluggish reaction, normal optic disc as seen by slit lamp examination. HFA24-2 test showed central scotoma. Following antiviral medication visual acuity generally improves. Most studies suggest that systemic steroid has a good clinical response for the treatment of optic nerve involvement of HZO(Marsh RJ et al, 1993). In this case also visual acuity improved after administration of oral Acyclovir along with oral steroid.

Here we present a rare case of zoster- related retrobulbar neuritis. Prompt treatment with oral Acyclovir and steroids have improved the visual outcome. Ocular complications of HZO if detected early and managed properly can prevent or minimize visual damage.

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Consent
Informed consent for publication of this case report and accompanying images was obtained from the patient.

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