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Case Report

Cavernous Sinus Syndrome Causing Complete Ophthalmoplegia in Acute Herpes Zoster Ophthalmicus: A Rare Occurrence

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Abstract

Herpes Zoster Ophthalmicus (HZO) is associated with variety of ocular manifestations like episcleritis, keratitis, glaucoma, and cataracts. Extraocular muscle palsies are rare complications occurring in 7-31% of HZO. Complete unilateral ophthalmoplegia in HZO is rare. We report an elderly patient with left HZO who developed progressive left complete ophthalmoplegia with complete ptosis within 10 days of onset of a zosteriform rash. Magnetic resonance imaging brain showed contrast enhancement in cavernous sinus which has not been reported so far.

Introduction

Herpes zoster refers to a typically vesicular rash caused by reactivation of the latent Varicella Zoster Virus (VZV) from the dorsal root ganglia neurons. It usually presents in thoracic or cranial dermatomes. Herpes Zoster Ophthalmicus (HZO) is a disease wherein VZV replicates in gasserian (trigeminal) ganglion and produces neural inflammation and epidermal facial lesions in areas supplied by sensory branches of the ophthalmic division (V1) of the trigeminal nerve [1]. HZO is associated with variety of ocular manifestations like episcleritis, keratitis, glaucoma, and cataracts. Extraocular muscle palsies are rare complications occurring in 7-31% of HZO [1]. Complete unilateral ophthalmoplegia defined as impaired ocular duction movement in all 4 directions within 3 months of onset of manifestations of HZO is rare [2]. Herein, we report an elderly patient with left HZO who developed progressive left complete ophthalmoplegia with complete ptosis within 10 days of onset of a zosteriform rash. Brain Magnetic Resonance Imaging (MRI) showed contrast enhancement in cavernous sinus.

Case Report

A 62-year-old man presented with an inability to open his left eye of one week duration. He was apparently asymptomatic 5 weeks before the present complaints. He developed excruciating pain in left side of the forehead, left retrobulbar pain lasting for 5-7 days, five weeks back. Subsequently, after 7 days, he noticed skin rash in the form of vesicular eruption around the left eye, left side of the forehead and nasal bridge up to its tip. The pain decreased with the onset of skin rash. He received oral acyclovir (800 mg, five times a day for 5 days). Over the next 2-3 weeks, the vesicular eruptions got healed with crusting. Ten days after the vesicular eruption, he developed pain again in the left hemicranial region. It was associated with double vision which was binocular, horizontal and appearing at primary position and gaze towards left. His family noticed that his eye was becoming increasingly droopy culminating in it being permanently closed for 2 days prior to the admission. No history of decrease in vision. No limb weakness. No significant co-morbidities in the past. On examination, cicatricial skin lesions with crusts involving the left scalp, left side of the forehead, nasal bridge up to its tip, left eyelids were observed. No evidence of keratitis. Visual acuity and fundus examination in both eyes was normal. Right pupil was 2.5mm, reactive to light; left pupil was dilated and non-reactive to light. There was complete ptosis on the left side with complete ophthalmoplegia which was painless (Figure 1). Other cranial nerves were normal. The remainder of his neurological examination did not reveal any abnormality. Complete hemogram, renal, hepatic and thyroid function tests were normal. Glycosylated haemoglobin was normal. Serological testing for Antinuclear Antibody (ANA), Human Immunodeficiency Virus (HIV), Hepatitis B virus (HBsAg) and Venereal Disease Research Laboratory (VDRL) were negative. Brain MRI with contrast showed contrast enhancement in sellar and parasellar region (Figure 2). No brainstem lesion. Cerebrospinal Fluid (CSF) analysis did not reveal pleocytosis or presence of malignant cells. The diagnosis of left cavernous sinus/ superior orbital fissure syndrome was made. As the ipsilateral HZO preceded the ophthalmoplegia by 2 weeks, it was considered his signs and symptoms were

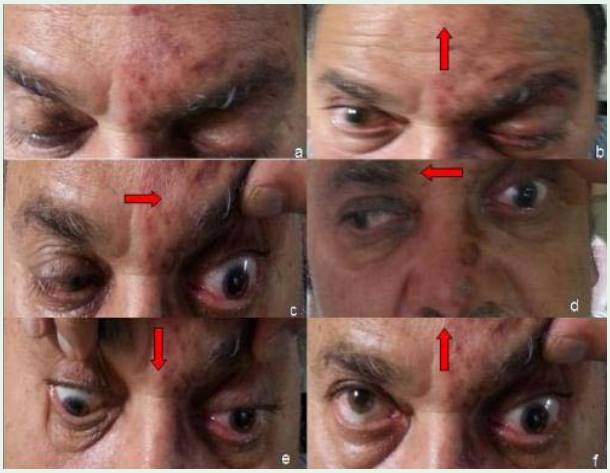

Figure 1: (a) Crusted erythematous skin lesion over left forehead; (b) complete ptosis of left upper eyelid; (c), (d), (e) & (f) complete restriction of left eye movements with crusted lesion over nasal bridge (d).

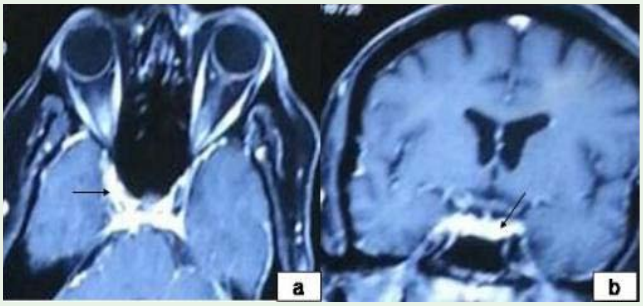

Figure 2: (a) Brain MRI T1 weighted imaging post gadolinium contrast axial view showing contrast enhancement in both parasellar region (black arrow); (b) coronal view showing contrast enhancement in sellar region (black arrow).

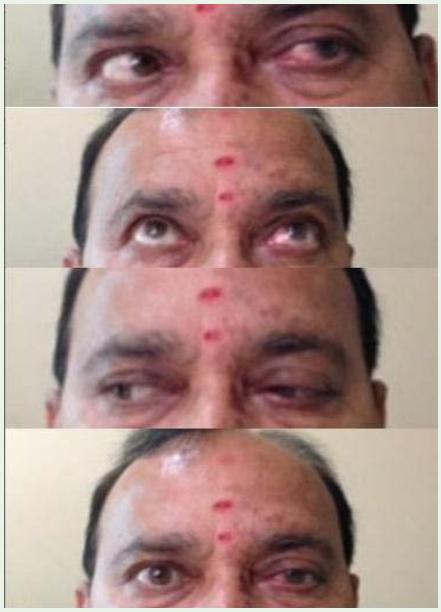

Figure 3: Normalization of eye movements in the left eye.

related to Varicella Zoster Virus (VZV). He was treated with oral prednisolone (60 mg daily for the first 5 days, then gradual reduction to 5 mg) with intravenous acyclovir (500 mg three times a day for 14 days). There was improvement in the left eye opening during hospital stay. At 3 months follow up, eye movements returned to normality (Figure 3).

Discussion

Ophthalmoplegia secondary to HZO has been described typically as a delayed complication, often up to 2 to 3 months after the initial herpetic rash and is seen in only 7-31% of patients with HZO [3]. However, our patient developed the same as part of the acute viral infection which is rarely reported. The oculomotor nerve is most commonly affected, followed by the abducens nerve; the trochlear nerve appears to be the least frequently involved [4]. Complete ophthalmoplegia involving oculomotor, trochlear and abducens nerves together in HZO are rare [2].

In HZO, VZV reactivates in the trigeminal ganglion due to a diminished virus-specific, cell-mediated immunity, which is related to age. They migrate along the ophthalmic division (V1) of the trigeminal nerve and spreads to the corresponding dermatome. Various hypotheses have been proposed for ophthalmoplegia in HZO. The reactivated virus causes inflammation of the axons that supply the dermatomes. Inflammation of the trigeminal nerve could actually spread via the cavernous sinus to affect the oculomotor nerve and neighboring cranial nerves causing direct viral cytopathic effect [4]. Secondly, a reactive, immunologically mediated response to the virus might induce perineural inflammation, peripheral nerve demyelination, contiguous orbital inflammation, cranial vasculitis, myositis [5] and meningitis. In our patient, brain MRI showed enhancement in parasellar region involving the cavernous sinus suggesting possible inflammatory response to the viral reactivation. Earlier reports on ophthalmoplegia following HZO have reported high signal intensity on MRI in the extraocular muscles suggesting myositis. Enhancement of ocular motor cranial nerves has not reported [2]. Other conditions which can present with ophthalmoplegia with cavernous sinus enhancement on MRI imaging are the inflammatory conditions like Tolosa-Hunt syndrome, sarcoidosis, wegener's granulomatosis, Giant cell arteritis, and idiopathic hypertrophic pachymeningitis; infectious disease like fungal (mucormycosis, aspergillosis, actinomycosis), tubercular, pyogenic and parasitic; lymphomas, melanomas.

The use of systemic corticosteroids may be effective to prevent occlusive vasculitis along with nucleotide analogues. The prognosis is good after complete ophthalmoplegia following HZO. Chang-Godinich, et al. (1997) reported 16 patients with complete ophthalmoplegia and on follow up of 9 patients; they showed significant improvement in symptoms after 2 months and almost complete resolution by 18 months [3].

Conclusion

Cavernous sinus syndrome in acute HZO with its correlation with MRI brain has not been reported. Complete unilateral ophthalmoplegia is a rare complication of HZO. Contrast enhancement in cavernous sinus as demonstrated in our patient suggests possible inflammatory response to the viral reactivation.

Treatment includes systemic treatment with corticosteroids (to control the inflammatory response) together with systemic antiviral.

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