Superior Orbital Fissure Syndrome with Orbital Abscess: Presentation of a Herpes Zoster Ophthalmicus Case.

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Abstract: Orbital abscess and superior orbital fissure syndrome (SOFS) are rare manifestations of herpes zoster ophthalmicus. Herein, we report a case of SOFS along with orbital abscess in a 5 year old male child secondary to herpes zoster infection. He presented with a 5-day history of proptosis and ptosis of the right eye that had been preceded by vesicular eruptions on the right forehead and scalp. CT scan of the head and orbit showed orbital abscess. A diagnosis of SOFS with orbital abscess secondary to herpes infection was made. The condition subsequently improved following antiviral therapy, intravenous vancomycin and amikacin, and oral corticosteroids.

Key words: Herpes zoster, orbital abscess, proptosis, superior orbital fissure syndrome

I. Introduction

Herpes zoster ophthalmicus (HZO) is caused by varicella-zoster virus (VZV). Ocular manifestations include keratoconjunctivitis, keratitis, anterior uveitis, acute retinal necrosis, acute phthisis bulbi, central retinal artery occlusion, optic neuritis, orbital pseudotumors, and partial or complete paralysis of ocular motility¹. Rarely, it can manifest as superior orbital fissure syndrome (SOFS)². Orbital abscess along with SOFS has not been reported in the searched literature. We herein report a case of a 5-year-old child presenting as SOFS with orbital abscess secondary to HZO.

II. Case Report

A 5-year-old-male child was brought with a chief complaint of acute onset drooping of the right upper eyelid that persisted without improvement for 5 days. There was a preceding history of fever of 10 days duration followed by a vesicular eruption on the right scalp and forehead extending to the tip of the nose. Prior to the presentation at our institution, he had received oral paracetamol and Neosporin eye ointment applied locally. There was no improvement of condition with the treatment. Review of systems was negative for a history of seizures, throat pain, and no similar rashes in any other part of the body. The child when seen in outpatient department was afebrile and quiet with the pulse rate of 88/min and respiratory rate of 22/min. Ocular examination revealed right eye axial proptosis with restriction of extraocular movements in dextroversion. There was a moderate blepharoptosis on the right side. Visual acuity was grossly normal. The anterior segment and fundus examination revealed no abnormalities. The left eye was normal.

There were multiple healed hyperpigmented lesions on the right side of the scalp, forehead, and nose, suggestive of herpes zoster infection. There were no signs of meningitis. Hematological examination showed a total leukocyte count - 12,700 cells/mm cu, hemoglobin - 10.8 g/dl, platelet count - 150,000 cells/mm cu, random blood sugar - 95 mg/dl, HIV/hepatitis B surface antigen/hepatitis C virus test - negative, and amylase - 29 IU/L (25–200). Microbiological examination of the urine was normal. Blood and urine cultures revealed no organism. X-ray of the chest and paranasal sinuses were normal. Computed tomography (CT) scan of the orbit and head showed orbital abscess in the right retrobulbar space. Maxillary and ethmoidal sinuses had features suggestive of sinusitis. The child was commenced on intravenous injection vancomycin 60 mg/kg/day in two divided doses, amikacin 20 mg/kg 3 times daily and oral acyclovir 30 mg/day for 14 days. In view of stable general condition, we continued with oral acyclovir. Intravenous antibiotics were added to treat intraorbital abscess with maxillary and ethmoidal sinusitis. With treatment, ptosis and proptosis improved, and the patient regained full extraocular movements. Visual acuity was grossly normal. The patient was discharged after 2 weeks on oral Amoxyclav for further 2 weeks. At 5 months follow-up, the condition was improved.

III. Discussion

SOFS is an infrequently described and reported symptom complex. According to Kurzer and Patel, the syndrome was first described by Hirscfield in 1858.³ SOFS consists of the following signs: ptosis of the upper eyelid, proptosis of the globe, ophthalmoplegia, fixation and dilatation of the pupil, and anesthesia of the upper eyelid and forehead.⁴

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The superior orbital fissure serves as a pathway that allows communication between the orbit and the middle cranial fossa.⁵ It lies at the apex of the orbit, bounded medially by the lesser wing of the sphenoid, inferiorly and laterally by the greater wing of the sphenoid, and superiorly by the frontal bone.⁵ The fissure transmits the oculomotor, trochlear, and abducens nerves (cranial nerves III, IV, and VI), as well as the first three branches of the trigeminal nerve: the frontal, lacrimal, and nasociliary nerves.⁵,

Numerous etiologies of the syndrome have been reported in the literature including syphilis, craniofacial fractures, hematoma of the cavernous sinus or retrobulbar space, infection, neoplasm, aneurysm of the internal carotid artery or arteriovenous fistulae, or idiopathic etiologies^{3,4,5,6,7}. Regardless of the etiology, the clinical symptoms are primarily the result of inflammation and compression of adjacent nervous tissue.⁶

HZO is an infection triggered by the reactivation of VZV that lies dormant in the trigeminal nerve ganglia. In literature, 7% of all cases of herpes zoster present as HZO. Of these cases, 20–79% has orbital involvement⁸. HZO is usually reported in older and immunocompromised patients. The clinical presentation of the patient with a typical distribution of herpes zoster lesions, ptosis, proptosis, limited extraocular movements with intact pupillary reflex, and normal visual acuity were consistent with the diagnosis of SOFS with orbital abscess secondary to HZO. CT scan findings supported this diagnosis.

Many pathogenic mechanisms are invoked as the cause of total ophthalmoplegia in herpes zoster infection. These include the third, fourth, and sixth cranial nerve compression from orbital soft tissue edema⁹ and direct spread of the VZV from the fifth cranial nerve to the third, fourth, and sixth in the region of the superior orbital fissure or cavernous sinus⁹. The available literature on HZO with SOFS has shown improvement with the use of systemic antiviral drugs and steroids². Treatment regimens are controversial, and the effects of steroids or antiviral treatment have not been formally studied, requiring further randomized controlled clinical trials. There are reported cases of orbital abscess that have improved with medical management alone¹⁰. Given the patient's general stable condition, he was treated with a combination of intravenous antibiotics and oral acyclovir which led to improvement of clinical signs and symptoms. Patients <9 years respond to medical management more frequently than older patients, but recent studies confirm that even children over 9 years of age with small or moderate-sized abscesses and normal vision deserve a trial of medical therapy before surgical intervention¹⁰. Had there been no improvement, we would have considered surgical drainage of the abscess.

IV. Conclusion

The co-occurrence of orbital abscess with SOFS is a relatively unusual presentation of HZO. In this case, early diagnosis with an appropriate treatment led to a favorable outcome for the patient.

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