MRI IN A CASE OF HERPES ZOSTER OPHTHALMICUS

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ABSTRACT

A case report of herpes zoster ophthalmicus was demonstrated by MRI study in a 61 year-old female patient. There was an abnormal enhancement of the intraorbital structures of left eye, the tissue in left eye was mildly thickened . The increased enhancement of left trigeminal nerve was noted.

INTRODUCTION

The most commonly reported complication of herpes zoster is postherpetic neuralgia.[1] Ocular involvement is shown in 20%-71% of cases of herpes zoster ophthalmicus[2] and includes: keratoconjunctivitis,[3] ocular motor palsies,[4] acute retinal necrosis,[5] acute phthisis bulbi,[6] optic neuritis,[7] and central retinal artery occlusion.[8] MR is proved to be useful for identifying the wide array of inflammatory and ischemic complications associated with herpes zoster ophthalmicus. Serial MR may document both the regression and progression of various aspect of this unusual disorder.[9]

CASE REPORT

A 61-year-old man came to the eye clinic because of decreasing vision to hand movement of his left eye for 20 days. Scar and ptosis of the left eyelid was found. Slit-lamp examination of left eye revealed ciliary injection, generalized bedewing with stromal edema and pigmented keratic precipitates of the cornea. There was hyphema about 1.6 mm in height in the left anterior chamber. There was impaired downward and medial movement of the left eye. The left pupil was semidilated and did not react to light, whereas the right pupil showed positive reverse relative afferent pupillary defect. Slit-lamp examination of the right eye was unremarkable. ESR was 68 mm/ hour. Complete blood count and blood chemistry was within normal limit. Anti- HIV titer was nonreactive.

MRI of the orbits was performed. It showed abnormal enhancement in left orbit: along left optic nerve (Fig.1) , in the mildly enlarged extraocular muscles of left orbit (Fig.1,2), of the intraorbital fat (Fig.1,2), of the mildly thickened sclera of left eyeball (Fig.3), at the cranial nerve V (Fig. 4) (cisternal and intracavernous portion) and the 1st division of the cranial nerve V. The pre-septal soft tissue was slightly thickened and enhanced (Fig.5). The left ethmoid sinus was mildly inflammed (Fig.5).

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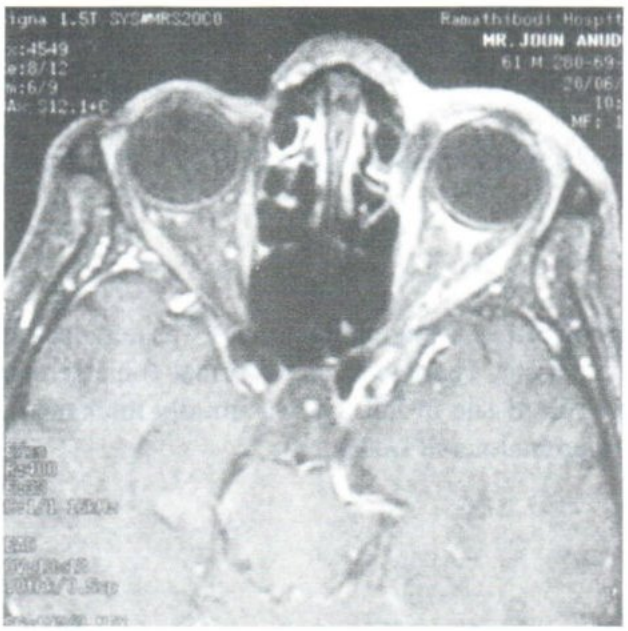

Fig. 1. I.V. enhanced axial MRI of the orbits showed abnormally increased enhancement of the intraorbital fat, subcutaneous fat plane over left eye.

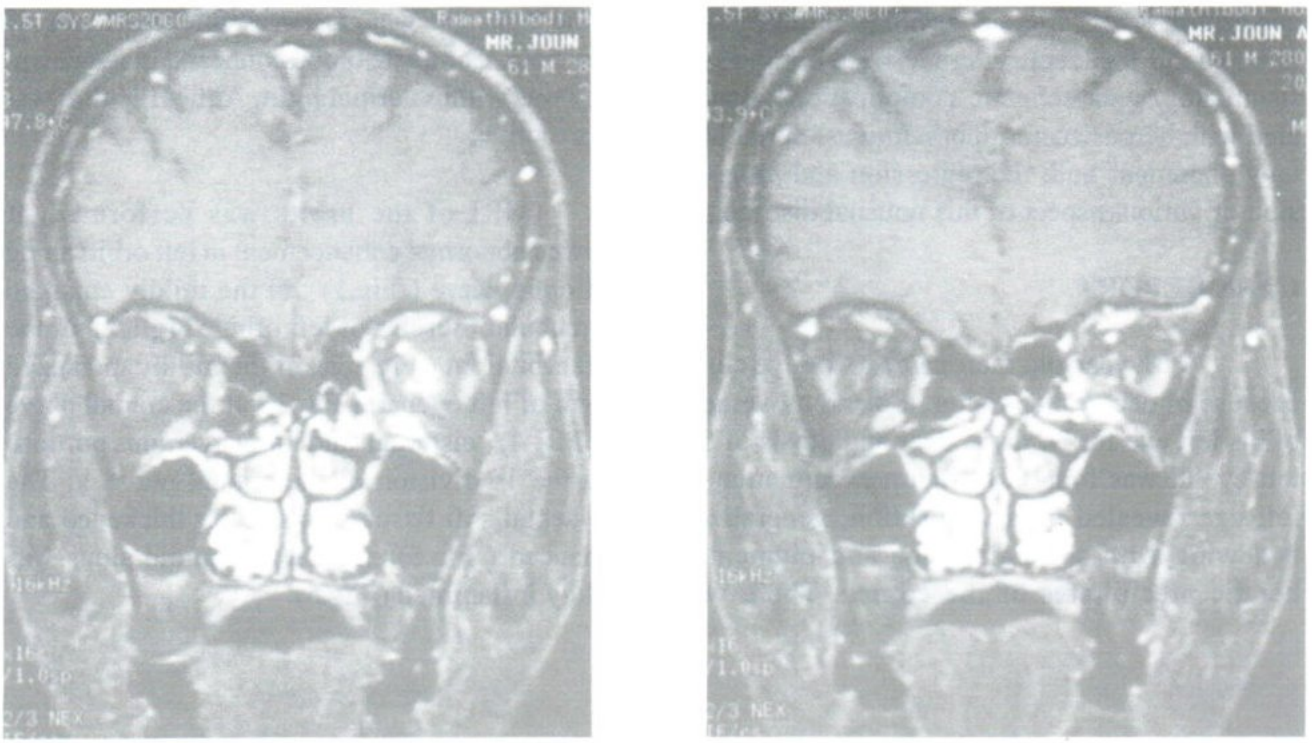

Fig. 2. I.V. enhanced coronal MRI of the orbis showed abnormally increased enhancement of the mildly enlarged extraocular muscles of left orbit.

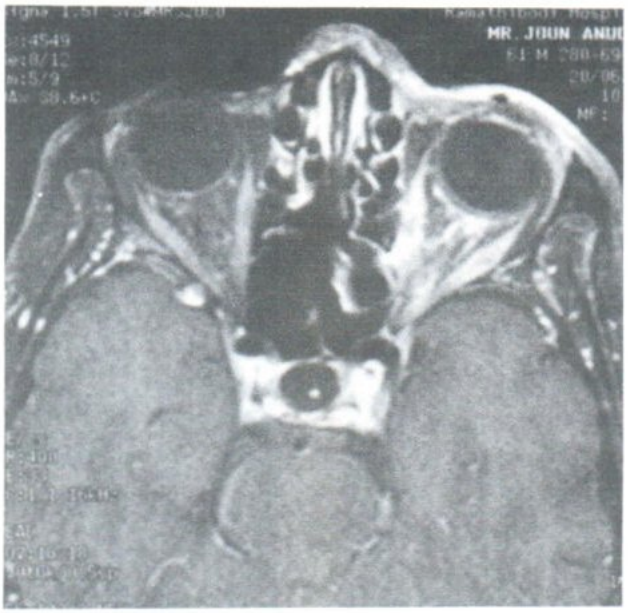

Fig. 3. I.V. enhanced axial MRI of the orbits showed abnormally increased enhancement of the mildly thickened sclera of left orbit.

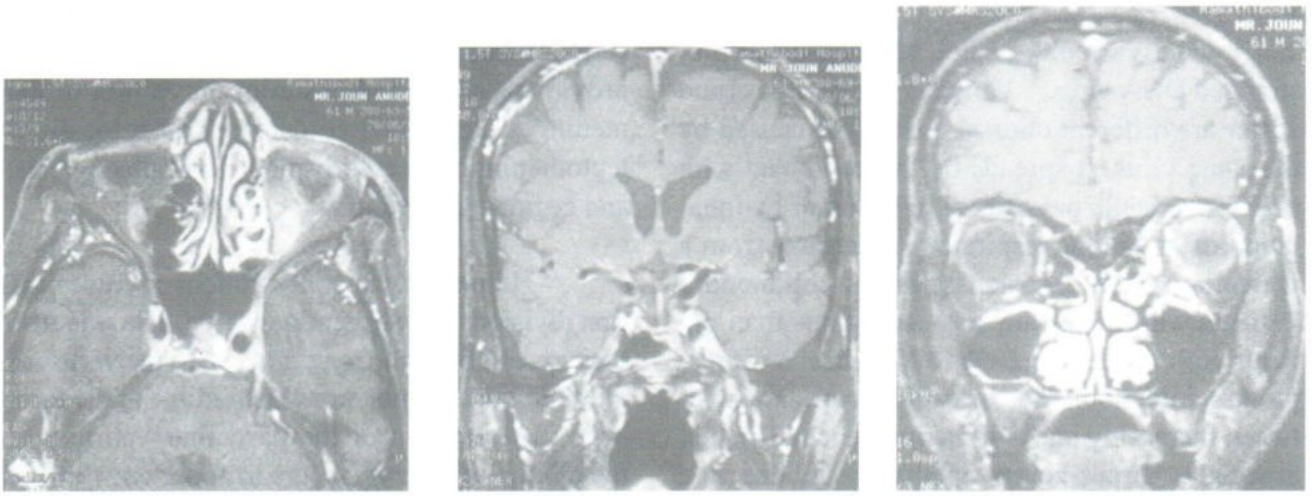

Fig. 4. I.V. enhanced axial and coronal MRI of the orbits showed abnormally increased enhancement of the cisternal, intracavernous portions of the trigeminal nerve and the V1 branch of left side.

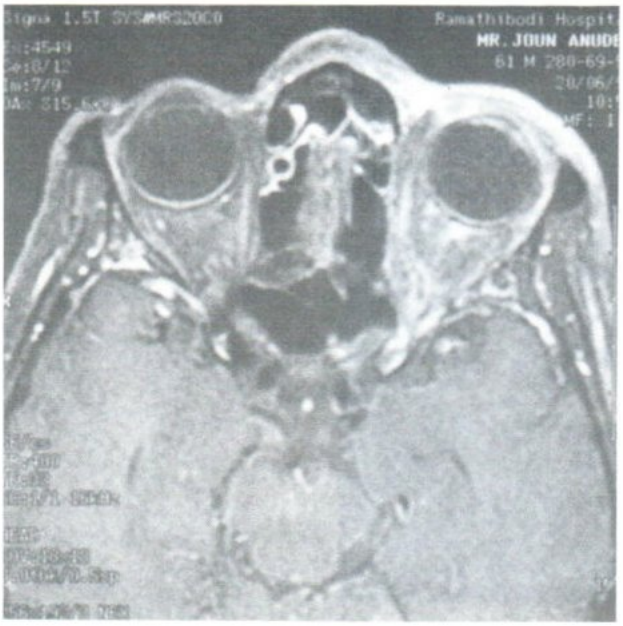

Fig. 5. I.V. enhanced axial MRI of the orbits showed abnormally increased enhancement of the preseptal soft tissue, subcuta neous fat over left eye and nose, besides enhancement of the intraorbital fat.

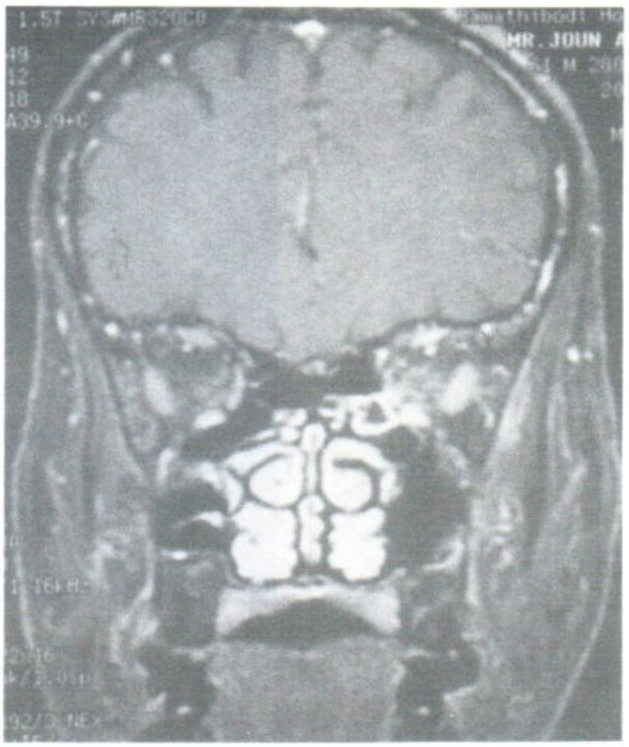

Fig. 6. I.V. enhanced coronal MRI of the orbits showed mild ethmoidal sinusitis.

DISCUSSION

The varicella-zoster virus is a double-stranded DNA virus.[10] Varicella (chickenpox) and zoster are different clinical conditions caused by the same virus. It spreads by direct contact with either a varicella or zoster skin lesion or by inhalation of infectious respiratory secretions from a varicella patient.[11] Zoster is rare in childhood.[12] Patients with zoster are less contagious than patients with varicella because of the absence of respiratory infection. There is a direct correlation between increasing age and the incidence of zoster, especially herpes zoster ophthalmicus, due to a decline in cell-mediated immunity.[13] Factors that increased risk of zoster include previous history of cancer, anticancer therapy, bone marrow transplantation, surgery and trauma.[14]

A prodrome of fever, malaise, headache, and dysesthesia herald the neuralgia component that precedes and eventuates in the cutaneous lesions of zoster. Spread of ganglionic infection proximally along the posterior nerve root to the meninges and spinal cord results in a localized leptomeningitis, cerebrospinal fluid pleocytosis, and segmental myelitis.

Herpes zoster ophthalmicus involves the first division of the trigeminal nerve. The skin rash may extend from the nose and eye to the vertex of the skull but does not cross the midline of the forehead. Involvement of the nasociliary portion of the ophthalmic nerve occurs in about one third of patients with herpes zoster ophthalmicus and is significantly correlated with the development of ocular complications.[2,11] The development of ocular complications, is not related to age, sex, or the severity of the skin rash. Overall, ocular involvement occurs in about 50% of patients with herpes zoster ophthalmicus. Zoster affects the second and third division of the trigeminal nerve less fre-

quently and rarely in contiguity with the first division.

The multiple and diffuse ocular complications of herpes zoster ophthalmicus are related to multiple mechanisms including presumed viral spread, nerve damage, ischemic vasculitis and an inflammatory granulomatous reaction.The complications may present acute, chronic, or relapsing.[14] Lid vesicles of zoster evolve with pitting, pigmentation,and scarring of the eyelids along the lid retraction, damage to lash roots and damage to meibomian glands. Ischemic vasculitis may lead to lid ulcers or necrosis. Orbital complications include proptosis, ptosis, chemosis, and ocular motor palsies resulting from local spread of infection within the orbit and /or brain stem or as a result of ischemic vasculitis. Conjunctival inflammation with hyperemia, petechial hemorrhage, follicular reaction, and occasionally conjunctival vesicles or membranous conjunctivitis is seen in the early stages of the disease. Inflammation of the episclera or sclera is frequently delayed for several months.

The corneal complications are frequent, resulting from a combination of factors including presumed replicating virus, limbal vasculitis, abnormal tear film, hyperesthesia and neurotropic damage, corneal exposure, and the host inflammatory and vascular response to injury.[15] Punctate epithelial keratitis and pseudodendrites develop within a few days of disease onset due to viral replication in the corneal epithelium. Later corneal complications are associated with presumed viral infection, vasculitis, immunologic mechanisms, or host inflammatory reactions. These include anterior stromal infiltrates, sclerokeratitis, keratouveitis, endotheleitis, serpiginous ulceration, and disciform keratitis. Corneal sensation is often diminished and when it persists, it leads to neurotropic keratitis. Exposure keratitis from cicatricial lid changes or the development of trichiasis may add additional insult to the cornea. Prolonged chronic disease results in corneal edema or an interstitial keratitis accompanied by vascularization , crystalline lipid deposits, stromal scarring, thinning, and even perforation

Uveitis is the most frequent finding heralding intraocular involvement. It results from presumed viral replication, ischemic vasculitis, or from lymphocytic infiltration of iris, stroma, or intraocular nerves.The disease may settle into a chronic or relapsing keratouveitis. Posterior subcapsular cataracts are seen in chronic disease from uveitis and/ or steroid use.

The vasculitis of herpes zoster may manifest in the retina and optic nerve with central retinal vein occlusion, central retinal artery occlusion, retinal vasculitis, or with ischemic optic neuropathy. Acute retinal necrosis could occur.

In herpes zoster ophthalmicus, the lesions are centered in the trigeminal ganglion but also extend into the brain-stem, orbital tissues, meninges, vessels of the brain, and ocular tissue.[16] The ocular damage presumably results from a combination of factors including viral invasion, vasculitis, and direct inflammation with involvement of posterior ciliary nerve and vessels and with a prominent ischemic necrosis.[17] With chronicity of the disease, a granulomatous reaction with epithelioid and giant cells develops within the cornea ,ciliary body, choroid, and retina in some patients. Virus had been identified at some stage in all tissues and is clearly the inciting agent in ocular disease. The end stage of inflammation is a granulomatous reaction in some tissues; the nerves develop thickening of the sheaths, fibrosis, and disappearance of ganglion cells.

MR orbital images[9] of Herpes Zoster infection include uveal-scleral thickening of the globe, ill-defined soft tissue throughout the pre- and postseptal soft tissues, and the rectus muscle and tendon enlargement. The optic nerve sheath complex showed abnormal peripheral enhancement, particularly prominent about the nerve head, with slight enhancement within the nerve head it-

self. The ophthalmic histopathologic examination showed periaxial infarction of the optic nerve and chronic inflammation in the uveal tract and vitreous and also the retinal perivasculitis in that case report of Lexa et al.

REFERENCE

1. Ragozzino MW, Melton LI III, Kurland LT Chu CP, Perry HO. Population-based study of herpes zoster and its sequalae. Medicine 1982; 61: 310-316
2. Bucci FA, Salvia PV, Mauriello JA. Herpes zoster ophthalmicus. Am Fam Physician 1987; 35:121-128
3. Harding SP, Lipton JR, Wells JC. Natural history of herpes zoster ophthalmicus: predictors of post-herpetic neuralgia and ocular involvement. Br J Ophthalmol 1987; 71:353-358
4. Carmody RF. Herpes zoster ophthalmicus complicated by ophthalmoplegia and exophthalmos. Arch Ophthalmol 1937;18: 707-711
5. Forster DJ, Dugel PU, Frangieh GT, Liggett PE, Rao NA. Rapidly progressive outer retinal necrosis in the acquired immunodeficiency syndrome. Am J Ophthalmol 1990;110:341-348
6. Amanat LA, Cant JS, Green FD. Acute phthisis bulbi and external ophthalmoplegia in herpes zoster ophthalmicus. Ann Ophthalmol 1985; 17:46-51
7. Winward KE, Hamed LM, Glaser JS. The spectrum of optic nerve disease in human immunodeficiency virus infection. Am J Ophthalmol 1989;107:373-380
8. Scharf Y, Kraus E, Zonis S. Optic neuropathy and central retinal artery occlusion in a case of herpes zoster ophthalmicus. Ann Ophthalmol 1987;19:77-78
9. Lexa FJ, Galetta SL, Yousem DM, Farber M, Oberholtzer,et al. Herpes Zoster ophthalmicus with orbital pseudotumor syndrome complicated by optic nerve infarction and cerebral granulomatous angiitis: MR-pathologic correlation. AJNR 1993;14:185-190
10. Barnes DW, Whitley RJ.CNS diseases associated with varicella zoster virus and herpes simplex virus infection: pathogenesis and current therapy. Neurol Clin 1986;4: 265-283
11. Liesegang TJ. Diagnosis and therapy of Herpes Zoster ophthalmicus. Ophthalmology 1991;98:1216-1229
12. Brunell PA, Kotchmar GS Jr. Zoster in infancy: failure to maintain virus latency following intrauterine infection. J Pediatr 1981;98:71-3
13. Miller AE. Selective decline in cellular immune response to varicella-zoster in the elderly. Neurology 1980;30:582-7
14. Cobo M, Foulks GN, Liesegang T,et al. Observations on the natural history of herpes zoster ophthalmicus. Curr Eye Res 1987;6:195-9
15. Liesegang TJ. Corneal complications from herpes zoster ophthalmicus. Ophthalmology 1985; 92: 316-24
16. Hedges TR III, albert DM. The progression of the ocular abnormalities of herpes zoster: histopathologic observations in nine cases. Ophthalmology 1982;89:165-77
17. Naumann G, Gass JDM.,Font RL. Histopathology of herpes zoster ophthalmicus. Am J Ophthalmol 1968; 65: 533-4