

Acute Orbital Myositis Heraldng Herpes Zoster Ophthalmicus: Report of a Case

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Abstract- We report a rare case that developed orbital myositis before appearance of zoster rashes. A 54 year-old man came to our hospital with a 4-day history of left orbital shooting pain extending to left temporal area. Neurological examinations demonstrated mild left proptosis and hyperemic conjunctiva without ophthalmoplegia. Brain magnetic resonance imaging (MRI) revealed left orbital myositis and periorbital skin eruptions appeared two days after this MRI study. The symptoms were improved after antiviral therapy and a follow-up MRI showed resolution of orbital myositis. Herpes zoster ophthalmicus may present as acute orbital myositis preceding skin eruptions and the recovery of orbital myositis was excellent in these patients. Our patient had postherpetic neuralgia which did not develop in previously reported cases. We conclude that herpes zoster should be listed as a cause of orbital myositis even without skin rashes.

Key Words: Orbital myositis, Herpes zoster ophthalmicus, Magnetic resonance imaging

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INTRODUCTION

In the literature, 7% of all cases of herpes zoster present as herpes zoster ophthalmicus (HZO), and 20 to 70% of HZO patients have orbital involvement⁽¹⁾. Ophthalmic complications following HZO result from inflammatory changes, nerve damage or secondary to tissue scarring. These complications vary from mild disease, which may pass unnoticed, to severe disease that threatens life or sight⁽²⁾. Nearly all orbital tissues including extra-ocular muscles can be affected by the varicella zoster virus⁽³⁾. Orbital involvement of HZO may presents as keratitis, uveitis, scleritis, optic neuritis, ocular motor

palsy and postherpetic neuralgia (PHN)⁽⁴⁾. HZO is readily suspected when ophthalmic symptoms and signs follow characteristic skin rashes and edema. Here, we report an unusual case whose orbital myositis preceded vesicular skin eruptions.

CASE REPORT

A 54 year-old man, who had liver cirrhosis for 5 years, was hospitalized on March 29, 2007 due to a 4-day history of left orbital shooting pain. This pain extended to his left temporal area and worsened with eye movements.

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The patient was afebrile. On neurological examination, he was alert and well oriented. His neck was supple. Cranial nerves examination was nearly normal except for mild proptosis and hyperemic conjunctiva on the left side. Ophthalmologic examination revealed normal intraocular pressure and normal eye ground. There was no ophthalmoplegia. Auscultation revealed no bruit over the orbits. The muscle strength and sensation were normal at four limbs. His deep tendon reflexes were brisk and plantar responses were flexor.

Laboratory data including routine biochemical and

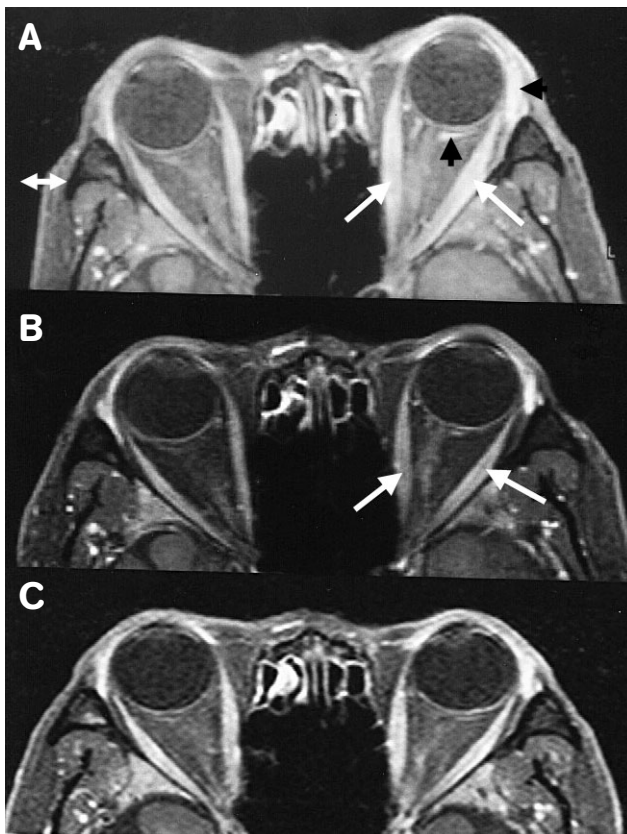


Figure 1. (A) On March 30, 2007, MRI showed relatively moderately enhanced and enlarged left medial and lateral rectus muscles (white arrows) after contrast medium injection on T1-weighted axial image. Periorbital and retrobulbar tissues were also enhanced (black arrow heads). (B) The follow-up MRI on May 10 showed symmetrical slight enhancement of the left medial and lateral rectus muscles (white arrows) after contrast medium injection on T1-weighted axial image. (C) On July 30, the third MRI demonstrated total resolution of extraocular myositis.

hematological examinations were normal except for aspartate aminotransferase (AST): 53 U/L, total bilirubin: 1.9 mg/dl, direct bilirubin: 0.8 mg/dl. On March 30, brain magnetic resonance imaging (MRI) showed smooth-bordered, fusiform enlargement of the medial and lateral rectus muscles in left orbit. These enlarged muscles were enhanced with gadolinium on T1-weighted image (Fig. 1A). Periorbital and retrobulbar tissues were also enhanced (Fig. 1B). The results of visual evoked potential (VEP) studies were normal.

The erythrocyte sedimentation rate (ESR) was 9 mm/hr, C-reactive protein (CRP) was 2.523 mg/dl. Immunologic studies, including IgA, IgM, C3, C4, anti-mitochondrial antibody (AMIA), anti-nuclear antibody (ANA) and Anti-double-stranded DNA, were all within normal limits. His thyroid function was normal and the VDRL/TPHA test was negative.

On April 1, he developed vesicular skin rashes at the distribution of ophthalmic branch of the left trigeminal nerve and along the nasal ridge (Hutchinson's sign). (Fig. 2) Immediately, we treated him with 7-day intravenous acyclovir. The headache, eyelid swelling and

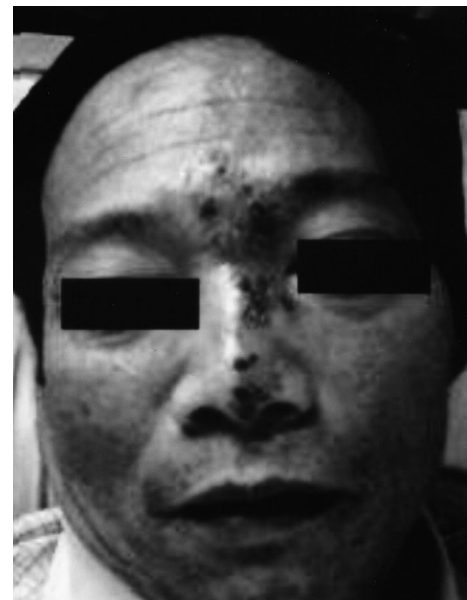


Figure 2. This patient developed vesicular skin eruptions at the territory of the ophthalmic branch of the left trigeminal nerve and along the nose ridge (Hutchinson's sign) on April 1, 2007.

orbital pain were once gradually lessened when he was discharged on April 9.

The patient returned to our clinic with persistent left orbital pain on April 16. We prescribed 7-day oral valacyclovir for him. A follow-up MRI on May 10 revealed partially resolved myositis in the left medial and lateral rectus muscles (Fig. 1B).

The third MRI on July 30 revealed total resolution of extraocular myositis (Fig. 1C). However, he suffered from PHN with paroxysmal and lancinating pain along the nose ridge in these four months.

DISCUSSION

Except for diplopia, our patient developed characteristic symptoms of orbital myositis, such as orbital pain worsening with eye movements, proptosis, swollen eyelid and hyperemic conjunctiva⁽⁵⁾. He had Hutchinson's sign which is typical for HZO⁽¹⁾. Normal immunologic and serologic surveys excluded other etiologies of orbital myositis, for example, thyroid disease, syphilis and auto-immune diseases. His ocular symptoms did improve after antiviral therapy and follow-up orbital MRI four months later revealed total recovery of orbital myositis.

Typically, ophthalmic complications of HZO occur between 5 days and 14 days following cutaneous lesions⁽²⁾. Our case is unusual in that the orbital myositis precedes vesicular rashes.

Two similar patients had been reported previously^(6,7). Both patients came to the hospital for retrobulbar pain with diplopia. Volpe et al. demonstrated orbital myositis on computed tomographic (CT) scan in their patient one day before the development of skin vesicles⁽⁶⁾. As reported by Kawasaki et al., MRI demonstrated orbital myositis three days prior to appearance of typical skin eruptions in their patient⁽⁷⁾. In these two patients and ours, their extraocular myositis has excellent recovery. But our patient suffered from PHN, which did not occur in the previously reported patients.

Although Marsh and Cooper had proposed extraocular myositis as a possible cause of ophthalmoplegia in HZO⁽⁴⁾, it is not well documented until these case reports.

To date, there is no histopathologic study of orbital myositis in HZO⁽⁷⁾.

Orbital myositis preceding vesicular skin eruptions is a diagnostic challenge in HZO. Since zoster rashes may develop one week or more after dermatomal pain⁽¹⁾, serological and immunological tests may be helpful for early diagnosis in extraocular myositis preceding zoster rashes.

In 1958, Lewis reported a syndrome of "ophthalmic zoster sine herpette"⁽⁸⁾, in which orbital pain, extraocular palsy and periorbital skin swelling occurred without skin rashes. "Ophthalmic zoster sine herpette" further confounds the diagnosis of HZO. We emphasize that, even without vesicular skin rashes, the diagnosis of extraocular myositis case as idiopathic should be reserved before the availability of negative serological and immunological results for herpes zoster.

In conclusion, orbital myositis can be the presenting sign of HZO. In these patients, recovery of extraocular myositis is excellent and serological and immunological studies maybe helpful for early diagnosis. HZO should be listed as a cause of acute orbital myositis even without skin eruptions.

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