

Orbital Cellulitis Presenting as Giant Cell Arteritis: A Case Report

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Abstract-

Purpose: To present a case of orbital cellulitis initially mimicking giant cell arteritis.

Case report: An 80-year-old man with a history of hypertension and type 2 diabetes mellitus was referred with a prominent progressive headache over the right temporal and periorbital areas. Non-contrast brain CT results were normal, but ESR was elevated. Giant cell arteritis was suspected initially. However, the symptoms progressed under oral corticosteroid therapy. The subsequent brain MRI with contrast revealed extensive contrast enhancement along the right optic nerve and optic canal with a rim-enhancing lesion in the posterior aspect of the optic nerve. Treatment included intravenous antibiotics and surgical drainage. Culture of the drainage revealed growth of *Pseudomonas aeruginosa*.

Conclusion: Orbital cellulitis should be considered in patients with progressive headache over the unilateral temporal and periorbital areas, in addition to giant cell arteritis. Brain imaging with contrast should be performed for detecting occult orbital infection or other intracranial etiologies.

Key Words: Orbital cellulitis; Giant cell arteritis; Temporal arteritis; Secondary headache.

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INTRODUCTION

Orbital cellulitis is a potentially life-threatening condition most commonly caused by bacterial infection of the post-septal orbital region. Orbital cellulitis can manifest in several ways because of the multiple types of tissue posterior to the orbital septum, and an infection can affect orbital soft tissues including muscle and fat, nerves, and bones. The consequences of misdiagnosis and inadequate treatment can be disastrous. Treatment of orbital cellulitis requires hospital admission and

administration of broad-spectrum antibiotics. Surgical drainage may be necessary in critical cases and in cases with abscess formation ⁽¹⁾.

In patients with headache as the main complaint, orbital cellulitis may be initially misdiagnosed as headache with other etiologies such as autoimmunity or primary headache. Occasionally, when elevated erythrocyte sedimentation rate (ESR) and serum C-reactive protein (CRP) level are concurrently found, a diagnosis of giant cell arteritis (GCA) may be made.

We present a case of new-onset headache with high

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ESR initially suspected as being GCA. However, the symptoms worsened after temporarily improving under oral corticosteroid therapy.

CASE REPORT

An 80-year-old man has hypertension and type 2 diabetes mellitus presented at the outpatient department due to a new-onset progressive headache over the right temporal and periorbital areas. There was no body weight loss or fever before this visiting, and his most recent glycated hemoglobin (HbA1c) was 8.1%. He had taken some analgesics but in vain. Physical examinations disclosed normal appearance without ecchymosis, swelling, or open wound of the head. Tenderness was noted when the temporal and periorbital areas were touched. Jaw claudication was noted. Non-contrast brain computed tomography (CT) scan was performed to exclude organic intracranial lesion or other periorbital lesions that may cause secondary headache, but revealed no suggestive findings. A high ESR (50 mm/hr) and mildly elevated CRP (0.99 mg/dL) were observed because of which GCA was suspected and the patient was administered prednisolone 15 mg twice daily for one week, which was then tapered to 10 mg twice daily due to subsiding of the headache and lowering of ESR (27 mm/hr) and CRP (0.6 mg/dL) in the second week. The jaw claudication also subsided.

However, sudden loss of vision of the right eye developed after the second week and the patient was sent to our emergent department. As deterioration of cranial vasculitis was suspected, the dosage of prednisolone was increased to 15 mg twice daily. A biopsy showed negative findings for GCA. The ophthalmologist was consulted for further evaluation but no remarkable findings emerged except for limited eye movements in all directions and ptosis over the right eye. Brain magnetic resonance imaging (MRI) with contrast was performed for further evaluation.

The patient's brain MRI with contrast disclosed extensive contrast enhancement along the right optic nerve and optic canal with a rim-enhancing lesion in the posterior aspect of the optic nerve, measuring 1 cm in diameter. Extensive contrast enhancement was also noted in the right inferior fissure, pterygopalatine fossa, cavernous sinus, posterior aspect of the conal and

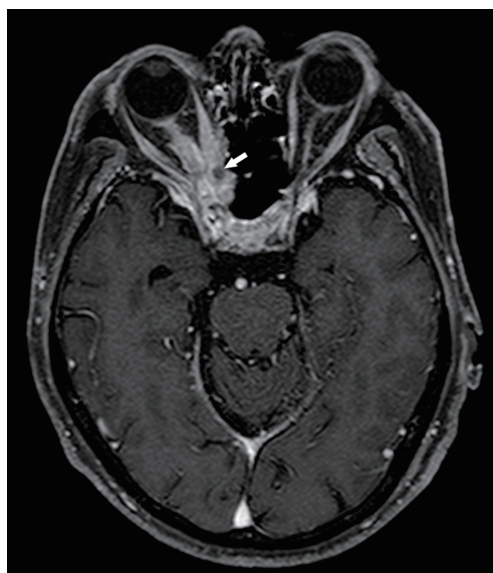


Figure 1. The brain MRI with contrast disclosed extensive contrast enhancement along the right optic nerve, optic canal, and cavernous sinus with a rim-enhancing lesion (arrow) in the posterior aspect of the optic nerve.

extraconal spaces of the right orbit, right masticator space, and right ethmoid and maxillary sinuses, which indicated extensive infection with abscess formation (Figure 1).

The patient was hospitalized and treated with intravenous antibiotics and underwent surgical drainage. The culture of the drainage revealed growth of *Pseudomonas aeruginosa*. However, the patient eventually experienced permanent visual loss after treatment without recurrence of headache.

DISCUSSION

Orbital cellulitis is more common in children than in adults. Rhinosinusitis is the source of most cases; coexisting rhinosinusitis is present in 86–98% of cases of orbital cellulitis, and the most commonly identified pathogens are *Staphylococcus aureus* and streptococci^(2,3). *Pseudomonas aeruginosa* is an uncommon cause that has also been reported⁽⁴⁾. Orbital cellulitis can cause swelling and inflammation of the extraocular muscles and fatty tissues within the orbit, leading to headache in the orbital area and pain with eye movements. Ptosis, proptosis,

eyelid erythema, and ophthalmoplegia with diplopia may also present. In rare cases, there may be visual impairment and even blindness arising from inflammation or ischemia of the optic nerve ⁽⁵⁾.

The diagnosis of orbital cellulitis once suspected clinically can be confirmed by CT or MRI. Ophthalmological examination is also important in order to evaluate the extraocular movements and visual acuity. However, some cases of orbital cellulitis may only have subtle abnormalities or even show normal results for CT or MRI.

The differential diagnosis of orbital cellulitis includes a range of infectious and noninfectious diseases. Importantly, some autoimmune vasculitis such as GCA, Wegener granulomatosis, polyarteritis nodosa, and Takayasu arteritis might present with clinical symptoms mimicking infectious disorders. GCA is categorized as a chronic systemic inflammatory necrotizing granulomatous vasculitis of large- and medium-sized vessels containing an elastic lamina ⁽⁶⁾ and is one of the most common mimickers of orbital cellulitis. The greatest risk factor for developing GCA is aging, and females are affected more frequently than males ⁽⁷⁾. The diagnosis of GCA should be considered in patients who are older than 50 and have symptoms including new onset of headache, abrupt onset of visual disturbances, jaw claudication, and high ESR or serum CRP level ⁽⁸⁾. Visual loss is the most feared complication of GCA when involving cranial arteries. A brain imaging study such as CT or MRI and subsequent temporal artery biopsy should be performed in all patients suspected of having GCA to rule out cysts, tumors, bleeding, and other organic brain lesions. Conventional angiography and color doppler sonography (CDS) also help to confirm the diagnosis of GCA and to evaluate large vessel involvement in GCA and in other forms of vasculitis, including Takayasu arteritis, a disease closely related to GCA. With CDS, the assessment should include the whole length of bilateral temporal and axillary arteries thus extra-cranial artery involvement could be detected ⁽⁹⁾. The presence of halo sign in temporal arteries with a positive compression test indicates vessel edema that is sensitive for GCS and might have a higher sensitivity compared with temporal artery biopsy ⁽⁹⁾. The universally accepted treatment of GCA is high-dose corticosteroid therapy ⁽⁸⁾.

Our report illustrates the difficulties of managing

patients with presumed GCA with negative findings on initial brain imaging and lack of histologic confirmation. Although improvement of symptoms with corticosteroid therapy is a hallmark in patients with GCA, this treatment had disastrous consequences in our patient and prevented early identification of orbital cellulitis. Furthermore, in our case, the pathogen was *Pseudomonas aeruginosa* (PA). It is a rare cause of orbital cellulitis but is a common organism in some diabetic related infections, such as diabetic foot infections ⁽¹⁰⁾; however, the incidence of PA related orbital cellulitis in diabetic patient has not established yet. Furthermore, combined antibiotic therapy is recommended for higher rate of multidrug resistance in PA cellulitis. ⁽¹⁰⁾. Therefore, in patients with classic clinical presentation of GCA, an initial non-contrast brain CT may be insufficient; with negative findings on initial brain imaging and lack of histologic confirmation, high-dose corticosteroid treatment should be accompanied by careful monitoring of the ESR and clinical symptoms. A contrast-enhanced CT or MRI is warrant in high-risk patients. Also, in our case, the patient was male gender, and GCA less frequent affect men then women as mentioned above. The diagnosis should be made more cautiously in men with GCS and other acquired inflammatory or infectious disorders must be considered. A lack of efficacy with corticosteroid therapy should prompt critical reevaluation of the original diagnosis. A follow-up brain MRI with contrast should be performed for detecting occult orbital infection or other intracranial etiologies.

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