Orbital Apex Syndrome in an Uncontrolled Diabetes Mellitus Patient: Case Report

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Abstract

Orbital apex syndrome (OAS) is a complex neurological disorder characterized by a constellation of multiple dysfunctions of cranial nerves II, III, IV, V_1, and VI, typically resulting from pathologies affecting adjacent structures, such as the orbit and paranasal sinuses. Early recognition and appropriate treatment are essential due to its potentially rapid progression and risk of permanent vision loss. Case Report**:** A 63-year-old male with uncontrolled diabetes mellitus, hypertension, and gout arthritis who presented with left periorbital pain, swelling, redness, followed by vision loss and ophthalmoplegia. Clinical examination showed no light perception, severe ptosis, proptosis, decreased corneal sensation, and a relative afferent pupillary defect (RAPD) in the left eye. Laboratory investigations showed leukocytosis and an elevated HbA1c (12%). A brain CT scan demonstrated opacification of the left ethmoid and sphenoid sinuses and enhancement at the left orbital apex. Based on clinical, radiologic, and laboratory findings, the patient was diagnosed with orbital apex syndrome secondary to ethmoid and sphenoid sinusitis. The clinical presentation and imaging findings indicated orbital apex syndrome resulting from the contiguous spread of bacterial sinusitis in an immunocompromised host, such as those with uncontrolled diabetes. Uncontrolled diabetes likely contributed to the rapid progression. Early diagnosis and identification of the underlying causes are essential for initiating targeted therapy, including antibiotics and supportive care. OAS secondary to sinusitis should be considered in patients presenting with painful ophthalmoplegia and visual impairment, particularly those with poorly controlled diabetes. Early recognition and intervention are crucial for improving outcomes.

Keywords: orbital apex syndrome, ophthalmoplegia, sinusitis, cranial neuropathy, vision loss

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Introduction

Orbital apex syndrome (OAS) is a complex neurological disorder characterized by a constellation of signs resulting from multiple cranial nerve involvement, including the Optic Nerve (II), Oculomotor nerve (III), Trochlear nerve (IV), Abducens nerve (VI), and the first division of the trigeminal nerve (ophthalmic division) (V_1)[1]. The causes of orbital apex syndrome include infectious, inflammatory, traumatic, iatrogenic, hormonal, and neoplastic pathologies. They primarily involve one of the adjacent structures, like the paranasal sinuses or the orbit, from which they spread to the orbital apex.[2] Establishing the underlying etiology of this syndrome is particularly important when considering some specific treatments[1].

We present a case of orbital apex syndrome in a patient with uncontrolled diabetes mellitus, secondary to sphenoid and ethmoid sinusitis to highlight the clinical presentation and diagnostic process.

Case Illustration

A 63-year-old male was referred from the Internal Medicine Ward Department at Abdoel Moeloek Hospital with complaints of swelling and pain around the left eye for one week. These symptoms were followed by decreased vision, redness, and drooping of the upper eyelid in the left eye. The patient reported pain in the upper left molars over the past two months, which had worsened over the previous week and was accompanied by swelling in the left cheek extending to the periorbital region and fever for a couple of days. There was no history of trauma or ocular surgery. The right eye was unaffected, and there were no similar complaints in the past.

The patient had a medical history of uncontrolled diabetes mellitus, hypertension, and gout arthritis. He had

been receiving treatments from both a dentist and an internist. After several days of hospitalization, the facial swelling improved, but ocular symptoms in the left eye persisted.

On ophthalmological examination, visual acuity on the left eye was no light perception (NLP) with normal intraocular pressure (IOP) at 12.1 mmHg. There was periorbital edema, severe ptosis, and proptosis of the left eye (Figure 1). Corneal sensations were decreased in the left eye. Relative afferent pupillary defect (RAPD) was present in the left eye. The extraocular movements of the left eye were limited in all directions (Figure 2). Fundoscopy revealed atrophy of the optic nerve.

Figure 1. Severe ptosis in the left eye

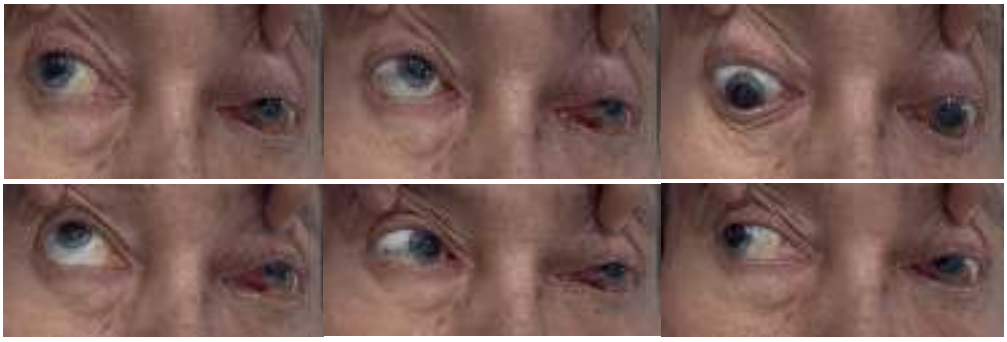

Figure 2. Restricted extraocular movements in all directions of the left eye

Laboratory findings revealed leukocytosis (WBC 20.610/µL), anemia (Hb 10 g/dL), and an HbA1c level of 12%, indicating poor glycemic control. After several days of hospitalization, the patient underwent a brain Computerized Tomography (CT) scan, which showed abnormal enhancement in the left orbital apex and opacification of the left sphenoid and ethmoid sinuses (Figure 3). Based on clinical features, imaging, and laboratory findings, the patient was diagnosed with orbital apex syndrome of the left eye, secondary to ethmoid and sphenoid sinusitis.

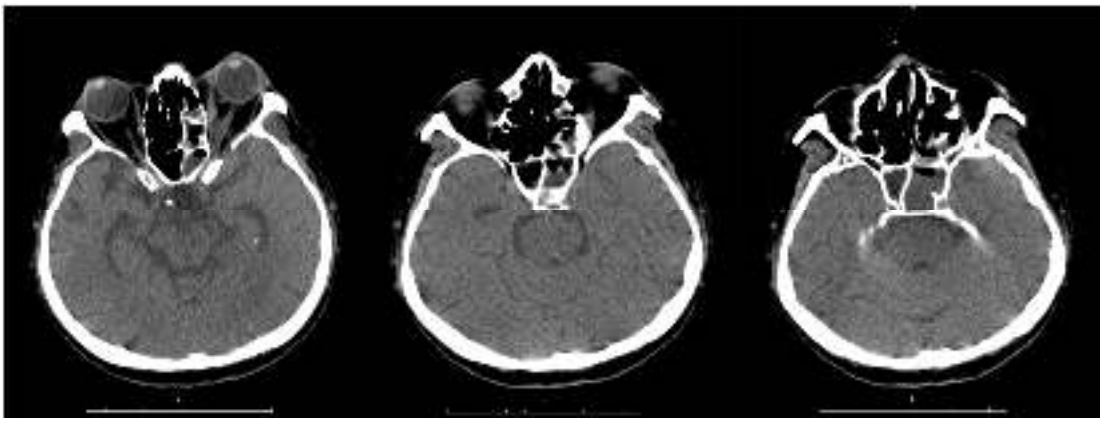

Figure 3. A brain CT scan showed abnormal enhancement in the left orbital apex and opacification of the left sphenoid and ethmoid sinus

Discussion

The orbital apex is an opening connecting the orbit and the cranial cavity that consists of the optic canal, the superior orbital fissure, and the inferior orbital fissure[3,4]. The roof of the orbital apex is formed by the lesser wing of the sphenoid, the lateral wall by the greater wing of the sphenoid, the medial wall by the ethmoidal sinus, and the floor by the orbital plate of the palatine bone[5]. The optic canal is bordered by the sphenoid bone, superior by the lesser wing, inferolateral by the optic strut, and medial by the body[3].

Orbital apex syndrome (OAS) is defined as a constellation of signs and symptoms resulting from pathology at the orbital apex, affecting cranial nerves II (optic), III (oculomotor), IV (trochlear), V_1 (ophthalmic branch of the trigeminal), and VI (abducens) which can be caused by infections, inflammation, trauma, neoplasia, etc[6]. Clinical manifestations typically include proptosis, defective vision, a relative afferent pupillary defect (RAPD) due to involvement of the optic nerve, restricted ocular movement due to the involvement of the oculomotor, trochlear, and abducens nerve, facial pain and paresthesia over the forehead and the upper lid due to the involvement of the ophthalmic division of trigeminal nerve, and anisocoria due to the participation of the pupillary fibers[7]. Due to the proximity of the orbital apex to the paranasal sinuses, infections such as sinusitis can readily spread contiguously, especially from the ethmoid and sphenoid sinuses[8].

In the case described, the patient had uncontrolled diabetes, elevated white blood count, fever and rapid progression of symptoms with cranial nerves II to VI involvement, including ptosis, positive RAPD, restriction of eye movements in all directions, positive RAPD, limitation of eye movements in all directions, and vision loss. Fundoscopy revealed atrophy of the optic nerve. A Brain CT scan showed abnormal enhancement in the left orbital apex, and opacification in the left sphenoid and ethmoid sinus. This finding could be suggestive of sinusitis that leads to orbital apex syndrome.

OAS can be triggered by a wide range of infectious organisms, including viral, bacterial, fungal, and parasitic infections. These organisms primarily involve the surrounding structures such as the orbit or the paranasal sinuses, from which they spread contiguously to the orbital apex, resulting in the typical clinical features. Gram-positive cocci, such as *Staphylococcus* and *Streptococcus pneumoniae,* and gram-negative bacilli,

including Pseudomonas, Klebsiella, Proteus , and anaerobic bacteria, can cause sinusitis or orbital cellulitis that spreads to involve the orbital apex[8]. The susceptible group of patients includes those with immunocompromised conditions and individuals with uncontrolled hyperglycemia. The dissemination of infection may be facilitated by microbial toxins and endotoxins, such as lipopolysaccharides, which degrade tissue barriers and promote the spread[9]. Hence, in this case, we attributed the patient's orbital apex syndrome to bacterial sphenoid and ethmoid sinusitis. Especially, our patient has uncontrolled diabetes that induced an immunocompromised state, which would have probably contributed to the rapid progression of the infection from the sinus to the orbital apex.

Diagnosis of OAS relies on clinical features and various tests. The tests include laboratory evaluations (complete blood count, peripheral smear, erythrocyte sedimentation rate, C-reactive protein, Gram stain and culture on blood agar, etc) and radiological investigations (CT scan and MRI)[6]. A Brain CT scan may help in imaging the paranasal sinuses to look for features of sinusitis, pre-existing orbital cellulitis, or a subperiosteal abscess. At the same time, MRI shows hypointense signals in T1-weighted images and hyperintense signals in T2-weighted images. It has a cone shape when the infection is localized to the orbital apex and a dumbbell shape when the superior orbital fissure is involved[10].

Management of OAS depends on the underlying etiology. Bacterial infections respond with antibiotics[11]. Fungal infections, such as mucormycosis, are usually treated with an intravenous infusion of liposomal amphotericin B, a broad-spectrum antifungal agent, and aspergillosis is usually treated with intravenous voriconazole[12]. Virus infections respond with combined antiviral and steroid therapy[13]. Parasite infections respond with combined anti-helminthic agents and steroids to avoid adverse reactions due to intense inflammation caused by the death of the cestode[14]. Inflammatory causes are usually managed by systemic steroid therapy[15]. In the case of orbital apex syndrome secondary to trauma, surgery is required in appropriate clinical settings, and high-dose systemic steroids may also be necessary to reduce inflammatory soft tissue edema and hematoma[16].

Conclusion

Orbital apex syndrome (OAS) is a rare but serious condition that results from the involvement of multiple cranial nerves at the orbital apex, often due to infections, inflammatory, traumatic, or neoplastic causes. In this case, the patient presented with classic signs of OAS—including vision loss, ptosis, ophthalmoplegia, and reduced corneal sensation—alongside evidence of uncontrolled diabetes and sinusitis. Imaging revealed opacification of the left ethmoid and sphenoid sinuses with enhancement at the left orbital apex, consistent with infectious spread. The patient's immunocompromised state likely facilitated the rapid progression of bacterial sinusitis into OAS.

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