



Orbital Apex Syndrome - A Case Report

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Abstract

Orbital apex syndrome is characterized by vision loss and ophthalmoplegia due to the involvement of the orbital apex. The signs and symptoms vary depending upon the involvement of the structures within the orbital apex, the superior orbital fissure or the cavernous sinus. Clinical evaluation is the key to the diagnosis which is aided by neuro-imaging modalities including brain and orbital Magnetic Resonance Imaging and Computed Tomography scans. In rare instances, a biopsy may be needed to aid in diagnosis. Treatment depends on what the nature of the lesion.

Keywords:- Orbital Apex Syndrome, Ophthalmoplegia, Cranial Nerves.

INTRODUCTION

“Orbital apex syndrome” - refers to a constellation of symptoms and signs which result from the involvement of various structures in the region of the orbital apex. These structures include the four rectus muscles taking their origin from the annulus of Zinn, the optic nerve and ophthalmic artery. Orbital Apex syndrome constitutes a syndrome characterized by involvement of the following cranial nerves i.e., Optic nerve, Oculomotor nerve, Trochlear nerve, Abducens nerve, the first division of the trigeminal nerve (ophthalmic division).^[1] Due to anatomical proximity, two other syndromes that can have overlapping features are the Superior orbital fissure syndrome and the cavernous sinus thrombosis. In cavernous sinus

syndrome, clinical features result from the combination of orbital apex syndrome and involvement of the sympathetic fibers and the maxillary division of the trigeminal nerve. The superior orbital fissure syndrome is caused by lesions anterior to the orbital apex. It is characterized by multiple cranial nerve involvement but generally spares the optic nerve.

CASE REPORT



Figure 1: Showing severe ptosis in the left eye with frontalis overaction.

A 56-year-old female patient presented with complaints of swelling and pain around the left eye from three days. These symptoms were followed by diminution of vision and dropping of the upper lid in the left eye. There was history of headache from the last one week which was severe in intensity. There was no history of fever, trauma, ocular surgery. Patient was not a known case of diabetes, hypertension or any other systemic disease. There was no history of similar complaints in the past. There was no history of any ocular complaint in the right eye. On Ophthalmological examination, visual acuity was 6/6 in the right eye and hand movement close to the face in the left eye. The colour vision was normal in the right eye while it could not be assessed in the left eye due to low vision. On pupil examination, Relative afferent pupillary defect was present in the left eye. There was severe ptosis [Figure 1] with wrinkling of the ipsilateral forehead and elevated left eyebrow suggesting frontalis overaction with proptosis in the left eye. The extraocular movements of the left eye were restricted [Figure 2]. Corneal sensations were decreased in the left eye. Hyperemic disc was

documented in the left eye on fundus examination. Diagnosis of orbital apex syndrome was made clinically and patient was started on injectable antibiotics. Blood investigation showed raised total leucocyte count ($15000/\text{mm}^3$) with predominant leucocytosis and raised ESR (Erythrocyte sedimentation rate) i.e. $21\text{mm}/1^{\text{st}} \text{hr}$ suggesting infective etiology. Urgent MRI (magnetic resonance imaging) brain and orbit was done which showed sphenoid sinusitis. Thus, it was a case of orbital apex syndrome secondary to sphenoid sinusitis. Endoscopic transnasal sphenoidotomy was done. Within one week of starting treatment, patient showed marked improvement in symptoms but vision loss persisted.



Figure 2: Showing restricted extraocular movements of the left eye.

DISCUSSION

Orbital apex syndrome is a rare presentation which can be due to infections, inflammation, trauma, neoplasia etc.^[2] It is defined as collection of signs involving multiple cranial nerves i.e. optic nerve, oculomotor, trochlear, abducens nerve and the ophthalmic branch of the trigeminal nerve. Thus, it can lead to ophthalmoplegia and patients can present with vertical, horizontal or torsional diplopia with or without compensatory abnormal head postures. Other clinical signs are Proptosis, Absence of corneal sensations, Pupillary abnormalities, Optic disc edema and Choroidal folds.^[3] Differentiation between cavernous



sinus syndrome and orbital apex might be difficult but the main difference is the optic nerve involvement in orbital apex syndrome. To differentiate among these is important because the etiologies might be significantly different. Treatment depends upon the etiology. Infectious causes respond to antimicrobials and surgical debridement while systemic steroids are prescribed in inflammatory causes. Similarly neoplastic causes are resected surgically along with chemotherapy or radiotherapy. Although vision loss remained permanent in our case, prompt initiation of broad-spectrum antibiotics and antifungals prevented intracranial extension of infection.

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CONCLUSIONS

Orbital apex syndrome is a rare presentation but overlapping signs and symptoms with cavernous sinus thrombosis and superior orbital fissure syndrome. The etiology of the orbital apex syndrome determine the treatment. Infective cause, as in our case can involve intracranial structure and ultimately leading to mortality. Early treatment in these patients can save the life of the patient though improvement in the vision is rare despite treating the cause.