

Sudden Visual Loss in a Diabetic Patient

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ABSTRACT

We report a case of sudden monocular visual loss with drooping of upper eyelid in a 65 year old diabetic patient. Examination revealed complete ophthalmoplegia with optic neuropathy and paranasal sinusitis. Early diagnosis and prompt management with radical debridement of sinuses along with antifungal therapy helped in preventing further complications.

KEY WORDS: mucormycosis; ophthalmoplegia; optic neuropathy; orbital apex syndrome; sinusitis; visual loss

INTRODUCTION:

Orbital involvement in sinusitis is a well-recognized entity. An infection from sinuses can easily spread to the orbit, either by direct extension through the bone or indirectly through valve less venous plexus surrounding the orbit and the sinuses^[1]. Orbital apex syndrome (OAS) is a rare form of complication that classically presents with visual loss and ophthalmoplegia, but with minimal or no signs of orbital inflammation. The infection starts in the paranasal sinuses and extends into the orbital apex resulting in blindness. We present a case of acute fungal sinusitis that was complicated by monocular and irreversible visual loss with ophthalmoplegia in an elderly diabetic patient.

CASE REPORT:

A 65 year old man farmer by occupation, presented with periocular pain, swelling over left eye, loss of vision and drooping of upper eyelid since one week. He was on oral steroids for chronic dermatitis for past 3 years. He developed diabetes mellitus 4 month back and was put on oral antidiabetic drugs but he discontinued taking oral antidiabetic treatment 15 days prior to presentation.

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Examination of left eye revealed periorbital swelling; complete Ptosis with III, IV, VI cranial nerve palsy (Figure 1). Perception of light was not present and pupil was dilated and fixed. Right eye vision was 20/20 and examination was within normal limits. Nasal examination showed blackish deposits on left side near middle meatus.



Figure 1: Clinical photograph of the patient showing complete ophthalmoplegia.

Laboratory investigation revealed leukocytosis (17000cells/mcL) with high polymorph count (85%). His blood sugar was high. Fasting blood glucose was 168 mg/dL and post prandial blood glucose was 240mg/dL. Therefore he was put on systemic antibiotics and insulin therapy. KOH preparation of nasal swab was sent for direct microscopy and nasal swab was inoculated on sabourads dextrose agar. CT Scan of orbit and Para nasal sinuses was also done.

KOH preparation of nasal swab showed aseptate fungal hyphae with right angled branching. (Figure 2) Culture on sabourads dextrose agar showed dense cottony fluffy growth. Lacto phenol cotton blue mount revealed broad, aseptate hyphae with rhizoids and sporangia suggestive of *Rhizopus microsporus*

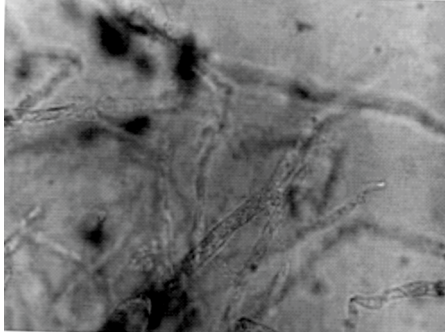


Figure 2: Aseptate hyphae in KOH mount.

species.

Plain CT scan of the paranasal sinuses and orbits showed increased soft tissue density in maxillary, anterior and posterior ethmoid air cells. Sagittal view showed increased thickness of optic nerve sheath complex with increased density at orbital apex and in cavernous sinus area. (Figure 3a,b)

On the basis of CT Scan findings and laboratory evidence, diagnosis of acute invasive mucormycosis with orbital apex syndrome was made. The patient was put on intravenous amphotericin B (0.3 mg/kg of body weight). Endoscopic debridement of the maxillary and ethmoid sinuses was done Patient's general wellbeing improved, the periorbital swelling subsided, but the patient was left with blindness in the left eye due to optic neuropathy.



Figure 3: CT Orbit showing increased thickness of optic nerve sheath complex with irregular margins, opacity in ethmoid and maxillary sinus on left side and heterogeneous soft tissue density at orbital apex and cavernous sinus area.

DISCUSSION:

Isolated cranial nerve palsies are common in patients with diabetes mellitus, but multiple simultaneous cranial nerve palsy with visual loss is rare. The differential diagnosis includes diabetic polyneuropathy, cavernous sinus thrombosis, Rhino orbital mucormycosis and the Tolosa Hunt syndrome^[2].

Visual loss due to optic neuropathy and ophthalmoplegia involving III, IV, VI, and V1 are the

hallmarks of an orbital apex syndrome (OAS). The cavernous sinus syndrome (CSS) may include the features of an OAS along with involvement of the maxillary branch of the trigeminal nerve (V2) and oculosympathetic fibers. Cavernous sinus lesions are also more commonly bilateral.

Orbital apex syndromes may result from a variety of inflammatory, infectious, neoplastic, and vascular conditions^[3]. A detailed history, laboratory investigations and imaging helps in narrowing the differential diagnosis.

Rhinorbital mucormycosis is a rare but potentially aggressive and fatal fungal infection. It usually starts in the nasal or oral mucosa after inhalation of fungal spores, then it rapidly spreads to the paranasal sinuses, and enters the orbit via the angular vein, lacrimal and ethmoid vessels as well as by direct extension from sinuses^[4,5].

Pathologically, mucormycosis is characterized by vascular invasion with fungal hyphae, infarction and necrosis of tissue^[6]. The predisposing factors for mucormycosis include poorly-controlled diabetes mellitus, alcoholism, prolonged corticosteroid treatment and immunosuppression. The diagnosis is confirmed by demonstrating tissue invasion and subsequent tissue reaction to the fungi, rather than just the presence of the organism^[7]. The current treatment strategy involves rapid diagnosis, treatment of underlying medical conditions, systemic antifungal therapy and surgical debridement of sinuses when needed^[8].

CONCLUSION:

In orbital apex syndrome prognosis is good with control of predisposing factors, aggressive treatment by radical debridement of sinuses and antifungal therapy. Early diagnosis and aggressive management is important to prevent blindness and intracranial complications.

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