

Misdiagnosed Case Of Aspergillosis

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Citation

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Abstract

A young male patient presented with total loss of vision in left eye, since he had alcoholic liver cirrhosis and normal MRI, a diagnosis of Toxic amblyopia was made and patient was given steroids. He later developed proptosis and retrobulbar mass in left orbit, biopsy ruled out malignancy but by the time repeat biopsies and radiological investigations proved it to be aspergillosis, the patient died. An early and accurate diagnosis followed by required management could have saved his life.

Thirty two year old male patient came with chief complaints of bulging of Left Eye (Figure 1 & 2). It was not associated with any aggravating or relieving factors. He was unable to eat since five days and had loss of bladder control since two days. There was generalized deterioration in his systemic condition since one week.

Figure 1



Figure 2



Patient had presented with no perception of light six months

back and was diagnosed with optic atrophy of the Left Eye (Toxic amblyopia) secondary to alcoholic liver disease. Patient had no nasal discharge or obstruction. MRI orbit and brain report at that time was normal. The patient was given Intravenous Methyl Prednisolone and then short course of oral steroids. Due to lack of response to Methyl Prednisolone there was a high suspicion of a compressive lesion in its early stage hence the patient was referred to a tertiary care Ophthalmic Centre. Unfortunately the patient did not follow instructions and failed to follow-up. He went to his local hospital where he was started on Anti Koch's treatment.

Three months later patient developed axial proptosis and ophthalmoplegia. A CT scan was advised by an ophthalmologist at a hospital near his home. The report showed a fusiform swelling around the left optic nerve just distal to optic foramen. The lesion was also invading left cavernous sinus. A transeptal transphenoidal biopsy was taken which showed an inflammatory lesion with no malignant cells. Repeat biopsy was done which showed Candida elements and no malignant cells. Patient's proptosis decreased after the biopsy. However he did not take any treatment. As his condition deteriorated after 15 days he reported back to us.

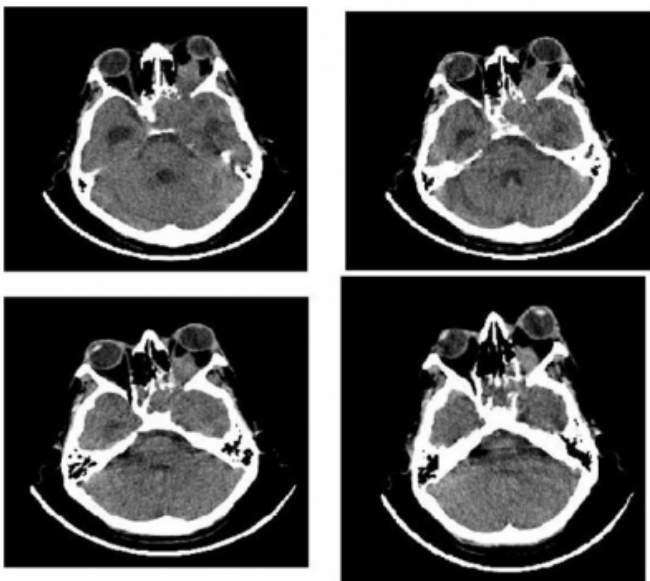
Patient was a known case of alcoholic liver cirrhosis. He was HBsAg positive. He had undergone band ligation for oesophageal varices eight months back.

Patient was incoherent and disoriented with generalised weakness in whole body. On ocular examination, in right eye the extra ocular movements were normal, pupils were 2mm sluggishly reacting to light, fundus was normal with

0.3 cupping and normal disc margins. In left eye there was total ophthalmoplegia with severe proptosis, conjunctival congestion, corneal abscess with central thinning and rest details were not seen. On MRI (plain MR of the brain using T1 T2 and flair sequences) a heterogeneously enhancing infiltrative mass lesion in the left orbital apex which extended into ethmoidal and sphenoidal sinuses, cavernous sinus, pituitary fossa and anterior left cerebral hemisphere was seen. Patient was admitted in intensive care unit and was started on antibiotic and antiepileptic. On high suspicion of fungal infection Amphotericin Inj. 3mg/kg/day was started.

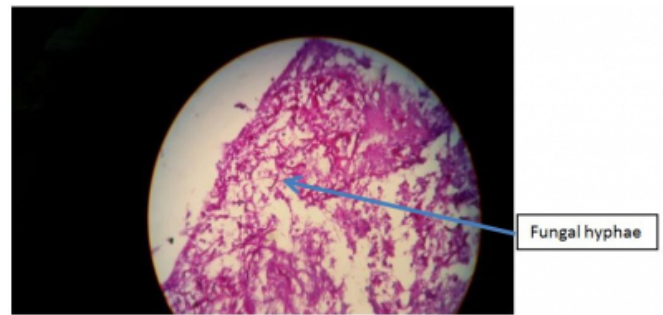
CT paranasal sinuses (axial and coronal) plain and contrast study was done and it showed diffusely infiltrating mass involving left optic nerve, medial, lateral, superior and inferior recti, left temporal lobe and pituitary fossa. The mass was heterogeneously enhancing destroying the optic foramina, left lamina papyracea, left lateral wall of pituitary fossa and sphenoid sinus. There was an infarct in left anterior cerebral artery territory with an extra ventricular communicating hydrocephalus (Figure 3).

Figure 3



Total WBC count was 12500. Left ethmoidal sinus biopsy was taken and was sent for histopathology. But before the report was generated patient had cardio-respiratory arrest. Histopathology report showed aspergillus fumigatus infection (Figure 4).

Figure 4



DISCUSSION

Orbital aspergillosis is a rare yet potentially fatal clinical entity in immunocompetent patients.¹ In the review of literature we could find only 30 cases reported from India over 15 years.²⁻⁵ The close proximity between the orbit and the middle cranial fossa provides a direct pathway for intracranial extension of the disease; the mortality rate can be over 50%.¹ It is also frequently related to immunocompromised conditions such as intravenous drug addiction, HIV infection, malignancy and the use of systemic immunosuppressive agents.⁶ Orbital aspergillosis can also mimic other conditions including bacterial orbital cellulitis, idiopathic orbital inflammatory syndrome and neoplasia.⁷

The patient generally presents with persistent retrobulbar pain and headache that precedes the ophthalmic findings of decreased vision, proptosis and ophthalmoplegia.² Pain is consistent with a lesion arising in the sphenoid sinus, which is commonly the site of origin. Our patient however had no pain and presented with total loss of vision four months before all other ocular symptoms and was then diagnosed as toxic amblyopia as he was a known case of alcoholic liver disease.

Reported cases required more than one biopsy to make the tissue diagnosis due to the difficulty in surgical access thus making the diagnosis difficult.⁸

Histopathologically aspergillosis contains necrotic tissue with occasional histiocytic giant cells. The identification of fungal hyphae in this type of necrotic tissue may be difficult due to degeneration and ballooning of the hyphae. The two main primary fungal infections affecting the region of the nasal sinuses and brain are Aspergillus and Zygomycosis.^{9,10}

Management often begins with surgical debridement and amphotericin B local irrigation followed by a systemic antifungal drug, usually intravenous amphotericin B. Local

debridement, evisceration and exenteration has been reported with variable outcomes. A combined open and endoscopic approach gives good exposure to the orbital apex, and allows the abscess in this region to be adequately drained.¹¹

The vague presentation of orbital aspergillosis suggests a long differential diagnosis viz. Giant cell arteritis, orbital apex syndrome (OAS), cavernous sinus thrombosis, intracranial aneurysm and pituitary apoplexy which require urgent recognition and management due to their vision-threatening and potentially life-threatening nature.

The long term survival of patients with orbital-paranasal aspergillosis despite intracranial extension is attributed to early diagnosis, optimal antifungal therapy, complete surgical debridement, and the improvement in the patient's systemic condition.¹² Hence we would like to emphasize on the importance of an early diagnosis and high suspicion of aspergillosis in an aggressively progressive intraorbital mass irrespective of the patients immunological status which would then decrease the morbidity and mortality of the patient.

We report this case because it presented with symptoms of optic neuritis that is decreased vision. As the patient had been treated for hepatic encephalopathy, the concerned ophthalmologist might have been misled into treating for Toxic amblyopia. Though, it is possible that the initial diagnosis of toxic neuropathy might have been correct given the negative scans and aspergillus might have been an opportunistic invader following the time of first presentation, however, it is emphasized that an early correct

diagnosis could have saved the life.

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