

# Orbital apex syndrome secondary to myocysticercosis: a rare case report

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**Dear Editor,**

We present a rare case report of orbital apex syndrome secondary to myocysticercosis. Human cysticercosis is caused by larval form (*Cysticercus cellulosae*) of *Taenia solium*. The cysts of *Cysticercus cellulosae* can lodge in the central nervous system, muscles, subcutaneous tissue, or eye, resulting in varied clinical manifestations<sup>[1]</sup>. This case highlights an unusual presentation of orbital apex syndrome secondary to cysticercosis of medial rectus.

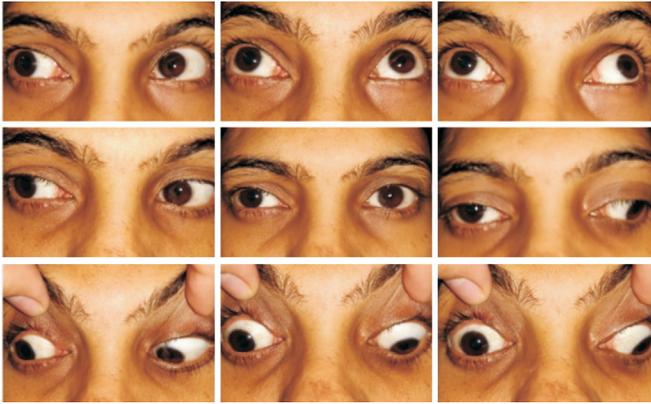
The study was conducted in accordance with the Declaration of Helsinki. Proper consent for publication of photographs of patient was taken. After explanation of the nature and possible consequences of the study, informed consent to participate was taken.

A 28-year-old female presented with pain in the right eye (RE), right sided headache for 1wk and diplopia with markedly decreased ocular movements for 3d. On examination, best corrected visual acuity (BCVA) in the RE was 6/12 and left eye (LE) was 6/6. RE had 15° exotropia with marked limitation of adduction, elevation and depression with mild limitation of abduction (Figure 1). Cornea was clear. Sensations were normal and relative afferent pupillary defect (RAPD) was present. Posterior segment showed disc edema (Figure 2A). LE was normal. Keeping in mind the clinical presentation of ophthalmoplegia with optic neuropathy, the patient was investigated for retro-orbital, orbital apex or cavernous sinus lesions. A thorough systemic evaluation was done to rule out

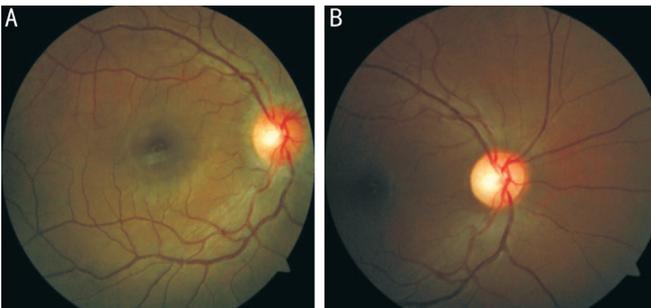
underlying pathology. Patient was afebrile with normal blood pressure (BP) and pulse. Complete blood count was normal except for a slightly raised total leucocyte count (TLC). Erythrocyte sedimentation rate (ESR) was 62 mm. Mantoux test and chest X-ray were normal. Human immunodeficiency virus (HIV) was non-reactive. Visual field charting was normal in both eyes. Contrast sensitivity was decreased in the RE. Patient was started on intravenous methylprednisolone (IVMP; 500 mg BD) for 3d followed by oral steroids (1 mg/kg·d). RE orbital ultrasound (USG) did not reveal any abnormality. MRI of orbit revealed a peripherally enhancing well defined cystic lesion measuring 8.9×5.1 mm<sup>2</sup> with an eccentric T2 hypointense nodule suggestive of scolex in posterior aspect of right medial rectus muscle near orbital apex (Figure 3). MRI brain and paranasal sinus (PNS) were normal. Final diagnosis of myocysticercosis was made and patient was started on oral albendazole (15 mg/kg·d) under steroid cover. Patient showed alleviation of symptoms with decrease in pain, improvement of ocular motility and visual acuity (Figure 4). At 4wk follow up, BCVA in both eyes was 6/6 with normal pupillary reactions and RE disc edema resolved (Figure 2B).

In our case, patient presented with ophthalmoplegia and optic neuropathy. We investigated for retro-orbital, orbital apex or any cavernous sinus lesion. In retro-orbital lesions there may be associated proptosis, dystopia, conjunctival congestion, chemosis, choroidal folds. However no such findings were seen in our case and there was no mass on orbital USG. Lesions in cavernous sinus usually present with proptosis, chemosis, conjunctival congestion, multiple cranial nerve palsies along with pupillary involvement, optic neuropathy, decreased corneal sensations, and periorbital sensations. There may be associated fever, vomiting, and severe headache. In our case optic neuropathy was present but patient was stable systemically.

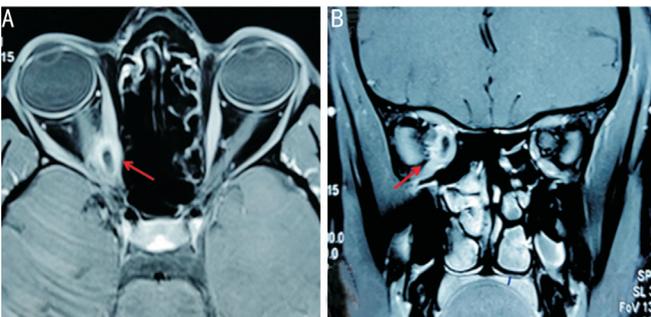
Lesions at orbital apex usually present with multiple cranial nerve palsies, pupillary involvement, optic neuropathy, and decreased corneal sensations and periorbital sensations. In our case optic neuropathy was present along with 3<sup>rd</sup> and 6<sup>th</sup> cranial nerve involvement which confirmed the diagnosis of orbital apex syndrome.



**Figure 1** Patient on presentation with exotropia and marked limitation of extraocular movements in RE.



**Figure 2** Fundus photograph of patient A: Disc edema on presentation in RE; B: Resolved disc edema in RE at 4wk post treatment.



**Figure 3** MRI brain and orbit A: Axial section; B: Coronal section, showing a peripherally enhancing well defined cystic lesion measuring 8.9×5.1 mm<sup>2</sup> with an eccentric T2 hypointense nodule suggestive of scolex in posterior aspect of right medial rectus muscle near orbital apex.

The differential diagnosis of orbital apex syndrome include Tolosa hunt syndrome, thyroid orbitopathy, mucormycosis, herpes zoster, carotico-cavernous fistula, cavernous sinus thrombosis, hematological malignancies, metastasis, and pseudotumor<sup>[2]</sup>. In our case, MRI orbit revealed cysticercosis of medial rectus at orbital apex.

Orbital apex syndrome secondary to myocysticercosis was a diagnostic challenge in the present situation due to its rarity. There is only one case report in literature where patient



**Figure 4** Patient with full and free extraocular movements at 4wk follow up.

developed orbital apex syndrome secondary to optic nerve cysticercosis<sup>[3]</sup>. In our case patient presented with orbital apex syndrome which was secondary to myocysticercosis. Rare cases of cysticercosis have also been reported in lacrimal sac and optic nerve<sup>[4-5]</sup>. In such cases, medical therapy is most effective when started early<sup>[4]</sup>. Our patient was started on oral albendazole and systemic steroids which not only helped in good functional recovery but also prevented compressive damage to optic nerve. Orbital apex syndrome is a rare clinical presentation of orbital cysticercosis. High index of clinical suspicion and awareness of varied clinical spectrum helps in early diagnosis and prevention of blinding complications. Neuroimaging plays a vital role in diagnosis especially in such cases. Timely medical therapy instituted timely helps in good recovery.

#### ACKNOWLEDGEMENTS

**Conflicts of Interest:** Dhiman S, None; Anand K, None; Rastogi A, None; Dutta P, None; Jain P, None; Mishra M, None; Nagpal V, None.

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