

# Orbital apex syndrome secondary to optic nerve cysticercosis

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**Abstract** A 22-year-old male presented to us with complaints of sudden painful loss of vision in left eye 10 days ago along with inward deviation of the left eye. Best-corrected visual acuity (BCVA) in right eye was 20/20 and 20/50 in left eye. Left eye showed limitation of abduction, a relative afferent pupillary defect, normal anterior segment with optic disc oedema. Contrast-enhanced MRI of the brain and orbit showed thickening of left optic nerve along with a cystic lesion near the orbital apex with a central iso- to hyperintense spot resembling a scolex. A diagnosis of left orbital apex syndrome secondary to optic nerve cysticercosis was made. Patient was treated with oral albendazole and intravenous corticosteroids for 3 days followed by oral corticosteroids. Ten weeks post-treatment, his BCVA in the left eye improved to 20/20 and colour vision and visual fields improved. Pallor of the left optic disc was noted, and ocular motility improved completely. MRI after treatment showed a decreased thickness of left optic nerve with disappearance of the cystic lesion.

**Keywords** Optic nerve cysticercosis · Orbital apex syndrome · Sixth nerve palsy

## Case report

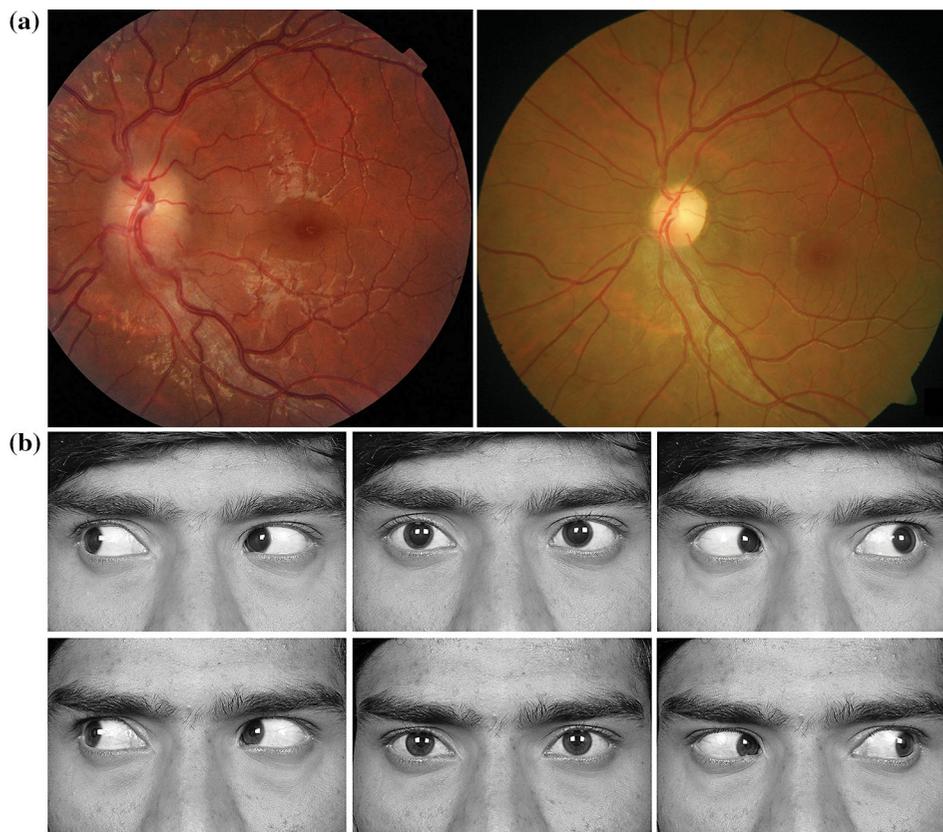
Orbital apex syndrome is characterized by optic neuropathy and ophthalmoplegia. Causes of orbital apex syndrome include carotid cavernous fistula, orbital and optic nerve neoplasms, orbital inflammation and infections like aspergillosis, mucormycosis and herpes zoster. We present a case of orbital apex syndrome secondary to optic nerve cysticercosis. The appropriate consent from the patient was obtained.

A 22-year-old male presented to us with complaints of sudden, painful loss of vision in left eye since 10 days with inward deviation of the left eye. There was no history of trauma, nausea, vomiting or other neurological symptoms. Best-corrected visual acuity (BCVA) was 20/20, N6 at near and 20/50, N18 at near in the right and left eye, respectively. Colour vision, using Ishihara pseudo-isochromatic chart in right eye, was normal and was decreased in left eye. There was no proptosis. Left eye showed – 1 limitation of abduction (Fig. 1c), with 14 prism dioptres (PD) esotropia in primary gaze at distance fixation, which increased to 18 PD esotropia in left gaze and orthotropia in right gaze. The esodeviation measured 6PD at near. Anterior segment and intraocular

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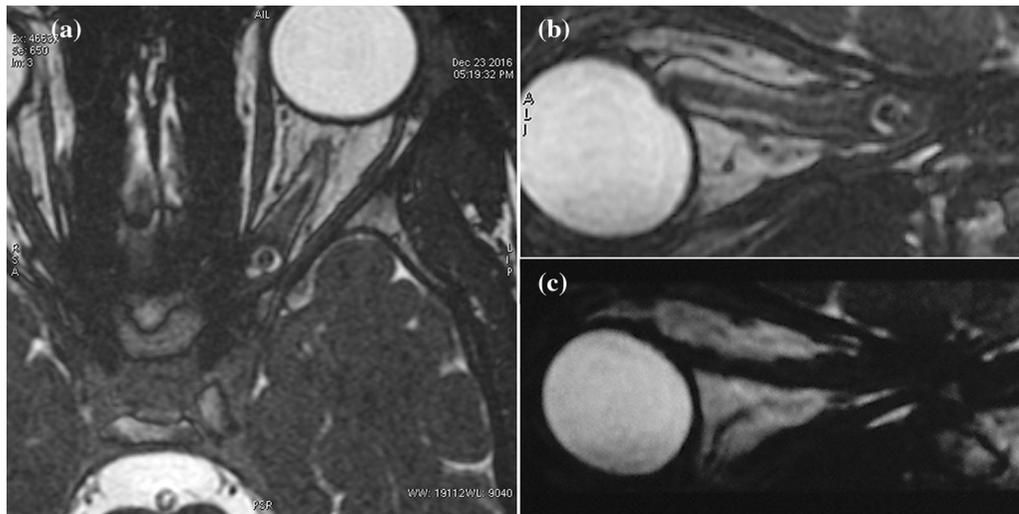
**Fig. 1** **a** Fundus photograph of left eye showing optic disc oedema with mild pallor. **b** Right side—resolved disc oedema with pallor of disc and gliosis of RNFL—5 weeks after treatment. **c** Ocular motility examination showing limited abduction in the left eye

pressure in both eyes were normal. Left eye showed a relative afferent pupillary defect (RAPD). Fundus was normal in right eye, while the left eye showed an elevated, oedematous disc with the obscuration of the cup (Fig. 1a). Visual field testing with automated perimetry revealed an inferior altitudinal defect. Magnetic resonance imaging (MRI) with gadolinium contrast of the brain and orbits showed thickening of the left optic nerve and its sheath along with a cystic lesion within the optic nerve at the orbital apex (Fig. 2a). The cyst had well-defined margins and a small isointense body within the cyst suggestive of a scolex with post-contrast enhancement of the surrounding area. Serum ELISA for immunoglobulin G (IgG) for cysticercosis was negative.

A diagnosis of left eye orbital apex syndrome, likely secondary to optic nerve cysticercosis, was made. Patient was started on pulse therapy of intravenous methyl prednisolone 1 g per day for 3 days along with oral albendazole 400 mg twice a day. This

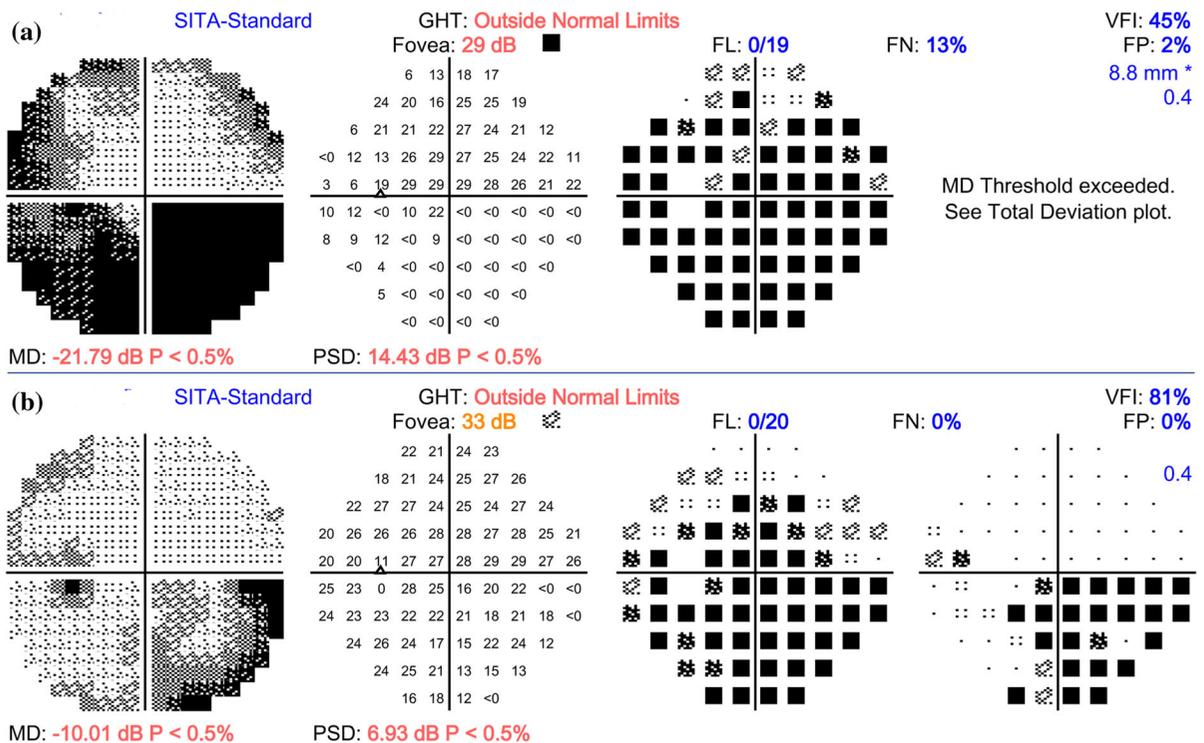
was followed by oral steroids, which were tapered over 5 weeks. Vision improved to 20/20 at the end of IVMP pulse therapy, colour vision returned to normal at 1 week, and optic disc oedema resolved clinically by 3 weeks with some residual pallor of the optic nerve (Fig. 1b). Ocular motility in both eyes was normal and painless. Visual fields showed a progressive improvement (Fig. 3a, b). No side effects were noted. MRI was repeated after 5 weeks, which showed a decrease in optic nerve thickness, disappearance of the cystic lesion and resolution of surrounding inflammation (Fig. 2c).

Cysticercosis is caused by the infestation of the cyst form of *Taenia solium* and rarely *Taenia saginata*. The most common sites of infestation are subcutaneous tissue (24.5%) [6], brain (13.6%) and eyes (12.8%) [1]. Even though ocular and neurocysticercosis is fairly common, optic nerve cysticercosis is quite rare with only a few cases reported till date.



**Fig. 2** High-resolution 3D FIESTA images in axial (a) and quasisagittal (b) planes reveal a thin-walled cystic lesion expanding the left optic nerve in the region of the orbital apex. Eccentrically located nodular component (scolex) is seen within

the lumen of the cyst. Distension of the optic nerve sheath and protrusion of optic papilla are also seen. Quasisagittal FIESTA image after therapy (c) revealed the complete resolution of the cystic lesion and other changes in the left optic nerve



**Fig. 3** a Pre-treatment visual field showing an inferior altitudinal defect. b Post-treatment visual field showing improvement in the inferior visual field

To our knowledge, this is the first case of optic nerve cysticercosis presenting as orbital apex syndrome to be reported in the literature. Recently, Jain et al. [2] reported a case of retro-orbital optic neuritis with the adduction limitation in case of cysticercosis. But neuroimaging showed cysticercosis of the medial rectus with surrounding inflammation affecting the optic nerve. In our case, the cyst with the scolex was located inside the nerve and the surrounding inflammation caused the abducens nerve palsy, which resolved completely with treatment. Chandra et al. [3] postulated that haematogenous spread through central retinal artery could be the cause for optic nerve cysticercosis. However, in our case, posterior part of the optic nerve was involved, which is not supplied by the central retinal artery. The route for such posterior spread could be the pial vessels supplying the optic nerve.

Imaging plays a vital role in making the diagnosis of optic nerve cysticercosis. Involvement of the anterior part of the optic nerve can be picked up by an ultrasound B scan. In our case, posterior part of the optic nerve was involved close to the orbital apex. Such posterior lesions may not be picked up with a B scan. CT and MRI show a well-defined cystic lesion, filled with fluid and small central dot-like lesion within the cyst suggestive of a scolex, with surrounding inflammation. Chandra et al. recommend an orbital CT scan with 1-mm-thick slices, for the detection of a scolex, which appears in the vesicular stage. On MRI, we suggest the 3D FIESTA (fast imaging employing steady-state acquisition) sequence to be especially helpful to identify the cyst along with the scolex.

Serological testing for cysticercosis could provide ancillary evidence of infection; however, its utility in endemic regions is doubtful. Also, in most cases of single lesional neurocysticercosis, immunodiagnostic tests show low sensitivity [4]; hence, in spite of a negative serological test, in the presence of the typical imaging features, we persisted with a diagnosis of cysticercosis.

The natural course of the disease is highly variable. The cyst may enlarge causing either compression effects or chronic fibrosis in the surrounding tissues or may die and cause a severe inflammatory reaction.

Anthelmintic medications like albendazole and praziquantel are effective, safe and inexpensive. Steroids help to control the secondary inflammatory reaction to cysticidal therapy. Hence, a combination of steroids with cysticidal medications is advocated in the treatment of cysticercosis. Earlier reports show conflicting results of this therapy with inadequate response noted in some [5] and good visual recovery seen in some cases [6].

In our case, after treatment with pulse therapy of intravenous steroids along with oral albendazole, dramatic visual recovery was seen, although there was a significant residual field defect in the left eye. Patient tolerated the therapy well.

Optic nerve cysticercosis, although rare, must be considered as a differential diagnosis in patients with atypical optic neuritis, with suspicious lesion found on neuroimaging, especially in regions endemic for the disease and in patients with history of recent travel to such areas. Prompt diagnosis and treatment with anthelmintics and steroids can result in a reasonably good visual recovery.

#### Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interests.

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