A 25-year-old woman presented with a low-grade fever, loss of weight and appetite of 4 months duration and intermittent vomiting of two months duration. The diagnosis was tubercular meningitis and the patient was on anti-tubercular therapy from one month ago. Two weeks ago, a rapidly progressive visual loss emerged in two days. In general observation, she was thin, had mild pallor and no icterus, was conscious and also irritable. In physical examination, she was febrile (100°F), there were bilateral crepitations in the chest and she had mild neck rigidity. On eye examination, there were bilateral dilated sluggishly reacting to light pupils, no projection or rays or perception of light in both eyes, the fundus showed bilateral papilloedema with features of secondary optic atrophy. Extra ocular movements were restricted in all directions suggestive of 3rd, 4th and 6th nerve paresis. Other cranial nerves were normal. There were no focal motor or sensory deficits. Blood investigations were normal except for a raised erythrocyte sedimentation rate (64 mm in the 1st hour). Three consecutive samples of sputum for acid fast bacilli were negative. The brain CT scan showed mild dilation of the third and lateral ventricles and thick basal exudates (Fig. 1.A&B). MRI of the brain showed hypertrophy of the chiasma and the cisternal segment of both optic nerves after contrast enhancement (Figs. 2&3).

What is your diagnosis?
Diagnosis: Tubercular Optochiasmatic Arachnoiditis

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Based on clinical features and imaging findings the diagnosis of optochiasmatic arachnoiditis was made. Anti-tubercular therapy and steroids were prescribed. Though her headache and constitutional symptoms ameliorated, there was no improvement in her vision.

Optochiasmatic arachnoiditis is a rare manifestation of many disorders including tubercular meningitis,1,3 bleeding from a rupture of an internal carotid artery aneurysm,4 radiation injury,5 or systemic vascular disease.6 It has been postulated that involvement of the optic nerve and chiasm is an expression of a generalized disease of the arachnoids, with predominant clinical symptoms and signs referable to the optochiasmal region and histopathological findings emphasize the importance of vascular factors in the pathogenesis of this syndrome.6 It usually presents with progressive and profound visual loss in patients who are receiving anti-tubercular treatment and are recovering from the illness.1,3 The visual evoked cortical potential is used to localize the site of the lesion along the visual pathway as well as to assess the postoperative recovery.3 MRI gives better details of the lesions and will demonstrate the perichiasmal enhancement.2,7 It will also show any associated hydrocephalus or cerebral infarction.2 Apart from the anti-tubercular therapy and steroids,1,3,8 microneurosurgical decompensation of the optic nerves and chiasm is recommended in the selected group of patients to hasten the visual recovery.1,3 The role of surgery is controversial and deterioration may follow the initial temporary improvement after surgical lysis of adhesions.8 This may be due to the facts that many factors influence the outcome of surgical treatment and include inability to determine whether the visual loss is because of papilloedema or optic atrophy, or it is due to fibrous strangulation or direct ischemic parenchymatous damage.6

References