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the patient developed diabetes insipidus, which was managed with desmopressin.

The second patient was a 6‑year‑old girl who presented with headache and vomiting for 2 months. CT scan showed a calcified in third ventricle with hydrocephalus [Figure 2a]. MRI showed a heterogeneous lesion occupying the entire third ventricle [Figure 2b]. There was no evidence of recent hemorrhage. She underwent VP shunt followed by subfrontal approach, and only partial excision of the lesion as the lesion was densely adherent to the floor of the third ventricle. Histopathology was cavernoma. After surgery she developed transient hyponatremia, which was managed medically.

Third ventricular cavernomas can present with signs and symptoms of any third ventricular tumors, however they usually present with symptoms of hydrocephalus. Presentation as intraventricular hemorrhage is uncommon, and only three cases have been reported. Our first patient presented with hemorrhage, and second with hydrocephalus. They can arise from the suprachiasmatic region, foramen of Monroe, lateral wall or floor of third ventricle. Both of our patients had cavernomas arising from floor of third ventricle. The imaging appearance is that of typical cavernomas. The close differential diagnosis in the absence of hemorrhage is third ventricular craniopharyngioma. The natural history of intraventricular cavernoma is not known. There is a risk of hemorrhage and acute hydrocephalus if the lesion is not excised completely. However, there is risk of hypothalamic damage in case of complete resection of lesion arising from floor of third ventricle, as it happened in our first case. It may be worthwhile doing only a VP shunt for hydrocephalus if the lesion is arising from the floor of the third ventricle.

A. R. Prabhuraj, Dhaval Shukla
Department of Neurosurgery, National Institute of Mental Health and Neurosciences, Bangalore, Karnataka, India

Address for correspondence:
Dr. Dhaval Shukla, Department of Neurosurgery, National Institute of Mental Health and Neurosciences, Bangalore ‑ 560 029, Karnataka, India. E‑mail: neurodhaval@rediffmail.com

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Isolated nuclear III nerve paresis secondary to neurocysticercosis showing spontaneous clinical improvement

Sir,
We wish to present a case of isolated nuclear IIIrd nerve paresis secondary to neurocysticercosis (NCC) showing spontaneous clinical improvement. Isolated third cranial nerve palsy is rare in NCC. A 14-year-old
male child of Nepali origin presented with diplopia for 6 days in left lateral gaze and drooping of his upper eyelids in both eyes. The complaints were insidious in onset, non-progressive simultaneous in both eyes with no diurnal fluctuation. Rest of his present and past medical history was not significant. He had history of pork consumption. His general physical examination and systemic examination including neurological examination was unremarkable.

His visual acuity was 6/6 OU with increased wrinkling on forehead. He had severe neurological ptosis in both eyes with poor levator palpebrae superioris (LPS) action with no Marcus Gunn phenomenon and no sign s/o fatigueability. He also had an exotropia of about 15 degree and mild paresis of elevation of eyeball in right eye maximum in lateral gaze. He had mild paresis of his right medial rectus with end gaze nystagmus in left gaze. Rest of the ophthalmological examination was unremarkable including papillary reactions. Diplopia charting showed horizontal diplopia in all gaze positions with maximum difference in left lateral gaze positions [Figure 1]. While waiting for his investigation reports his sign and symptoms showed spontaneous clinical improvement. His magnetic resonance imaging (MRI) showed a solitary ring-enhancing lesion in the pre-aqueductal region of mid brain of size 0.72 × 0.68 cm involving IIIrd nerve nucleus with peri-lesional edema [Figure 2]. His lumbar puncture was done and it revealed CSF protein levels 60 and it also showed 9 white blood cells mainly lymphocytes. ADA levels in his CSF were within normal limit. His serology revealed positive IgG levels for taenia solium (1.13 U/ml) by ELISA. In the light of the above findings the patient was diagnosed as a case of nuclear third nerve paresis secondary to NCC of brainstem. After consultation with Department of Neurology the patient was put on oral albendazole and prednisolone. He was continuously followed up with no similar episodes till 1 year after.

NCC is the most common parasitic infestation of the nervous system. Globally, NCC is endemic in Central and South America, sub-Saharan Africa, and in some regions of the Far East, including the Indian subcontinent, Indonesia, and China, reaching an incidence of 3.6% in some regions. Del Brutto et al. have suggested diagnostic criteria for NCC. In our case these criteria were fulfilled for probable diagnosis of NCC. It usually presents with seizures. Other various CNS clinical manifestations include headache, hydrocephalus, chronic meningitis, focal neurological deficits, dementia and psychiatric manifestations. Involvement of third nerve is rare especially due to a parenchymal lesion. Fluctuation in perilesional edema was probably responsible for spontaneous clinical recovery in our patient. The lesion in such nuclear third nerve paresis involves the central caudal nucleus (CCN), which is a small subgroup of oculomotor nucleus containing subnucleus for levator palpebrae superioris. Involvement of this nucleus results in complete, bilateral ptosis.

We propose a more detailed evaluation of patients with fluctuating ptosis. In those patients where ptosis is restricted to ocular muscle without any other neuromuscular involvement, cranial imaging needs to be done, preferably MRI of the brain with contrast studies that may show lesions in the midbrain.

Sunil Chauhan, Gopal Damani, Ravinder K. Gupta, Kulbhushan P. Chaudhary
Letters to Editor

Department of Ophthalmology, Indira Gandhi Medical College, Shimla, Himachal Pradesh, India

Address for correspondence:
Dr. Sunil Chauhan,
Department of Ophthalmology, Indira Gandhi Medical College, Shimla, Himachal Pradesh, India. E-mail: skc041080@gmail.com

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