Cortical blindness with absent visually evoked potential in non-ketotic hyperglycemia

Dear Editor,

A 19-year-old boy was referred to us for loss of vision. He had presented to the hospital two days before with the history of one episode of generalized seizure lasting for 20 to 25 sec and was in altered sensorium for two days. His blood glucose and serum osmolarity were more than 600 mg% and 308 meq/L respectively. Blood ketone bodies were normal and urinary ketone bodies were negative. There was no metabolic acidosis. He was diagnosed as having non-ketotic hyperglycemia (NKH). He was not a known case of any seizure disorder or diabetes mellitus. He improved within three days of treatment, with insulin and intravenous fluids.

On recovering, he complained of bilateral loss of vision. Ocular examination revealed vision of hand movements in both eyes. Anterior segment and posterior segment examinations including pupillary reactions and fluorescein angiography were normal. Menace reflex was absent and malingering tests were negative. He was diagnosed to have cortical blindness. Visually evoked potential (VEP) was absent in both eyes. Lumbar puncture showed normal study. Electroencephalogram, computed tomogram and magnetic resonance imaging brain were normal.

His vision improved to 20/80 and 20/60 in the right eye and left eye respectively after two weeks of follow-up. It improved to 20/20 at eight weeks of follow-up and VEP returned to normal.

Transient cortical blindness is used to describe an apparent lack of visual functioning, despite anatomically and structurally intact eyes. Transient visual loss may occur with seizures as an ictal or post-ictal phenomenon and is usually seen with status epilepticus. Its duration varies from less than one minute to days or can be even permanent. In our case, though the patient complained of visual loss after two days of seizure the insult might have occurred at the time of seizure and since the patient was in altered sensorium for two days, he might not have realized.

Hyperglycemia may cause seizures commonly involving the occipital lobe and rarely, the frontal lobe. In NKH glucose metabolism is decreased and energy requirement is met by GABA shunt. By increasing GABA metabolism, which is an important neurotransmitter inhibiting the epileptogenic phenomenon, hyperglycemia reduces the seizure threshold.

Cortical blindness is caused by hypoxia or anoxia involving the occipital lobe or the frontal lobe. Hyperviscosity in NKH may cause dehydration of glial and other supporting tissues with accumulation of free radicals. The resulting cytotoxic edema might restrict diffusion of substances which may cause transient blindness. Non-ketotic hyperglycemia is known to cause homonymous hemianopia without any evidence of any structural lesion evident on scanning but there is no report of cortical blindness in NKH patients.

Some reports suggest that VEP may show a varied result and may not be useful in establishing a diagnosis, other reports suggest prognostic importance of VEP with absent VEP response foretelling poor prognosis for visual recovery. However, in our case, despite absent VEP at presentation, patient regained full vision.

Cortical blindness should be considered in a patient with visual loss following NKH with or without seizure, but radiological investigation should be carried to rule out any foci of seizure. Further, absent VEP may not be indicative of poor prognosis.

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Dear Editor,

Duane's retraction syndrome is a well-known congenital ocular motility disorder.1 In the most common type of Duane's retraction syndrome, there is marked restriction of abduction, with eyeball retraction and narrowing of the palpebral fissure on adduction. In inverse Duane's syndrome, abduction of the affected eye is possible to some extent. However, the globe retraction occurs on abduction accompanied by narrowing of the palpebral fissure and pseudoptosis. We report a case of inverse Duane's syndrome due to myocysticercosis involving the medial rectus muscle. To the best of our knowledge, based on systematic search of English language literature, no similar case has been reported before.

An 18-year-old lady presented to us with complaints of diplopia on right gaze of 20 days duration. Visual acuity in each eye was 20/20 without any refractive error. Cover test revealed an esotropia of 8 prism diopters. Ocular motility examination revealed mild limitation of abduction in the right eye with normal adduction [Fig. 1]. There was 3 mm of globe retraction and 3 mm of narrowing of the palpebral fissure on abduction in the right eye. Forced duction test revealed restriction of abduction in the right eye. Conjunctival congestion was observed near the medial rectus muscle insertion. Ultrasound B-scan [Fig. 2] showed a grossly thickened medial rectus muscle with a cystic lesion and a high reflective spot (scolex) within suggestive of medial rectus muscle cysticercosis. The scolex was not moving nor was there any evidence of calcification. The cyst was noted to be producing scleral indentation which was confirmed on fundus examination. She was started on oral albendazole 15 mg/Kg body weight and Prednisolone 1 mg/Kg body weight. The diplopia resolved within two weeks and...

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