Carbamazepine induced optic neuropathy in an adolescent boy with conduct disorder: A rare case report

Sir,

The etiology of optic neuropathy is multi-factorial and toxin-induced (toxic) optic neuropathy (toxic amblyopia) is one of the common cause of optic neuropathy.1,2 The clinical manifestations can be in the form visual impairment, which is usually progressive, painless and involve both eyes.1,2 Involvement of the color vision is the earliest sign which usually out of proportion to the visual impairment in totality.1,2 Abnormal pupillary response to light, optic disc changes and disc hemorrhage may be seen in ophthalmoscopic examination in toxic optic neuropathy.1,2

An antiepileptic drug like carbamazepine is known to cause ocular adverse effects like nystagmus, diplopia and paralysis of extra-ocular muscles, abnormalities of saccadic eye movements.4,5

The diagnosis of toxic optic neuropathy is based on detail medical history, ocular examination, hematological investigations, visual field examination, neuro-imaging and electrophysiological tests.1 Withholding the offending medications, intravenous steroids, multivitamin supplementation are the mainstay of treatment in toxic optic neuropathy.1

A 15-year-old boy with features of conduct disorder was brought for psychiatric consultation by a nongovernment organization for his attempts of deliberate self-harm.

The patient was hospitalized for his high-risk behavior. Behavioral therapy in the form of positive reinforcement was initiated for his conduct problems. For his impulsive behavior, tablet carbamazepine (controlled-release) 200 mg/day was initiated, which was later escalated to 400 mg over next 3 days, immediately after which he had developed generalized pruritic rashes all over the body, pain in both eyes, difficulty in identifying colors and difficulty in vision. He was very much distressed about his visual difficulties. There was no history of trauma to eyes or history of any form of visual difficulty, prior to this episode. Ophthalmology and dermatology opinion was taken for his above-mentioned complaints. His visual acuity was found to be 3/60 in the right eye and 6/24 in the left eye. Ophthalmological examination revealed anisocoria and sluggish pupillary response to light in the right eye. Ophthalmoscopic examination revealed bilateral mild disc hyperemia with nasal bulging of disc margin and mild venous tortuosity. On slit lamp examination, the anterior chamber of both eyes appears to be normal. Hematological investigations, including folic acid and Vitamin B12 levels and neuro-imaging (magnetic resonance imaging) were within normal limits except raised eosinophil percent (11% in differential leukocyte count). There was no concomitant use of any other medication in this patient.

A diagnosis of carbamazepine-induced optic neuropathy was considered for which he was prescribed an intravenous injection of methyl-prednisolone (1 g/day) for 3 consecutive days. Immediately, carbamazepine was stopped. There was a significant improvement in vision and retro-orbital pain with this treatment. In this case differential diagnoses of high hypermetropia, demyelinating conditions were thought off. The dermatologist diagnosed the pruritic rashes as carbamazepine-induced hypersensitivity skin rashes. With conservative management, skin rashes completely vanished in a week.

Patient’s visual difficulties (retro-orbital pain, impairment of color vision and visual field defects) following initiation of carbamazepine treatment was suggestive of optic neuropathy. Objectively it was substantiated by visual field defect, afferent pupillary defect, color vision abnormality and optic disc changes which are the core manifestation of optic neuropathy. As per the existing scientific literature, nystagmus, diplopia, and extra-ocular cranial nerve palsy were reported with carbamazepine. However there is no report of optic neuropathy. In this case, visual difficulties seemed to be secondary to hypersensitivity reaction of carbamazepine. Evaluation on Naranjo adverse drug reaction probability scale (score = 6) was suggestive of probable carbamazepine-induced side-effect.6

Usually patients with untreated optic neuropathy land up in complete loss of vision. Fortunately, in the index patient, the intervention was done early, which led to rapid recovery. Carbamazepine was stopped immediately, and injection methyl-prednisolone was given parenteral 1 g/day for 3 consecutive days. There was a significant improvement in visual complaints with above-mentioned therapy. We did not find any previous report of carbamazepine-induced optic neuropathy after an extensive web search. This index case highlights the need to be aware of the rare entity of carbamazepine-induced optic neuropathy, which if diagnosed and managed in time can save the sight.
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Conflicts of interest
There are no conflicts of interest.

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