Unilateral acquired colour Defectiveness - an unusual cause

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Introduction

Colour defectiveness is usually congenital and at times acquired. Acquired colour defectiveness may be due to macular or optic nerve pathologies and rarely due to cataract. An unusual cause of unilateral colour defectiveness due to extrinsic Indentation of prechiasmatic segment of left optic Nerve by internal carotid artery is reported here.

Case Report

A 60 – year old patient presented to our out-patient department with a peculiar complaint of colors getting washed out or faded when he viewed with his left eye. He has been having this compliant for the past 2 years which was investigated and no cause was identified. His discomfort has exacerbated considerably since two months with respect to red color. The patient who is an anaesthesiologist by profession noticed the disease, when he accidentally detected that the bright colored flowers in his garden had literally faded when he viewed them with his right eye closed. When he opened his right eye the picture dramatically changed.

There was no history of diabetes mellitus or hypertension or any other associated diseases. An ophthalmologic assessment with computerised tomographic scan (CT scan) one year ago did not contribute to any diagnosis.

On examination, the visual acuity of both eyes was 6/6 and near vision N6 with correction. Slit Lamp examination did not reveal any cataract of the lens. The eye was quiet. There was subtle left afferent pupillary defect on swinging torchlight test. Intraocular pressure of both eyes was within normal limits. Direct and indirect ophthalmoscopy revealed normal fundus

Findings

A colour vision assessment with Ishihara’s chart revealed gross red colour defect in the left eye and a normal colour vision on the right side. An automated Humphrey perimetry showed a relative paracentral scotoma in the supero and inferonasal aspect of the left eye and in the superonasal aspect of the right eye (Fig.1&2).

Based on the above findings a left optic nerve pathology was suspected and patient was sent for a magnetic resonance imaging scan (MRI) to rule out retro-bulbar and retro orbital optic nerve lesions.

The MRI turned out to be enlightening. The prechiasmatic segment of the left optic nerve was found to be indented on inferolateral aspect by supraclinoid segment of the internal carotid artery (Fig.3). There was no degeneration or demyelination of the optic nerve. The right optic nerve, optic chiasma and optic tracts were found to be normal.

A diagnosis of left optic nerve compression by left internal carotid artery was made.

Discussion

Optic nerve pathologies are one of the foremost causes of acquired red colour blindness or defectiveness. The commonest cause for this is optic neuritis, when the vision is drastically reduced, with slight ocular discomfort and tenderness. The causes may be viral, autoimmune, or demyelinating diseases. Although in papillitis the fundoscopy may clinch the diagnosis, in retro bulbar neuritis the only positive finding may be blurred vision and an afferent pupillary defect.

The initial clinical diagnosis in this patient was an acute on chronic optic neuritis, but the points against were the absolute normal vision and not so typical visual field defect. Fundoscopy was also normal in this case.

The other suspicion in this case was some retro-bulbar growth pressing on the optic nerve. However a two years
history, the disc appearing pink and healthy and not showing any atrophic changes was unexpected in such a case. The only factor suggestive of optic nerve compression was the subtle afferent pupillary defect. The history of a CT scan done a year ago was normal, and hence the patient was subjected to an MRI scanning with special emphasis on retrobulbar and retro-orbital region that helped in clinching the diagnosis. Internal carotid artery aneurysms, mostly of the supraclinoid and rarely of the infraclinoid region causes visual disturbances resembling parasellar tumor or retrobulbar neuritis. Carotid-ophthalmic aneurysms have intimate relationship with ipsilateral optic nerve and neighboring structures.

Jacobson DM described 18 patients with 24 affected eyes having compression of optic nerve by carotid artery. Those patients had features varying from optic neuropathy, bitemporal hemianopia and oculomotor nerve palsy with intracavernous mass effect due to dolichoectatic carotid artery. Coronal oriented MRI was used to confirm anatomic compression effects on optic nerve.

The field defect in the opposite eye may also be due to an early internal carotid artery compression not yet demonstrable radiologically.

In another study Jacobson DM compared optic nerve contact and compression by carotid artery, in asymptomatic patients. He concluded that supraclinoid carotid artery contact with intracranial optic nerve occurs frequently. Anatomic compression occurs infrequently and is directly proportional to the diameter of carotid artery.

In carotid-ophthalmic aneurysms the treatment of aneurysmal clipping is difficult and hazardous. Hence in such cases an indirect operation of carotid artery ligation in the neck could be done provided the brain perfusion is demonstrated angiographically to be satisfactory even after temporary of occlusion of the concerned carotid artery. In the present case, carotid artery ligation in neck could be employed if the condition is progressing. As the patient has not agreed for any intervention presently, he is on regular follow-up.

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Legend for figures
Fig 1 & 2
Perimetric findings showing a paracentral scotoma in superoand inferonasal aspect of left eye and superonasal aspect of the right eye
Fig 3
MRI showing indentation of inferolateral aspect of left optic nerve by internal carotid artery

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