Case Report

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Non Arteritic Anterior Ischaemic Optic Neuropathy Secondary to Acute Primary Angle Closure: A Case Report

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Abstract

Purpose: To describe a case of non-arteritic anterior ischemic optic neuropathy (NA-AION) secondary to acute angle closure (AAC). *Case Report:* We present a case of *48 years* old female who presented with painful diminution of vision in the OS. After examination of the anterior segment she was diagnosed of having an acute attack of primary angle closure glaucoma. On fundus examination OD was within physiological limits, OS the disc was edematous and pale with superficial haemorrhage at disc margin. Medical management and laser peripheral iridotomy relieved pain but did not improve vision. The visual fields showed superior altitudional field defect. Patient was not a known case of diabetes mellitus or hypertension. The patient was diagnosed with NA-AION secondary to acute angle closure glaucoma. *Results:* A *48-year-old* woman presented with AAC with reduced visual acuity. Investigations revealed concurrent NA-AION with classical disc and visual field findings. *Conclusion:* NA-AION secondary to AAC is a rare clinical entity that can result in severe vision loss. Thus, it is important to look beyond the angle in cases of acute angle closure glaucoma.

Keywords: NA-AION; ACC.

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Introduction

The prevalence of acute angle closure (AAC) is 0.3 to 2.7 percent in patients older than 40.[1,2] The incidence of non-arteritic anterior ischemic optic neuropathy (NA-AION) is two to ten per 100,000 in the population older than 50.[3–5] NAION secondary to acute primary angle closure was first described by Sonty and Schwartz in 1981.[6] Patients present with typical AAC symptoms and signs, which include pain, headache, nausea and vomiting, conjunctival injection, corneal edema, elevated intraocular pressure (IOP), closed angles, and a shallow anterior chamber. NA-AION was present at the time of presentation for AAC in five of seven eyes and developed one week after AAC in two eyes.[5-9]

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Case History

Reporting a case of a *48 years* old female presented to the ophthalmology OPD with complains of marked diminution of vision, pain and redness in left eye associated with left sided headache for 8 days. She gave history of two similar episodes in the past one *year*.

On examination her visual acuity was *OD-6/6* and *OS-6/24*, not improving with pinhole. The anterior chamber was shallow in OD with a Von Herick (VH) grading 2. The pupil was central circular sluggishly reacting to light and the rest of the OD structure were found to have no abnormality. While in os the lids were edematous and non tender conjunctival congestion with corneal edema, shallow anterior chamber with a VH grading 2, pupil was mid

dilated not reacting to light. The intraocular pressure in the OD was *14.6 mm Hg* and in the OS was more than *49.5 mm Hg* by schiotz tonometer. On fundus examination, OD the disc was vertically oval with the cup to disc ratio of 0.4 with rest of fundus within normal limits. The left eye disc margins were blurred with pallor of neuroretinal rim, superficial hemorrhage superonasal to the disc, the cup to disc ratio was not appreciated and the arteries and veins were normal in thickness and caliber (**Fig. 1**).

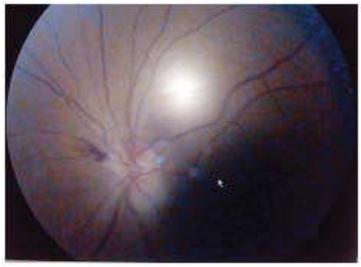

Fig 1: Fundus Photograph at presentation

Gonioscopy was done after controlling the IOP medically and the angles were Grade 2–3 in all quadrants in OD and Grade 1 in 3 quadrants in OS by Shaffers system. Visual fields in the OS showed superior altitudinal field defects (**Fig. 2**). Visual evoked potential suggested a mixed optic neuropathy in the OS. On presentation the patient was given systemic hyperosmolar agent (iv mannitol stat) and oral carbonicanhydrase inhibitor (tab. Acetazolamide *250 mg* QID) with topical pilocarpine and beta blocker (timolol 0.5% BD) along with supportive medications. Once the acute attack subsided, a therapeutic Nd-YAG laser peripheral iridectomy was done in the OS and prophylactic in OD. Intraocular pressure was well controlled, but the visual acuity does not improve. Patient was given a trial of systemic steroid (inj. Methyl prednisolone *1 gm* for *3 days*), which resulted in improvement of visual acuity upto 6/12 unaided improving upto 6/9 with pinhole. The patient was followed up for a period of *6 months*,

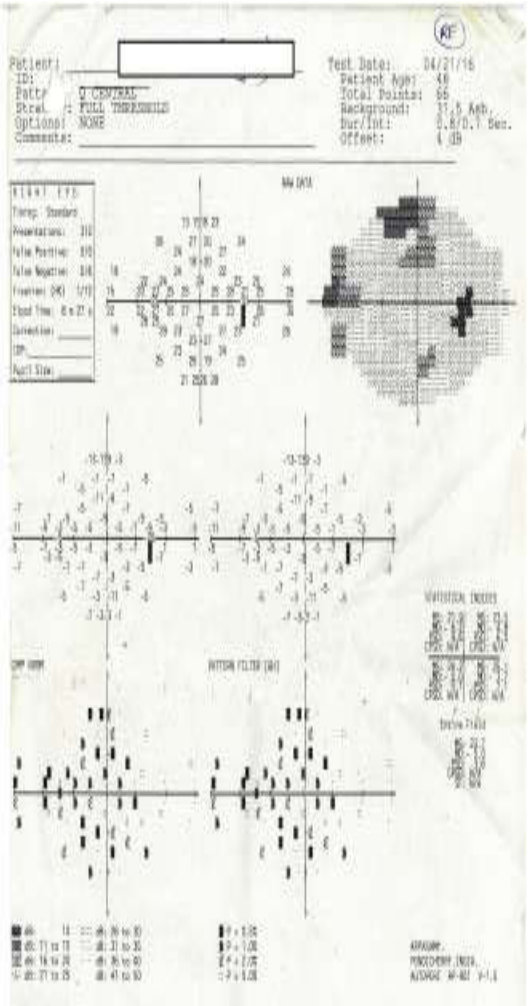

Fig 2A: Visual field charting

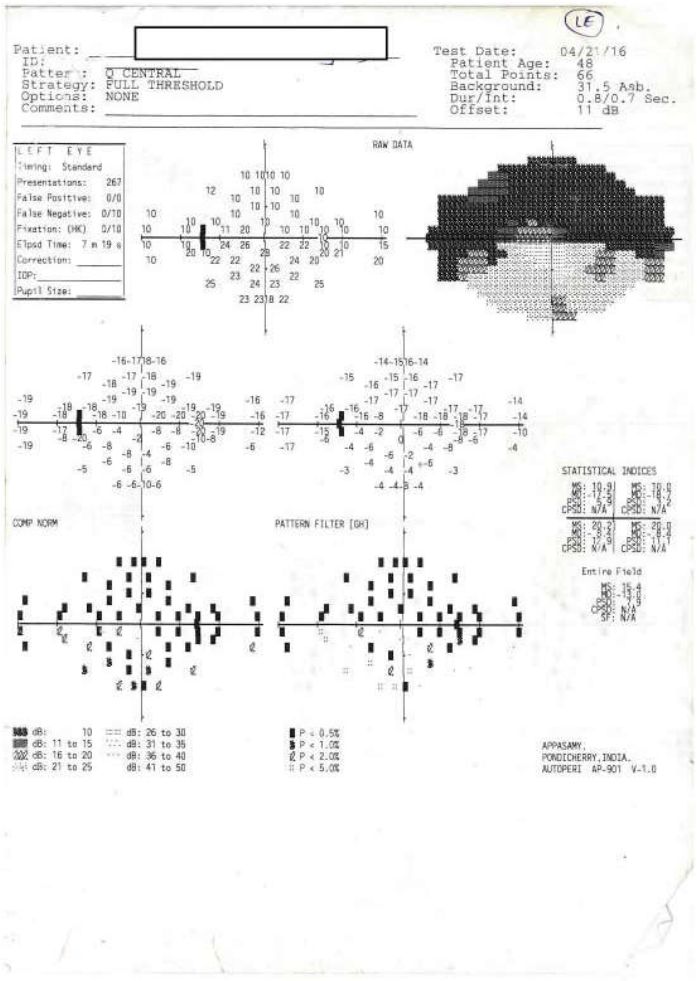

Fig. 2B: Visual field charting

the visual acuity and the intraocular pressure was maintained, but subsequent optic nerve head pallor was noted.

Discussion

NA-AION secondary to AAC is a rare clinical entity resulting in profound vision loss. Posterior ciliary artery and its branches are the principle blood supply of optic nerve head. NA-AION is due to ischemia of optic nerve head.[11, 17] Ratio of perfusion pressure and resistance to flow determines the blood flow to the optic nerve head.[12] Rheological properties of blood along with vascular autoregulation and integrity decides the resistance to flow.[12] Per-fusion pressure is the difference in mean arterial pressure (MAP) and IOP.[12] The MAP is reduced in cases of nocturnal hypotension or due to antihypertensive medication.[12] These are impaired in patients with hypertension, diabetes and atherosclerosis.[12] High IOP can precipitate episode of NA-AION by causing derangement in perfusion to optic nerve head. It has been reported in cases during high IOP in phacoemulsification leading to increased incidence of NA-AION after cataract surgery.[13–15] Elevated IOP has been identified as a causative factor for reduced perfusion to the optic nerve head in six cases (seven eyes).[14] It has been described that the cycle of NA-AION is due to ischemia of axons leading to axoplasmic stasis, which results in axoplasmic accumulation and swelling, compresses the capillaries, thus the vicious cycle ischemia sets in.[12]

Patients with AAC can also develop mild optic nerve edema that is not associated with NAAION.[18] Tsai *et al.* stated a statistically significant difference in the retinal nerve fiber layer thickness (RNFL) of the AAC eye and the contra-lateral eye measured by OCT, one week after the APAC event.[18] However, a statistically significant difference was not observed at the *4-* and *12-week* follow-ups.[18] These patients can be differentiated from NA-AION secondary to

AAC patients by the lack of subsequent optic nerve atrophy and preserved visual acuity and visual field.

NA-AION secondary to AAC is a rare but potentially blinding clinical entity. In addition to counseling patients about NA-AION risk factors (eg, evening antihypertensive medications and control of hypertension, diabetes, and dyslipidemia), patients must be counseled on the importance of performing an LPI in the contra-lateral eye. In AAC patients with an RAPD, a large amount of optic nerve edema, persistent decreased visual acuity, and a small cup-to-disc ratio in the contralateral eye should raise clinical suspicion for NA-AION.[19]

AAC glaucoma can per se present with some amount of disc edema secondary to angle closure. Cases with AAC and disc edema may have NA-AION. Thus all cases of AAC with disc edema need thorough examination and investigation in the lines of NA-AION. The visual acuity secondary to AAC is reversible after the proper management of acute attack, but visual loss secondary to NA-AION may not revert, if not diagnosed early and treated with steroids resulting in permanent vision loss. Thus, it is again stressed upon to diagnose case of acute angle closure with NA-AION, to prevent permanent vision loss and similar attack in the fellow eye. Also NA-AION can develop even days after the acute angle closure glaucoma attack so these cases require early follow up to diagnose and treat.

Key Message: AAC can be associated with NA-AION, thus fundus examination and prompt management is required to prevent permanent visual loss.

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