Ischaemic Optic Neuropathy – Non Arteritic/NA-AION

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Introduction

Ischaemic optic neuropathy (ION) is the commonest adult optic nerve disorder encountered worldwide and can be expected to increase in incidence in our ageing population. In a recent review of 121 cases [1] the mean age was 61 years. The condition has been classified as a) anterior (AION) affecting the optic nerve head and b) posterior (PION) involving that portion of the optic nerve behind its immediate retrolaminar portion. Furthermore there are two pathological varieties of the disease c) Arteritic (AAION) almost exclusively associated with Giant Cell Arteritis (GCA) and d) Non-arteritic (NA-AION or less correctly NAION) usually associated with diabetes, hypertension and hypercholesterolaemia. A recent treatise on the subject [2] runs to more than 600 pages.

In order to understand the pathology of ION knowledge of the complex vascular anatomy of the optic nerve head (ONH) and its more posterior part is required. This was not clarified until the mid1960's by the work again of Hayreh [3] when he showed that the ONH is supplied in the main by the ciliary vascular system and not by the ophthalmoscopically visible central retinal artery; furthermore the more posterior part of the nerve is supplied from its surrounding pial plexus fed from adjacent orbital branches of the ophthalmic artery (Figure 1a). The ophthalmic artery is the first intracranial branch of the internal carotid when it emerges from the cavernous sinus. The central retinal artery, also a branch of the ophthalmic, only supplies the surface/retinal layer of the ONH (Figure 1b) before it proceeds to supply the inner layers of the retina.

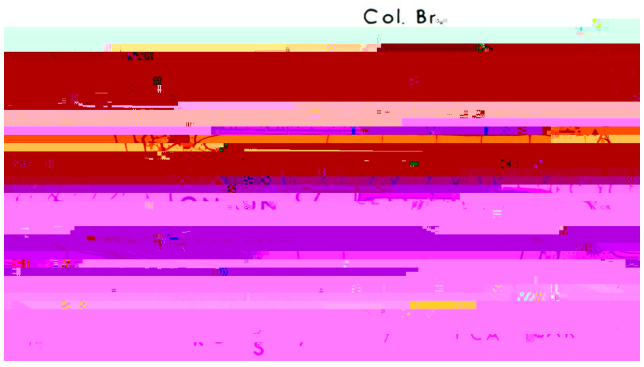

Figure 1a: Blood supply of the orbital optic nerve after Hayreh. CRA: Central Retinal Artery. CRV: Central Retinal Vein. PCA: Posterior Ciliary Artery. CZ: Circle of Zinn. D: Dura. A: Arachnoid. P: Pia [4].

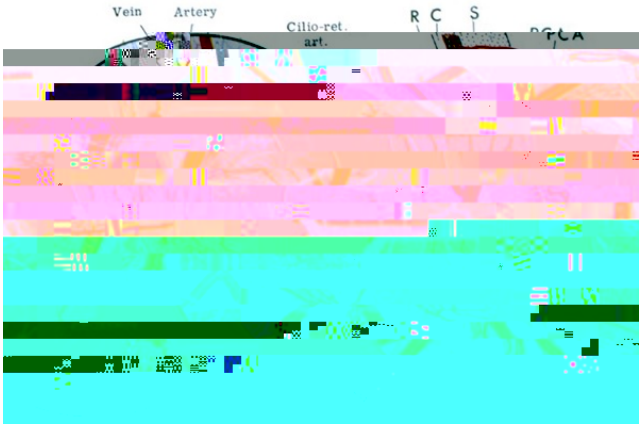

Figure 1b: Blood supply of the optic nerve head after Hayreh. CRA: Central Retinal Artery. CRV: Central Retinal Vein. PCA: Posterior Ciliary Artery. CZ: Circle of Zinn. D: Dura. A: Arachnoid. P: Pia [4].

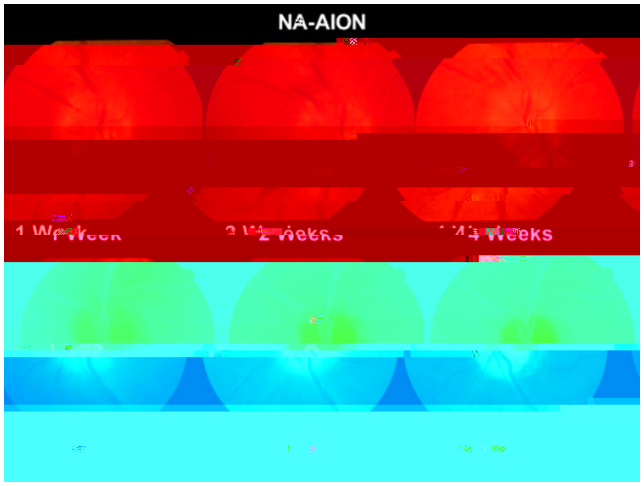

Figure 2: Progression of Optic Nerve changes in NA-AION from initial hyperaemia to optic atrophy at six months.

HXe condition usually occurs in a small so called “disc at risk” one with no signiẐcant cup. Visual Ẑeld examination is essential for diagnosis and a lower altitudinal or lower nasal defect (Figure 3) will be found in 75 % of cases [1]. Where the defect is central or paracentral as in about 10 % of patients’ central vision will be aẉected at the outset.

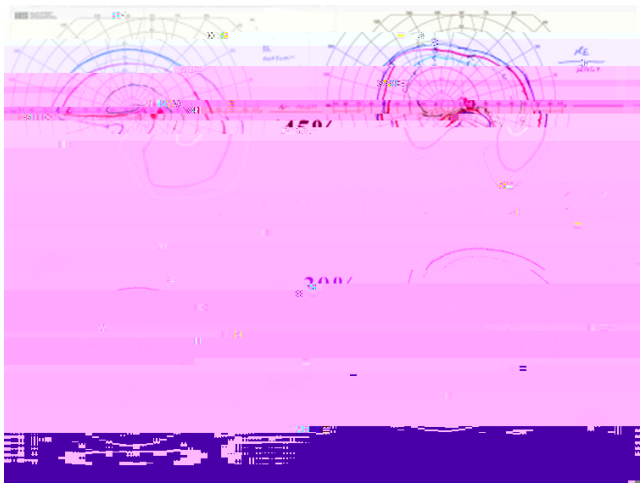

Figure 3: Goldmann visual Ẑelds showing characteristic inferior defects as seen in 75% of cases of NA-AION.

Functional recovery is to be expected in only a small percentage of cases [6] and complete recovery never occurs. HXe condition is stable around six months with no change expected thereaꟷer. Involvement of the fellow eye is reported in 14.5% of cases over Ẑve years [7].

HXere is no proven eẉective treatment other than control of underlying vascular disease and hypertensive patients should be told not to take treatment late in the day. A neuroprotective agent such as brimonidine tartrate eye drops may be tried and as it has an intraocular pressure lowering eẉect this may also be helpful, and at least in the absence of other therapy it is important to oẉer the patient some medication which has at least a logical basis for its use. Because the cause of the visual defect is loss of perfusion in the ONH circulation, not embolic, thrombotic or inẑammatory,

9. Parsa CF, Hoyt WF (2015) Non arteritic anterior ischemic optic neuropathy: A misnomer. Rearranging pieces of a puzzle to reveal a non-ischemic papillopathy caused by vitreous separation. Ophthalmology 122: 439-442.
10. Hayreh SS (2015) Re: Parsa et al.: Non-arteritic anterior ischemic optic Neuropathy: A misnomer. Rearranging pieces of a puzzle to reveal a non-ischemia papillopathy caused by vitreous separation. Ophthalmology 122: 439-442.
11. Cogan DG (1966) Neurology of the visual system. SpringŽeld 186.
12. Knox DL, Kerrison JB, Green WR (2000) Histopathologic studies of Ischemic Optic Neuropathy. Trans Am Ophth Soc 95: 203-220.