Introduction

Optic nerve is the second cranial nerve which is divided into 4 portions: an intracranial portion, an intracanalicular, an intraorbital and an intraocular portion. The optic nerve head is composed of the surface nerve fiber layer, the prelaminar region and the lamina cribrosa where nerve fibers become myelinated posterior to it. The optic disc is the exit site of all retinal ganglion cell axons and plays no perceptive function projected in visual field as an absolute scotoma, the blind spot of Marriotte **(Wolf, 1976)**.

Ischemic optic neuropathy (ION) is one of the major causes of blindness or seriously impaired vision, yet there is disagreement as to its pathogenesis, clinical features and especially its management. This is because ischemic optic neuropathy is not one disease but a spectrum of several different types, each with its own etiology, pathogenesis, clinical features and management. They cannot be lumped together. Ischemic optic neuropathy is primarily of two types: anterior (AION) and posterior (PION), involving the optic nerve head (ONH) and the rest of the optic nerve respectively.

Furthermore, both AION and PION have different subtypes. AION comprises arteritic (A-AION – due to giant cell arteritis) and, non-arteritic (NA-AION – due to causes other than giant cell arteritis); NA-AION can be further classified into classical NA-AION and incipient NA-AION. NA-AION is by far the most common type and one of the most prevalent and visually crippling diseases in the middle-aged and elderly. A-AION, though less

common, is an ocular emergency and requires early diagnosis and immediate treatment with systemic high dose corticosteroids to prevent further visual loss, which is entirely preventable.

Pathogenetically, AION and PION are very different diseases. PION has no specific location in the posterior part of the optic nerve and does not represent an ischemic disorder of any definite artery. Anterior ischemic optic neuropathy (AION) is one of the most prevalent and visually crippling diseases in the middle-aged and elderly population, although no age is immune, and is potentially bilateral **(Hayreh, 2009)**.

Controversy exists regarding the pathogenesis, clinical features and especially management of the various types of ischemic optic neuropathy because there are multiple misconceptions about its many fundamental aspects. Recently emerging information on the various factors that influence the optic nerve circulation, and also the various systemic and local risk factors which play important roles in the development of various types of ischemic optic neuropathy have given us a better understanding of their pathogeneses, clinical features and management. This knowledge should help us not only to manage better but also to reduce their incidence. **(Hayreh, 2009)**.

The main source of blood supply to the optic nerve head is from the posterior ciliary artery circulation **(Hayreh, 2001)**. AION is due to acute ischemia of the anterior part of the optic nerve (the optic nerve head) **(Luneau et al., 2008)**.Therefore, AION represents an ischemic disorder of

posterior ciliary artery circulation in the optic nerve head. The blood supply and blood flow patterns in the optic nerve head have a marked inter-individual variation which exercises a profound influence on the mechanism of development and clinical features of AION **(Hayreh, 1995)**.

AION present by prodromal symptoms either ocular or systemic which vary according to etiology whether arteritic or nonarteritic. Symptoms include blurring of vision and an altitudinal hemanopic defect. Photophobia, distorted colour vision and intermittent pain around the globe are usually in arteritic type while focusing difficulties, flickers and flashes are more common with nonarteritic type. Prodromal symptoms also include headache, systemic symptoms that vary according to cause and ocular symptoms as pain, sudden onset of deterioration of vision and loss of lower or central visual field **(Burde, 1993)**.

Diagnosis of those patients is through clinical examination by visual acuity, examination of the pupil, fundus examination and visual field changes (**Kline, 1988)**. Investigations of AION include Erythrocyte sedimentation rate (ESR), C-reactive protein and temporal artery biopsy which can confirm diagnosis of cases of arteritic AION. In nonarteritic type, special serological studies as fasting lipid profile and blood glucose may be of benefit. Other investigations include fluorescein angiography, electrophysiology, perimetry (**Miller, 1982)** and magnetic resonance imaging (MRI) (**Rizzo et al., 2002)**.

The mainstay in management of arteritic AION and giant cell arteritis is systemic corticosteroids either oral or intravenous megadoses without

waiting of temporal artery biopsy results. Management of non arteritic AION include Optic nerve sheath decompression, Systemic corticosteroid therapy, Aspirin**(Beck et al., 1997)** Use of intravitreal triamcinolone acetonide **(Jonas et al.,2007),** Use of intravitreal Bevacizumab (**Bennett et al., 2007)** and Other advocated treatments as the use of levodopa (**Johnson et al ., 2000)** and the use of Brimonidine eye drops. **(Wood et al., 2001)**