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Indian J Ophthalmol. 2011 Mar-Apr;59(2):123-36. doi: 10.4103/0301-

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Management of ischemic optic neuropathies.

Hayreh SS[1].

Author information

Abstract

Ischemic optic neuropathies (IONs) consist primarily of two types: anterior ischemic optic neuropathy (AION) and posterior ischemic optic neuropathy (PION). AION comprises arteritic AION (A-AION: due to giant cell arteritis) and non-arteritic AION (NA-AION: due to other causes). PION consists of arteritic PION (A-PION: due to giant cell arteritis), non-arteritic PION (NA-PION: due to other causes), and surgical PION (a complication of several systemic surgical procedures). These five types of ION are distinct clinical entities etiologically, pathogenetically, clinically and from the management point of view. In the management of AION, the first crucial step with patients aged 50 and over is to identify immediately whether it is arteritic or not because A-AION is an ophthalmic emergency and requires urgent treatment with high-dose steroid therapy to prevent any further visual loss in one or both eyes. Patients with NA-AION, when treated with systemic corticosteroid therapy within first 2 weeks of onset, had significantly better visual outcome than untreated ones. Systemic risk factors, particularly nocturnal arterial hypotension, play major roles in the development of NA-AION; management of them is essential in its prevention and management. NA-PION patients, when treated with high-dose systemic steroid therapy during the very early stages of the disease, showed significant improvement in visual acuity and visual fields, compared to untreated eyes. A-PION, like A-AION, requires urgent treatment with high-dose steroid therapy to prevent any further visual loss in one or both eyes. There is no satisfactory treatment for surgical PION, except to take prophylactic measures to prevent its development.

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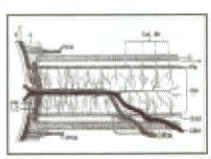
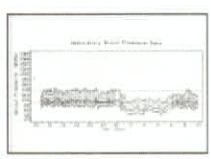
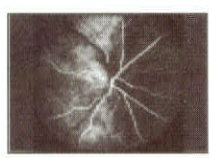
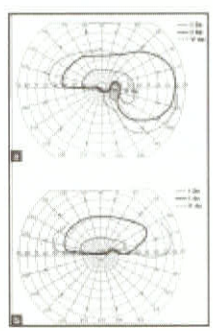
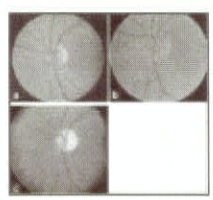

Extracts: (Prof. Hayreh)

A well-known neuro-ophthalmologist, while commenting on the role of corticosteroid therapy in NA-AION, stated: "Oral steroids in the setting of acute cerebral stroke are contraindicated". This statement is based on a serious misconception about pathogenesis of NA-AION.[74] Cerebral stroke is a thromboembolic disorder, involving a large mass of tissue in the cerebrum. In contrast to that, NA-AION is a hypotensive disorder, involving a minute amount of tissue in the optic nerve head. To equate the two conditions is a fundamental Mistake & responsible for confusion & controversy on various aspects of NA-AION, including its management.

Who, when and how to treat NA-AION patients with corticosteroid therapy? The sooner the treatment is started, the better are the chances of visual improvement. That may be because the shorter the duration of axonal ischemia, the fewer axons are likely to be damaged permanently. Secret of Corticosteroid Therapy Success. Over a period of almost five decades having treated several thousand patients with corticosteroid therapy for a variety of conditions, including giant cell arteritis, scleritis, uveitis, orbital myositis, retinal vasculitis and other conditions, I have found that the most effective way to use corticosteroid therapy is to hit hard at the beginning and then taper down. The major flaw in the way corticosteroid therapy has been given for NA-AION in some studies is "too small a dose, for too short a period".
This timidity has led to the prevailing misconception corticosteroid therapy does not help NA-AION

The usual advice given by ophthalmologist and neurologists to NA-AION patients is that nothing can be done. Having dealt with more than a thousand patients with NA-AION and having investigated various aspects of NA-AION over the years, I find that is an inadequate and incorrect response.
Firstly, because, as discussed above, systemic corticosteroid therapy during the early, acute stage of the disease has shown to be beneficial in visual outcome in a significant number of patients [73]. Secondly, as discussed above, NA- AION is a multifactorial disease and many risk factors contribute to it. The correct strategy is to try to reduce as many risk factors (discussed above) as possible to reduce the risk of NA-AION in the second eye or any further episode in the same eye. As discussed above, nocturnal arterial hypotension is a major risk factor in NA-AION patients who already have predisposing risk factors…

This strongly suggests that NA-AION may be emerging as an iatrogenic disease, stemming from the aggressive use of the very potent arterial hypotensive agents now available. In view of this, management of nocturnal arterial hypotension seems to be an important step both in the management of NA-AION and in the prevention of its development in the second eye. Therefore, I strongly recommend that when a patient is at risk of developing ocular and optic nerve head ischemic and vascular disorders, or has the following:..chronic optic disc edema due to any cause, the treating physician should be made aware of the potential risks of intensive arterial hypotensive therapy, particularly giving that drug in the evening. ..chronic optic disc edema due to any cause, the treating physician should be made aware of the potential risks of intensive arterial hypotensive therapy, particularly giving that drug in the evening.

If there is a reasonable index of suspicion of giant cell arteritis, as judged from systemic symptoms, high ESR and CRP (particularly high CRP) and sudden visual loss from A-AION or central retinal artery occlusion, high doses of systemic corticosteroid therapy, must be started IMMEDIATELY, as an EMERGENCY MEASURE. The physician should not wait for the result of the temporal artery biopsy because by the time it is available, the patient may have lost further vision irreversibly, in one or both eyes. Every minute counts; it is unwarranted to take chances of losing vision by starting with a small dose; once vision is lost, a subsequent higher dose will not restore it. the median starting oral Prednisone dose was 80 mg/day, with 40% of patients on >100 mg/day. Risk/benefit ratio of steroid therapy in giant cell arteritis patents:The concept that systemic steroid therapy is dangerous and must be given in the lowest possible dose and for the minimum period is over-stated….but most of them are either tolerable or easily manageable once the patient is fully aware of the alternative, i.e. the risk of going blind in one or both eyes. Most importantly, once a patient is made aware of the choice between side-effects of steroid therapy versus the risk of going blind (fear of going blind is next to fear of death), he/she will always choose the therapy, even at the risk of a certain amount of side-effects –
I have yet to find a single patient in more than 40 years of dealing with giant cell arteritis patients who opted against steroid therapy and took the risk of going Blind. To reiterate: in my experience of dealing with several hundred giant cell arteritis patients for about four decades, I have found that if they are treated promptly and aggressively with an adequate dose of corticosteroids, …, not a single patient suffered any further visual loss 5 days after starting adequate steroid therapy.. testimony to the effectiveness of my steroid therapy regimen.

I SHOWED HIM THESE - AND, HE STILL REFUSED TO HELP ME! WHY?

Extracts:

(2) 2/4

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FOR MOD PATROL (2 3/4

ttps://en.wikipedia.org/wiki/Anterior_ischemic_optic_neuropathy#Causes_and_risk_factors

Causes and risk factors.

The mechanism of injury for NAION used to be quite controversial. However, experts in the field have come to a consensus that most cases involve two main risk factors. The first is a predisposition in the form of a type of optic disc shape. The optic disc is where the axons from the retinal ganglion cells collect into the optic nerve. The optic nerve is the bundle of axons that carry the visual signals from the eye to the brain. This optic nerve must penetrate through the wall of the eye, and the hole to accommodate this is usually 20-30% larger than the nerve diameter. In some patients the optic nerve is nearly as large as the opening in the back of the eye, and the optic disc appears "crowded" when seen by ophthalmoscopy. A crowded disc is also referred to as a "disc at risk". While a risk factor, the vast majority of individuals with crowded discs do not experience NAION. The second major risk factor involves more general cardiovascular risk factors. The most common are diabetes hypertension and high cholesterol levels. While these factors predispose a patient to develop NAION, the most common precipitating factor is marked fall of blood pressure during sleep (nocturnal arterial hypotension) - that is why at least 75% of the patients first discover visual loss first on waking from sleep. These vascular risk factors lead to ischemia (poor blood supply) to a portion of the optic disc. The disc then swells, and in a crowded optic disc, this leads to compression and more ischemia.[citation] Since both eyes tend to have a similar shape, the optometrist or ophthalmologist will look at the good eye to assess the anatomical predisposition. The unaffected eye has a 14.7% risk of NAION within five years.[5]

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http://parade.com/47774/parade/aspirin-and-naion/

The vision loss is usually the result of some underlying cardiovascular disease, so it's important to control any cardiovascular risk factors (high blood pressure, elevated cholesterol, smoking, diabetes, obesity and lack of physical activity). Aspirin is appropriate for someone with this condition just as it is for anyone who either has cardiovascular disease or is vulnerable to it. There have been a few published studies of aspirin therapy in NAION; all are flawed, but they suggest that aspirin helps to prevent NAION in the second eye. There also appears to be a connection between NAION and sleep apnea. Some patients have reported the vision loss upon awakening, which has led researchers to theorize that the normal drop in blood pressure during the night may be a precipitating factor for developing the condition. (Avoid taking any meds. that can lower blood pressure right before bedtime.) In terms of what's new, surgery to increase the blood flow to the optic nerve has been unsuccessful in patients with NAION. However,…

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http://www.reviewofoptometry.com/content/d/case_report/c/28776/

From an etiological and pathogenic perspective, NAION presents in one of two forms:
—**Transient non-perfusion or hypoperfusion of the optic nerve head.**
—**Embolic lesion of the arteries/arterioles that feed the optic nerve head.**
Transient non-perfusion or hypoperfusion is the most common cause of NAION. The majority of NAION cases are due to transient reductions in blood pressure during sleep.[2] This explains why approximately 75% of patients with NAION report a sudden loss of vision upon awakening.[4] Less common causes of hypoperfusion include transient reductions in blood pressure secondary to shock and/or a sharp increase in IOP. Embolic causes of NAION are less likely, but when compared to NAION as a result of hypotensive events, the extent of optic nerve head damage is much more extensive and severe.[2] …..
… *Treatment.* Treatments for NAION are minimal and unsubstantiated at best.[2,4-6] include.. Anticoagulants, oral corticosteroids and, more recently, intravitreal triamcinolone acetonide.[1,2,10] Triamcinolone acetonide injections have been shown to decrease the duration of disc edema while increasing the prospect of a recovery in visual acuity, but not in visual field loss.[10] ……. …..
The primary treatment modality for NAION consists of 80mg to 325mg of aspirin q.d.[5,6] This treatment modality is controversial, however, because although aspirin has well documented effects on thromboembolic conditions, NAION is more often a hypotensive condition.[2] Furthermore, a few studies have shown that aspirin neither benefits eyes with NAION nor prevents the development of NAION in an unaffected eyes.[2] Consequently, due to a lack of viable and effective treatments for clinicians who manage these patients, the primary goal is to rule out an AAION, detect and preemptively control vascular risk factors in the hope of thwarting disease progression and/or occurrence in the contralateral eye (e.g., avoid steps to reduce the likelihood of nocturnal hypotension via consultation with primary care provider to eliminate q.h.s. anti-hypertensive medications).[4,10]

ttp://eyewiki.aao.org/Non-Arteritic_Anterior_Ischemic_Optic_Neuropathy_(NAION)

Nocturnal hypotension

There are normal nocturnal fluctuations in blood pressure and (prof.) Hayreh has theorized that nocturnal systemic hypotension may contribute to NAION. Patients with chronically altered optic disc auto-regulation, from such diseases as systemic hypertension and atherosclerosis, might be susceptible to exaggerated decreases in nocturnal blood pressure. This effect might be exacerbated in patients treated with aggressive antihypertensive therapy, especially if taken right **before bedtime.** 38.

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http://www.ncbi.nlm.nih.gov/pubmed/21350282

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…

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www.medicine.uiowa.edu/eye/AION-part2/

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In sharp contrast to that, my study [58] showed this to be a dangerous formula to prevent blindness…….in my experience of dealing with several hundred giant cell arteritis patients for about four decades, I have found that if they are treated promptly and aggressively with an adequate dose of corticosteroids, and reduction of steroid therapy is regulated by using ESR and CRP as the only criteria, not a single patient suffered any further visual loss.. 5 days after starting adequate steroid therapy…
testimony to the effectiveness of my steroid therapy regimen.

CONCLUSION.

These are just a sample of the *massive evidential support* for NOT applying Lumigan eye drops at ***bedtime***. As prescribed for me by Dr Kulshreska. ***Repeatedly***, telling me one drop at ***bedtime***. (Not evening) Further:

There is *known evidence* that Prednisone steroid therapy could have restored my sight if applied in 14 days. When blinded, by his treatment of **bedtime** drops, he said again *repeatedly* there was "***NO** treatment for it.*" When I appealed to him for ***any*** remedial treatment, he admits to "*making a clinical decision*" - to **deny** me this, and any other treatment. This treatment was denied to me **within the 14 day window of opportunity.** How any one may wish to construe my complaint, the above is FACT. Damaging destructive **malpractice**. All the detailed damage upon me was at **best**, incompetent. When from a trusted doctor, it's based in **evil.**

Sample 1 5 Pages.

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Management of ischemic optic neuropathies.

EXTRACT (File 18 1" THICK)

Hayreh SS[1].

Author information

Abstract

Ischemic optic neuropathies (IONs) consist primarily of two types: anterior ischemic optic neuropathy (AION) and posterior ischemic optic neuropathy (PION). AION comprises arteritic AION (A-AION: due to giant cell arteritis) and non-arteritic AION (NA-AION: due to other causes). PION consists of arteritic PION (A-PION: due to giant cell arteritis), non-arteritic PION (NA-PION: due to other causes), and surgical PION (a complication of several systemic surgical procedures). These five types of ION are distinct clinical entities etiologically, pathogenetically, clinically and from the management point of view. In the management of AION, the first crucial step with patients aged 50 and over is to identify immediately whether it is arteritic or not because A-AION is an ophthalmic emergency and requires urgent treatment with high-dose steroid therapy to prevent any further visual loss in one or both eyes. Patients with NA-AION, when treated with systemic corticosteroid therapy within first 2 weeks of onset, had significantly better visual outcome than untreated ones. Systemic risk factors, particularly nocturnal arterial hypotension, play major roles in the development of NA-AION; management of them is essential in its prevention and management. NA-PION patients, when treated with high-dose systemic steroid therapy during the very early stages of the disease, showed significant improvement in visual acuity and visual fields, compared to untreated eyes. A-PION, like A-AION, requires urgent treatment with high-dose steroid therapy to prevent any further visual loss in one or both eyes. There is no satisfactory treatment for surgical PION, except to take prophylactic measures to prevent its development.

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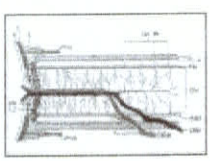
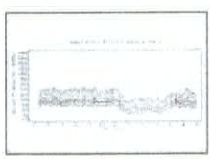
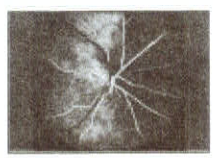

4. ***Early, massive visual loss:*** This is extremely suggestive of PA-AION. However, the presence of perfectly normal visual acuity does not rule out A-AION (see above).
5. ***Chalky white optic disc edema*** (Figs. 17, 18, 19-B, 20-A)***: This is almost diagnostic of A-AION*** and is seen in 69% of A-AION eyes. In NA-AION, chalky white optic disc edema occurs only very rarely with embolic occlusion of the posterior ciliary artery.
6. ***A-AION associated with cilioretinal artery occlusion*** (Fig. 20)***: This is almost diagnostic of A-AION.***
7. ***Evidence of posterior ciliary artery occlusion on fluorescein fundus angiography*** (Figs. 15-B,C, 20-B)***:*** If angiography is performed during the first few days after the onset of A-AION, and the choroid supplied by one or more of the posterior ciliary artery does not fill, ***this once again is almost diagnostic of A-AION***. However, later on, this information may be lost.
8. ***Temporal artery biopsy:*** This finally establishes the diagnosis and its role has been discussed above

EXTRACT (FILE 18 1/4 THICK)

Steroid therapy in giant cell arteritis to prevent blindness

A detailed discussion of this is extremely important in the interest of prevention of visual loss due to giant cell arteritis. This is a highly controversial subject; especially since all the available information is from the rheumatological literature. As mentioned above, there is a differing perspective on giant cell arteritis between rheumatologists and ophthalmologists, which has influenced their recommendations on steroid therapy – the regimen advocated by the former primarily concerns managing benign rheumatologic symptoms and signs, whereas the latter confronts the probability of blindness [58]. Moreover, I have found that rheumatologists often tend not to differentiate between polymyalgia rheumatica and giant cell arteritis in their management. A regimen of steroid therapy, which is adequate to control rheumatologic symptoms and signs and polymyalgia rheumatica, is often totally inadequate to prevent blindness associated with giant cell arteritis. With this in view, I did a 27-year prospective study [58] on steroid therapy in giant cell arteritis, to find a regimen that would prevent visual loss. That study showed marked differences between the rheumatologic and ophthalmic steroid therapy regimens. In the light of information from that study, the following are my guidelines to prevent visual loss.

a. If there is a reasonable index of suspicion of giant cell arteritis, as judged from systemic symptoms, high ESR and CRP (particularly high CRP) and sudden visual loss from A-AION or central retinal artery occlusion, high doses of systemic corticosteroid therapy, must be started **IMMEDIATELY, as an EMERGENCY MEASURE**. ***The physician should not wait for the result of the temporal artery biopsy*** because by the time it is available, the patient may have lost further vision irreversibly, in one or both eyes. Every minute counts; it is unwarranted to take chances of losing vision by starting with a small dose; once vision is lost, a subsequent higher dose will not restore it. In my study, the median starting oral Prednisone dose was 80 mg/day, with 40% of patients on ≥100 mg/day.
b. A high-dose steroid therapy must be maintained until both the ESR and CRP settle down to a stable level which usually takes 2-3 weeks – CRP usually settles much earlier than the ESR (Fig. 23).
c. After that, gradual tapering down of steroid therapy should be started. Recently, Salvarani and colleagues [89] stated that 2–4 weeks after the start of initial dose, "the dose can be gradually reduced each week or every 2 weeks by a maximum of 10% of the total daily dose." In sharp contrast to that, my study [58] showed this to be a dangerous formula to prevent blindness. According to my study, ***a titration of the steroid dosage with the levels of ESR and CRP is the only safe and reliable method for tapering down and follow-up of steroid therapy.*** Using clinical symptoms and signs of giant cell arteritis as a guide (as recommended by rheumatologists) is a dangerous practice to prevent blindness [50]. In my

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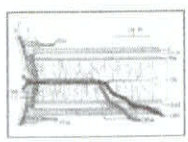

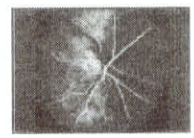
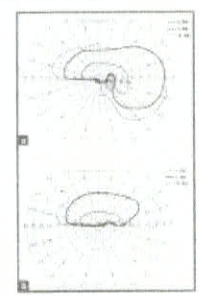
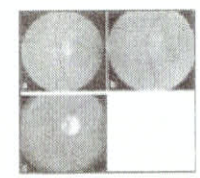
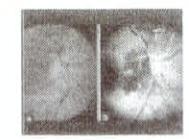

MeSH terms, Substance

Cómo hablarle a su oculista (/healthyeyes/spanish/oculista_sp)

What You Should Know

Does increased eye pressure mean that I have glaucoma?

Not necessarily. Increased eye pressure means you are at risk for glaucoma, but does not mean you have the disease. A person has glaucoma only if the optic nerve is damaged. If you have increased eye pressure but no damage to the optic nerve, you do not have glaucoma. However, you are at risk. Follow the advice of your eye care professional.

Can I develop glaucoma if I have increased eye pressure?

Not necessarily. Not every person with increased eye pressure will develop glaucoma. Some people can tolerate higher eye pressure better than others. Also, a certain level of eye pressure may be high for one person but normal for another.

Whether you develop glaucoma depends on the level of pressure your optic nerve can tolerate without being damaged. This level is different for each person. That's why a comprehensive dilated eye exam is very important. It can help your eye care professional determine what level of eye pressure is normal for you.

Can I develop glaucoma without an increase in my eye pressure?

Yes. Glaucoma can develop without increased eye pressure. This form of glaucoma is called low-tension or normal-tension glaucoma. It is not as common as open-angle glaucoma.

Who is at risk for open-angle glaucoma?

Anyone can develop glaucoma. Some people are at higher risk than others. They include the following:

- African Americans over age 40
- Everyone over age 60, especially Mexican Americans
- People with a family history of glaucoma.

A comprehensive dilated eye exam can reveal more risk factors, such as high eye pressure, thinness of the cornea, and abnormal optic nerve anatomy. In some people with certain combinations of these high-risk factors, medicines in the form of eyedrops reduce the risk of developing glaucoma by about half.

> "A comprehensive dilated eye exam can reveal risk factors, such as high eye pressure, thinness of the cornea, and abnormal optic nerve anatomy."