

Mistaken Diagnosis of Optic Neuritis and the Possible Role of Phosphodiesterase-5 Inhibitors (Sildenafil / Viagra)

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SUMMARY

The diagnosis of optic neuritis and particularly retrobulbar optic neuritis when atypical and not responsive to corticosteroid treatment may need to be revised. This is now especially so in male patients who should be questioned regarding their taking a phosphodiesterase –5 inhibitor in particular Viagra. The case history of such a patient is presented who sustained posterior ischaemic optic neuropathy mistaken for retrobulbar neuritis resulting in bilateral severe visual loss.

KEY WORDS:

optic neuritis, retrobulbar neuritis, ischaemic optic neuropathy, posterior ischaemic optic neuropathy, phosphodiesterase-5 inhibitors, Viagra.

INTRODUCTION:

Optic neuritis (ON) in Singapore and probably throughout Asia is more likely to be of the anterior variety with optic disc swelling rather than retrobulbar (RBN)⁽¹⁾ where the disc is normal initially but will become pale 4 to 6 weeks later. The aphorism of David Cogan⁽²⁾ in respect of this diagnosis is still relevant today when he reminded us that “probably no branch of neuro-ophthalmology has to its discredit the abundance of erroneous diagnoses as has optic neuritis”.

Anterior ischaemic optic neuropathy (AION) which presents with a hyperaemic swollen disc is on the other hand the commonest optic neuropathy encountered here and the commonest cause of irreversible, but fortunately incomplete, visual loss in our elderly population⁽³⁾. The much rarer posterior ischaemic variety (PION) presents with a normal optic disc and is a diagnosis of exclusion⁽³⁾. Furthermore giant cell arteritis (GCA) as a causative agent of either variety is extremely rare in the region⁽⁴⁾.

The case history presented below emphasizes the importance of reconsidering a diagnosis of ON or RBN in atypical cases, and where spontaneous recovery or response to corticosteroid treatment does not take place.

CASE REPORT:

A 57 year-old Chinese man presented to a hospital eye clinic in the Klang Valley area Malaysia on 16 February 2009 with sudden painless loss of vision in the left eye of two days duration and similar visual loss in his second eye the following morning. Apart from a history of hypertension for which he was taking a beta blocker Atenolol he was well, denied previous trauma or having taking any other medication, traditional or prescribed. His vision was reduced to bare perception of hand

movements (HM's) in the left eye and counting fingers (CF's) closely in the right with sluggish pupillary reaction. The rest of his eye examination was normal as was a full neurological examination. As his optic discs were normal a diagnosis of bilateral retrobulbar neuritis (RBN) was made and he was treated with IV methyl prednisolone followed by oral corticosteroids in accordance with the optic neuritis treatment trial (ONTT) protocol. His vision did not improve on treatment and he did not reattend on discharge from hospital. All investigations including MRI scans of the brain and orbits were normal.

Three months later he came to the Singapore National Eye Centre (SNEC) via our international patient service for a further opinion and was directed to our neuro-ophthalmology clinic where he was seen by the senior author (JFC) of this paper. He was accompanied, or in fact led in by his wife (who incidentally looked somewhat younger than the patient) and appeared blind. Vision was CF's right eye and HM's left eye with a right sluggish pupil and an inactive left pupil with a relative afferent defect. Both discs were atrophic. The right visual field (Goldmann) was reduced all round and the left showed only a tiny island of vision nasally (Fig). The diagnosis of bilateral RBN did not appear likely and he was again questioned regarding a now suspected toxic cause, which he denied.

A decision was made to see the patient again next day so that the possible diagnosis could be thought through overnight, but on recalling the family situation and the younger wife along with the unexplained severe visual loss, the possibility of a toxic aetiology had to be reconsidered. The possibility of the use of a phosphodiesterase inhibitor had also to be investigated which might explain this perplexing problem. Clearly in such circumstances delicate further enquiries were called for so the patient was phoned early in the morning before he came to the clinic next day and was diplomatically questioned about Sildenafil (Viagra) or another such substance having been prescribed or self administered prior to his visual loss.

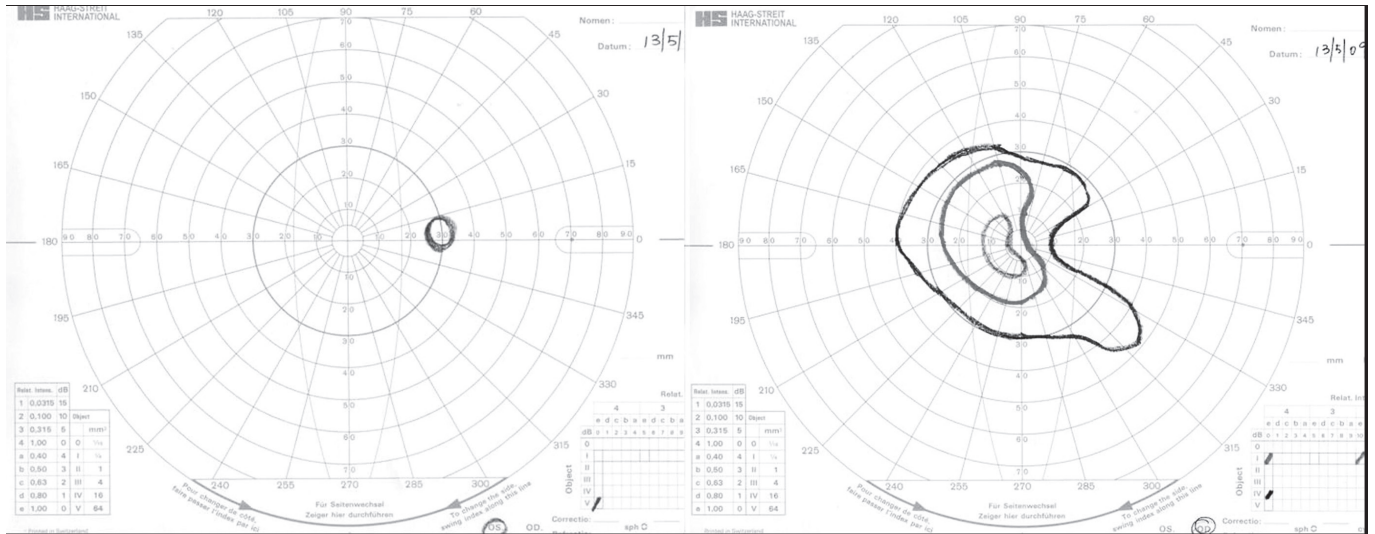
It transpired that the patient had been taking “Viagra” daily for two weeks prior to the visual loss and intermittently 2/3 times weekly for about 2 years usually in a dose of 25 mg daily. It was always taken in addition to his Atenolol. Thus the most likely diagnosis was bilateral ischaemic optic neuropathy of the posterior variety (PION) as the optic discs were normal at presentation and now atrophic after three months

DISCUSSION:

Non-arteritic anterior ischaemic optic neuropathy (NA-AION), our commonest optic neuropathy which is

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Goldmann visual field charts showing substantial generalized constriction of the right field and only a tiny island of vision remaining in the left (normal Goldmann field extends to 90 degrees temporally, and to around 50 degrees nasally, up and down).

associated with vascular risk factors especially diabetes and hypertension, has also been reported following the use of phosphodiesterase-5 inhibitors (PDE5) especially Viagra⁽⁵⁾. These drugs are known to have a hypotensive effect but their actual role in initiating the optic nerve head perfusion loss and subsequent infarction ie a NA-AION is not entirely clear, the blood supply of the optic nerve head being via the posterior ciliary circulation. The problem appears to develop in already predisposed patients taking antihypertensive treatment along with nocturnal arterial hypotension and the ingestion of Viagra or another PDE5 substance at nighttime.

Case reports of posterior ischaemic optic neuropathy (PION) are rarely encountered in the literature and the condition is usually reported to occur following major surgery or be associated with GCA. The subject has been comprehensively reviewed by Hayreh⁽⁶⁾. The occurrence of PION associated with Viagra has also been reported⁽⁷⁾ and is even more difficult to explain because the posterior optic nerve unlike the optic nerve head has an abundant blood supply via its surrounding pial plexus which is fed by the many small branches of the main ophthalmic artery. Extensive loss of perfusion in this system and substantial infarction of the nerve would be required to produce the extreme loss of vision reported above and in another case reported from our department where Viagra was unintentionally ingested in a Chinese "health product" which on analysis contained a large amount of PDE5 and the unfortunate patient lost all vision in both eyes⁽⁷⁾.

CONCLUSION:

In view of the widespread use of phosphodiesterase-5 inhibitors especially Viagra all general practitioners, physicians and ophthalmologists should be aware of their potential dangerous side effects in particular permanent loss of vision and even total blindness in patients already at risk of ocular vascular disease. The diagnosis of our two commonest optic neuropathies namely optic neuritis and ischaemic optic neuropathy in male patients if not typical must therefore be viewed with suspicion and among other investigations, diplomatic enquiries be made regarding the possible use of a PDE5 agent.

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