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OPHTHALMOLOGY
Case Report

Retinal Oximetry Findings in a Case of Bilateral Optic Disc Edema and Visual Loss

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Abstract

Retinal oximetry in bilateral optic disc edema after accidental minoxidil ingestion

Aim: To study the oximetry changes in bilateral optic disc edema

Methods: A 56 year old man, presenting with painless, bilateral, simultaneous visual loss and a history of accidental ingestion of topical Minoxidil 2% 20 days before presentation; diagnosed to have bilateral optic disc edema on clinical examination underwent spectral domain optical coherence tomography (SD-OCT) and retinal oximetry (Oxymap T1, Oxymap hf, Reykjavik, Iceland).

Results: The RNFL analysis (SD-OCT, Spectralis, Heidelberg, Germany) showed thickening bilaterally (Global peripapillary retinal nerve fiber layer [RNFL] thickness – OD – 175 µm; OS – 172 µm; Normal – 96 µm). Oximetry was done on the Oxymap T1 (Oxymap hf, Reykjavik, Iceland). The right eye showed arteriolar saturations (%) of 101, 97, 101 and 88 and venous saturations (%) of 46, 52,

Conclusion: Arteriolar saturations were increased, venous saturations remained close to normal and arterio-venous saturation difference had increased bilaterally in our case.

Summary statement: A 56 year old man with a history of painless bilateral simultaneous visual loss and disc edema after accidental ingestion of minoxidil 2% underwent retinal oximetry which revealed high arteriolar saturations, low venous saturations and a higher artery-venous saturation difference.

Keywords: *Oximetry; Minoxidil; Optic disc edema*

Introduction

Retinal oximetry is a non-invasive photo-spectrometric method of measuring oxygen saturations in the retinal arterioles and venules [1]. Since oxygen plays a key role in the metabolism and functioning of tissue, derangements in the physiological oxygen supply can result in disease pathology, especially in a tissue as highly metabolically active as the retina [2].

Bilateral optic neuritis has been previously reported following accidental systemic ingestion of minoxidil [3]. We present a case presenting with sudden bilateral visual loss and optic disc edema noticed after accidental minoxidil ingestion. Studying the oxygen saturation profiles and correlating them with clinical and other investigative findings may enable us to better understand the underlying disease processes.

Case report

A 56 year old man, presented with painless, bilateral, simultaneous visual loss. An informed written consent was obtained from the patient and follows the tenets of Declaration of Helsinki. He gave history of accidental ingestion of topical Minoxidil 2% w/w (Mintop, Dr.Reddy's, ethanol 63% v/v) 20 days before presentation, following which he had severe vertigo and loss of consciousness. He was shifted to a hospital, and on admission the blood pressure was found to be 80/40 mm Hg. He was treated with IV fluids. There was no documentation of visual acuity testing.

After IV hydration and improvement of blood pressure to 110/70 mm Hg, he awoke with bilateral vision loss, which has gradually improved since then till presentation to us. There was no report of systemic sepsis, uremia or signs of kidney failure. The patient is not a known diabetic or hypertensive. Humphrey visual fields (30-2 SITA Standard), done elsewhere revealed bilateral reduction of sensitivity (Mean deviation -8.5 db OD, -10.2 db OS). There was no documentation of ophthalmic evaluation or retinal findings. The patients confirmed that no ocular treatment was administered.

The patient gave a history of gradual improvement in vision since the diminution 20 days back. On examination, he had best corrected visual acuity of 6/9 bilaterally and a left relative afferent pupillary defect. His color vision was normal (16/16 tested on Ishihara) in both eyes. Intra-ocular pressures were 16mm Hg in both eyes by Non Contact Tonometry. On fundus examination he was found to have both eyes disc edema. The RNFL analysis (SD-OCT, Spectralis, Heidelberg, Germany) showed thickening bilaterally (Global peripapillary retinal nerve fiber layer [RNFL] thickness – OD – 175 µm; OS – 172 µm; Normal – 96 µm) (Figure 1).

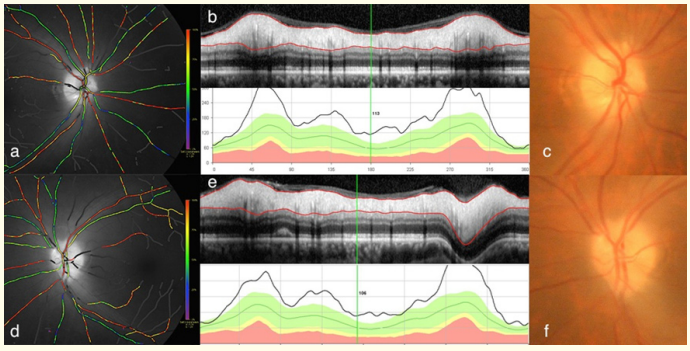

Figure 1: *a & d – 570 nm image with the pseudocolour map showing retinal (oxygen saturations of the right and left eye respectively; b & e – SD-OCT image of (the RNFL thickness (above) with the line graph showing comparison with (normal (below) for the right and left eye respectively; c & f – colour fundus photo showing the right and left optic disc with surrounding retinal nerve fiber layer).*

Oximetry was done on the Oxymap T1 as described by Hardarson., *et al*. [1].The right eye showed arteriolar saturations (%) of 101, 97, 101 and 88 and diameters (µm) of 131, 101, 98 and 142 in the supero-temporal (ST), supero-nasal (SN), infero-nasal (IN) and infero-temporal (IT) quadrants respectively. The venous saturations (%) were 46, 52, 48 and 36; diameters were 158, 130, 132 and 137 in the ST, SN, IN and IT quadrants respectively. The left arteriolar saturations were 106, 99, 94 and 96; diameters were 110, 123, 97 and 143; venous saturations were 63, 47, 55 and 43; diameters were 150, 173, 145 and 167 in the ST, SN, IN and IT respectively. Normal values can be found in our other study [4].

Discussion

The patient had bilateral optic disc edema with visual loss that improved over a period of 20 days to 6/9. We thought about 2 main differential diagnoses for this patient. The first was bilateral non-arteritic anterior ischemic optic neuropathy (NAION) and the second was bilateral toxic optic neuropathy.

The patient had severe hypotension and was admitted to the hospital. Ischemic optic neuropathy (ION) may develop in settings of hemodynamic compromise, such as systemic hypotension, blood loss and anemia. Prolonged immobilization and orthostatic hypotension can also cause ION and visual loss, bilateral optic disc edema and peripapillary fame shaped hemorrhages [5]. Some studies have shown recovery of 3 lines of acuity or better in 13% to 42.7% [6].

Minoxidil (6-amino-1,2-dihydro-1-hydroxy-2imino- 4-piperidinopyrimidine) is a direct smooth muscle vasodilator used in the treatment of refractory hypertension. The parent compound is hepatically transformed to an active metabolite, minoxidil N-O sulfate, which

possesses the primary vasodilating activity, presumably by enhancing potassium influx at smooth muscle cells of the arteriolar beds [7]. The nitric oxide group could play an important role in the therapeutic effects of the drug [8]. Nitric oxide can be neurodestructive or cause auto-regulatory failure of the local optic nerve head circulation [9].

In therapeutic doses, orally administered minoxidil is rapidly and nearly completely absorbed from the gastrointestinal tract, reaching peak plasma concentrations within 1 hour post-ingestion [7].

Minoxidil can also cause optic neuritis by inducing peroxides in the retinal vasculature [3].

The minoxidil preparation contains alcohol 63% v/v. The possibility of this chemical independently causing toxicity to the optic nerve head cannot be ruled out.

Hence it is difficult to point out the exact cause for the acute, painless, simultaneous bilateral vision loss and optic nerve head edema. It could be a non arteritic ischemic cause due to the systemic hemodynamic compromise or local auto-regulatory failure due to nitric oxide. It could also be toxic due to the peroxidases in minoxidil or due to the ethanol present in the lotion.

We found that there was an increased arteriolar saturation, decreased venous saturations and a wider arterio-venous saturation difference (AVSD). The higher arteriolar saturation could be explained by the lack of diffusion from the arterioles due to the gross retinal nerve fiber layer edema. Repair of locally damaged tissue can increase the metabolic demand and hence result in greater oxygen utilization and thus a lower venous saturation and a wider AVSD.

In our normative database [4] of 98 subjects (18-63 years), we found that the average arteriolar saturations were 90% and diameters were 123 μm; venous saturations were 57% and diameters were 167 μm. The average AVSD was 33%.

In this patient we also found increased diameters. This could be attributed to locally accumulated nitric oxide that is causing vascular dilation.

The above correlation with oximetry findings is our own hypothesis based on an anecdotal observation of a single patient. A larger case series would enable us to ascertain the oximetry findings and identify specific disease processes against which future therapy can be targeted.

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