American Journal of Ophthalmology Case Reports 25 (2022) 101373

Contents lists available at ScienceDirect

American Journal of Ophthalmology Case Reports

journal homepage: www.ajocasereports.com/

Recovery of vision after treatment of hemodialysis related bilateral optic nerve ischemia

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ARTICLE INFO

Keywords:
Posterior ischemic optic neuropathy
End-stage renal disease
Diabetes mellitus
Erythropoietin
Visual loss
Hemodialysis

ABSTRACT

Purpose: We present the case of a patient who lost light perception in both eyes after hemodialysis and subsequently recovered vision after treatment with erythropoietin and intravenous steroids.

Observations: Our patient reported loss of light perception in both eyes (NLP) 2 hours after hemodialysis. Examination confirmed NLP vision, chronic retinal vascular changes, and no acute changes in optic nerve appearance. A presumptive diagnosis of posterior optic neuropathy was made. The patient was treated with erythropoietin and intravenous steroids according to the protocol of Nikkah. Over a period of 14 hours, he recovered vision to his baseline.

Conclusions and Importance: Bilateral loss of light perception is a rare complication of hemodialysis. The presumed mechanism is posterior ischemic optic neuropathy. Prompt treatment with erythropoietin and intravenous steroids should be considered in similar situations that result in Posterior ischemic optic neuropathy (PION) related to procedure-based hypotension.

1. Introduction

Posterior ischemic optic neuropathy (PION) – ischemic damage to the retrobulbar optic nerve – presents as severe acute painless unilateral or bilateral loss of vision. It is distinguished from anterior ischemic optic neuropathy (AION) by the lack of the optic nerve edema that characterizes that more common condition.[1] The blood supply to the retrobulbar optic nerve arises primarily arises from the pial capillary plexus and is distinct from the blood supply of the optic nerve head.[2,3]

Causes of PION are usefully divided into arteritic (associated with giant cell or other vasculitides) and non-arteritic. Non-arteritic PION may be divided further into spontaneous cases associated with typical vasculopathic risk factors – hypertension, hyperlipidemia, diabetes, and smoking – and cases more clearly associated with peri-procedural hypoperfusion.[1,2,4] Spinal surgery is most commonly linked to PION. Any procedure that results in sufficient optic nerve hypoperfusion due to, for instance, intentional hypotension, intraoperative blood loss or prone positioning can cause retrobulbar optic nerve damage. Although PION due to hemodialysis is rare, it has previously been reported in a patient with a history of hypertension, chronic kidney failure, and gout. This patient had periprocedural hypotension and anemia. Computer Tomography (CT) and Magnetic Resonance Imaging (MRI)/Magnetic Resonance Angiography (MRA) of the head were normal.[4,5]

While there are reports of successful cases of treatment with hyperbaric oxygen treatment and steroids, there is no established treatment protocol and the prognosis for recovery of vision in patients with periprocedural PION is poor.

2. Case report

A 34-year-old male patient with a past medical history of type 1 diabetes since the age of 17 years, end-stage renal disease on hemodialysis, and diabetic gastroparesis presented to the emergency department with respiratory complaints and hypertensive emergency. His past ocular history was significant for bilateral proliferative retinopathy, bilateral epiretinal membrane, and pars plana vitrectomy and endolaser on both eyes for non-resolving vitreous hemorrhage. He had missed several scheduled hemodialysis sessions. He was admitted for blood pressure control, oxygen management and hemodialysis. Baseline visual acuity was 20/40 in the right eye and 20/150 in the left eye. The patient

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https://doi.org/10.1016/j.ajoc.2022.101373
Received 11 March 2021; Received in revised form 15 November 2021; Accepted 27 January 2022
Available online 29 January 2022

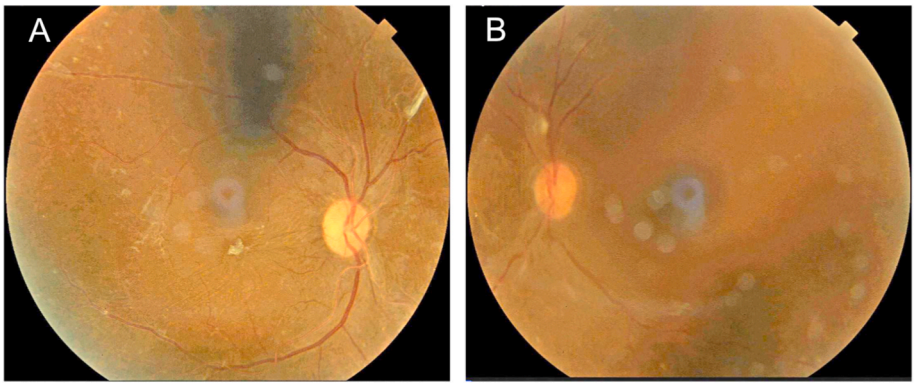

Fig. 1. Fundus photography of the right and left eyes, respectively. The retina shows a macular epiretinal membrane, normal optic nerve, sclerosed vessels with arteriolar narrowing can be seen throughout, more prominent inferotemporal and superonasal (A). Similar findings are seen on the left eye, with details being blurred due to the presence of a prominent cataract (B).

underwent emergent hemodialysis treatment overnight with ultrafiltration of 3 L the starting blood pressure was 224/135 mmHg, lowest blood pressure was 177/112 mmHg. He underwent a subsequent hemodialysis treatment with additional ultrafiltration of 3 L the following morning. Starting blood pressure was 128/75 mmHg, and Lowest intradialytic blood pressure was 117/73 mmHg.

Shortly after the second hemodialysis treatment, he complained of bilateral painless complete loss of vision. On examination he was found to have no light perception in either eye. Pupils were 3 mm and amaurotic. A dilated fundus exam showed perfused-appearing optic nerves, attenuated retinal arterioles and moderate panretinal photocoagulation (PRP) laser scars (Fig. 1). Aside from loss of vision he had no focal neurologic findings. A presumptive diagnosis of bilateral posterior ischemic optic neuropathy (PION) was made. After review with the internal medicine, nephrology, and ophthalmology services, the patient was treated with EPO 10,000 units BID and prednisone 75 mg for 3 days according to the protocol suggested by Nikkah et al. for non-arteritic anterior ischemic optic neuropathy. Examination 14 hours after the first dose showed marked clinical improvement, with a vision of 20/50 in the right eye and count fingers (CF) at face in the left eye. A CT head without contrast that day demonstrated no pathology. A subsequent ultrasound did not show stenosis of the carotid or vertebral arteries. Acuity subsequently stabilized to 20/40 in the right eye and CF 3' in the left eye. The patient underwent two more hemodialysis sessions with careful attention to avoiding hypotension and had no subsequent vision loss. When he was seen in the eye clinic four days after discharge, the visual acuity remained stable.

3. Discussion

As mentioned above, the prognosis for post-procedural non-arteritic PION is poor and there are no standardized treatments. Two prospective trials have shown a potential benefit of treatment of PION with EPO when it is started within 5 days of onset. The more robust trial comes from Nikkah et al., in which 10,000 units of EPO twice a day for three days showed superiority to steroids and placebo in the final VA in patients with ischemic optic neuropathy.[6,7]

The use of recombinant erythropoietin was FDA-approved in 1989. Indications for its use include anemia in chronic kidney disease both on and not on dialysis, effects of concomitant myelosuppressive chemotherapy, and reduction of allogeneic RBC transfusions in patients undergoing elective, non-cardiac, non-vascular surgery.[8] In the dialysis population, the major goal of treating anemia with erythropoiesis-stimulating agent (ESA) therapy is to prevent symptoms of anemia including fatigue, dyspnea, and decreased exercise tolerance, as well as to reduce the need for blood transfusions.[9] Erythropoietin is generally well tolerated when used according to established protocols.[10,11] Risks of use include worsening of hypertension and thromboembolic events particularly when high targets are set for hemoglobin.[9,11]

Ischemic events of the posterior optic nerve are caused by decreased perfusion to the pial vessels supplying the posterior portion of the optic nerve. These vessels are more sensitive to decreased oxygen supply. Because pial vessels are less affected by anatomical constriction, ischemic events take longer to produce permanent damage, and it is thought that prompt treatment in restoring perfusion by controlling blood pressure and increasing red blood cells can affect outcome. The neuroprotective effects of EPO should be considered.[12–14]

EPO is known to have anti-inflammatory effects, resulting from the release of nitric oxide that opposes the action of tumor necrosis factor-alpha, and the release of myeloperoxidase and glutathione peroxidase, which counteract the effects of oxygen free radicals.[15,16] Nitric oxide is one of the first immune mediators released after injury, released within hours of insult, and peaking during the first week.[17,18] In the central nervous system, including the optic nerve, EPO inhibits apoptosis and modulates excitability of neurons and their axons, retinal ganglion cells and glial cells via regulation of pro-inflammatory factors including nuclear factor (NF)-κB and the Janus-kinase 2 pathway.[15,16,19]

PION is uncommon and the possibility of other causes of transient vision loss such as migraine or bilateral occipital lobe ischemia should be considered. The pupil findings and the time course for loss (sudden) and recovery of vision (over days) are however much more suggestive of optic nerve ischemia. A diffusion weighted MRI was not obtained but could have helped to further bolster the case for PION.

4. Conclusion

PION an uncommon but potentially devastating post-procedural complication of hypoperfusion related to hemodialysis. Erythropoietin has been proposed as a treatment agent for ischemic optic neuropathy.[5,7,19] We have reported a case of hemodialysis bilateral vision loss related in which prompt treatment with erythropoietin and steroids resulted in a recovery of vision from NLP to baseline. Treatment with erythropoietin within the first 5 days of injury should be considered in similar situations that result in vision loss related to procedural-based hypotension.

Ethical consideration/patient consent

Written consent to publish this case has not been obtained. This report does not contain any personal identifying information.

Funding/grant

No funding or grant support.

Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Declaration of competing interest

All of the authors do not have any conflicts of interest to declare.

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