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Authors

Gui, Wei
Friesen, Erika Tanaka
Bonelli, Laura
[et al.](#)

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Case report

Severe bilateral optic nerve and retinal hypoperfusion in a patient with acute respiratory distress syndrome and septic shock



Wei Gui, MD ^{*}, Erika Tanaka Friesen, MD, Laura Bonelli, MD, Ye Elaine Wang, MD, Anthony C. Arnold, MD

Stein Eye Institute, UCLA Dept. of Ophthalmology, 100 Stein Plaza, Los Angeles, CA 90095, USA

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ABSTRACT

Purpose: To report a case of bilateral central retinal artery occlusion with both anterior and posterior ischemic optic neuropathy.

Observations: A 65-year-old Caucasian woman presented with acute respiratory distress syndrome and septic shock. After treatment with vasopressors and prolonged prone positioning, she was noted to be bilaterally completely blind on hospitalization day 12. Evaluation revealed evidence of bilateral central retinal artery occlusion and bilateral ischemic optic neuropathy. Magnetic resonance imaging of the orbits demonstrated severe restricted diffusion of both optic nerves consistent with ischemia. Both central retinal artery occlusion and ischemic optic neuropathy have been reported in cases of severe hypotension, blood loss, and prone positioning, most often postoperatively after spinal surgery.

Conclusions and importance: To our knowledge, this is the first reported case of bilateral central retinal artery occlusion with both anterior and posterior ischemic optic neuropathy, presumed due to the combination of severe systemic hypotension, hypoxemia due to the respiratory distress syndrome, and prolonged prone positioning.

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1. Introduction

We present the first reported case of bilateral central retinal artery occlusion with both anterior and posterior ischemic optic neuropathy, presumed due to a combination of severe systemic hypotension, hypoxemia, and prolonged prone positioning, in a patient with acute respiratory distress syndrome and septic shock.

2. Case report

A 65-year-old Caucasian female with a history of bipolar disorder, polysubstance abuse, and chronic pain was brought to the emergency room in acute respiratory distress secondary to aspiration and septic shock after being found down. Blood pressure measured 73/43 mm Hg [Normal: 120/80 mm Hg]. Laboratory studies showed hemoglobin of 10.3 g/dL [Normal: 11.6–15.2 g/dL] and platelet count of 439,000/ μ L [Normal: 143,000–398,000/ μ L], with oxygen saturation of 74% [Normal: >95%]. Drug toxicity

screening was positive for barbiturates, benzodiazepines, opiates, and acetaminophen. After initiation of vasopressors and intravenous antibiotics, she was placed in prone positioning to improve respiratory function, 18 h sessions for two consecutive days. Facial edema was noted after prone positioning but no evidence of ocular compression was present. Blood pressure fluctuated markedly during the first two days of admission, ranging from 42/33 to 190/107. On admission day four, the patient was noted to be in atrial fibrillation with a rapid ventricular rate, with recurrences on days six, 11, and 13 before eventual medical control was achieved after two unsuccessful cardioversion attempts. During the course of her hospitalization, she suffered myocardial infarction, acute kidney injury, and shock liver.

On admission day 10, the patient was extubated and, on awakening on day 12, she reported severe bilateral visual loss. Examination at that time revealed no light perception in either eye. The anterior segment examination was unremarkable and intraocular pressures were 6 mm Hg right eye, 7 mm Hg left eye. Pupils measured 5.5 mm right eye, 5 mm left eye, with minimal reaction to light bilaterally and no relative afferent pupillary defect. Ocular versions were intact. On dilated funduscopy, both optic discs demonstrated mild pallid edema. Retinal edema was also noted

^{*} Corresponding author.

E-mail address: Gui@sei.ucla.edu (W. Gui).

posteriorly in each eye, along with diffuse attenuation of retinal vessels and segmented blood flow columns. No retinal emboli were noted (Fig. 1A and B). Fluorescein angiography demonstrated delayed and poor filling of the retinal arteries and veins in each eye, with leakage from both optic discs (Fig. 2A and B). A diagnosis of bilateral ischemic optic neuropathy and bilateral central retinal artery occlusion was made. An extensive evaluation followed and included elevated C-reactive protein of 9.7 mg/dL [Normal: <0.8 mg/dL], negative cytoplasmic- and perinuclear- Anti-Neutrophil Cytoplasmic Antibodies (ANCA), proteinase 3 antibodies, myeloperoxidase antibodies, antinuclear antibody (ANA) and human immunodeficiency virus (HIV) testing. Transthoracic echocardiogram was negative for vegetations. Computed tomography (CT) and magnetic resonance imaging (MRI) of brain including angiography, were negative for stroke or large vessel abnormality. MR imaging of the orbits demonstrated no enhancement on T1 fat suppression (Fig. 3A and B), but severe restricted diffusion of both optic nerves was present (Fig. 4A and B), consistent with ischemia. No evidence of optic nerve inflammation or infiltration was present on imaging.

3. Discussion

Both central retinal artery occlusion and ischemic optic neuropathy separately have been well documented in association with severe hypotension and blood loss, related to gastrointestinal bleeding, cardiac surgery, and most notably after lumbar spine surgery, particularly with prolonged procedures, hypotension, anemia, and prone positioning.^{1–6} The combination of bilateral central retinal artery occlusion and bilateral ischemic optic neuropathy is unique and suggests severe diffuse hypoperfusion in the distribution of the ophthalmic arteries. These may have occurred in

our case because in addition to severe hypotension, dramatic fluctuations in blood pressure, and prolonged prone positioning, there was an additional component of hypoxemia due to respiratory distress. While focal ophthalmic artery occlusion typically is associated with more diffuse ocular ischemia, including choroidal and anterior segment components along with possible extraocular muscle and orbital ischemia,⁷ these were not evident in our case, presumably due to the presence of diffuse hypoperfusion rather than complete focal arterial occlusion.

Prolonged prone positioning in acute respiratory distress syndrome is an established technique which has been shown to reduce morbidity and mortality.^{8,9} The development of ocular complications has not been well documented and, to our knowledge, this is the first reported case of severe ocular ischemia in association with this technique. The parallel syndrome following lumbar spine surgery has been extensively studied, resulting in recommendations to reduce the risk of ocular complications. These include minimizing prolonged intraoperative hypotension, rapid reversal of hypovolemia due to blood loss, minimizing direct pressure on the eye and orbit using a 3-pin head holder, maintaining a neutral forward position with the orbit above the level of the heart, and minimizing time in prone positioning. In particular, authors have suggested that head elevation for 3 min every 30 min may reduce the risk of ischemia in patients with prolonged prone positioning.

The standard T1 and T2 MRI sequences of the orbits in cases of ischemic optic neuropathy typically do not demonstrate enlargement or enhancement of the optic nerves. The anterior form (anterior ischemic optic neuropathy, AION) is limited to the optic disc and is not well visualized by this technique. The rarer posterior form (posterior ischemic optic neuropathy, PION) involves the intraorbital portion of the nerves, but, in contrast to inflammation and infiltration, ischemia is poorly documented on these

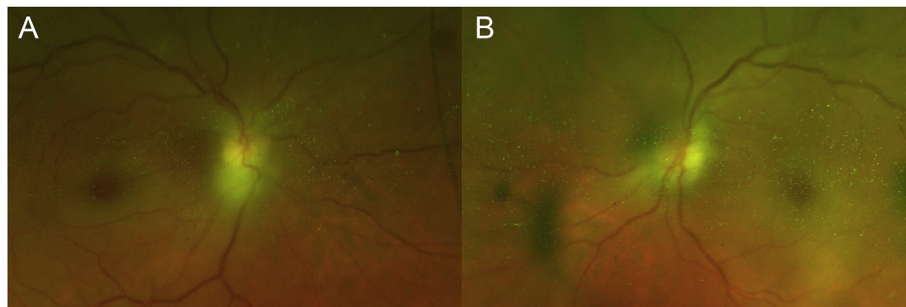


Fig. 1. A. Right eye, Optos wide-field fundus photograph showing optic disc edema, retinal edema with macular cherry red spot, and diffusely attenuated vessels. B. Left eye, Optos wide-field fundus photograph showing optic disc edema, retinal edema with macular cherry red spot, and diffusely attenuated vessels. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

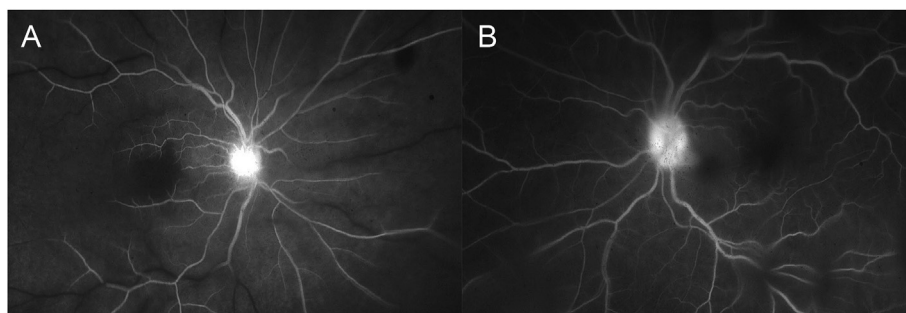


Fig. 2. A. Right eye, Optos wide-field fluorescein angiography image at 1 min, 33 s, showing optic disc leakage, incomplete retinal arteriole filling, and delayed filling of the venous system. B. Left eye, Optos wide-field fluorescein angiography image at 2 min, 26 s, showing optic disc leakage, incomplete retinal arteriole filling, and delayed filling of the venous system.



Fig. 3. T1-weighted orbital magnetic resonance imaging (MRI) with fat-suppression and gadolinium administration. A. Axial view. B. Coronal view. The optic nerves (arrows) show no enhancement.

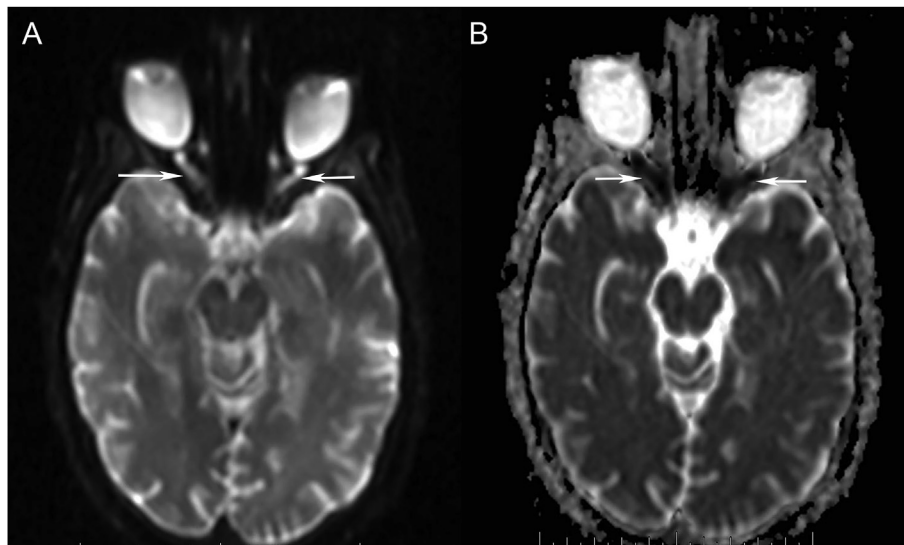


Fig. 4. A. Diffusion-weighted (DWI) orbital magnetic resonance imaging (MRI), axial view, showing bright signal for the entire length of the intraorbital optic nerves (arrows), consistent with restricted diffusion of ischemia. B. Apparent diffusion coefficient (ADC) image, showing dark signal for the entire length of the intraorbital optic nerves (arrows), confirming restricted diffusion of ischemia.

sequences. While diffusion-weighted MR imaging has become the standard for early identification of brain ischemia, it has only rarely been reported to document involvement of the optic nerves. Prior case reports^{10–12} documented optic nerve ischemia but none as severe as our case. Moreover, the presence of both anterior and posterior ischemia in our case reflected the severe ischemic insults to both optic nerves.

4. Conclusion

A 65-year-old Caucasian woman presented with acute respiratory distress syndrome and septic shock, and after treatment with vasopressors and prolonged prone positioning, was found to have suffered severe bilateral ischemic optic neuropathy and central retinal artery occlusion. Extensive optic nerve injury was documented on diffusion-weighted imaging (DWI) of the orbits. The case illustrates the severe hypoperfusion injury of the anterior visual pathway that may result from the combination of factors present.

5. Patient consent

Written patient consent was not obtained. As such, identifying information has not been included.

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Conflicts of interest

None.

Authorship

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

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