Consecutive Shock States Leading to Vision Loss

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Background

Sudden acute vision loss during a hospitalization can be a shocking event for a patient. In a hospitalized inpatient in whom multiple pathophysiological processes may be taking place, it can be challenging to elucidate the cause of vision loss. One common condition that can have devastating ophthalmic consequences is ischemia secondary to a shock state. Ischemia to the optic nerve can have a profound impact on visual acuity. Two ischemia-related ocular conditions are anterior ischemic optic neuropathy (AION) and posterior ischemic optic neuropathy (PION). AION affects the anterior 1 mm of the optic nerve, which includes the optic nerve head. PION affects the part of the optic nerve that is beyond 1 mm posterior to the optic nerve head. An important distinction between the two diseases is involvement of the optic nerve head: on examination of the fundus, the optic nerve head will appear swollen in AION. Once the diagnosis of AION is made, one must distinguish between arteritic and non-arteritic. Arteritic AION is generally due to giant cell arthritis (GCA) and can have devastating, bilateral vision loss if prompt treatment with steroids is not administered. No consensus has been reached regarding the treatment for NAION; however, vision improvement over time is seen in up to 43% of patients.

Clinical Case

A 62-year-old man with no previous medical history presented for fever, nausea, vomiting, diarrhea, and a diffuse maculopapular nonpruritic rash sparing no surfaces of his body, including his hands and feet. The patient was admitted to the MICU for septic shock. The patient’s infectious workup included a negative blood culture, urine culture, HIV, RPR, respiratory panel (Enterovirus, Rhinovirus, Respiratory syncytial virus, Parainfluenza virus, Mycoplasma pneumonia, and Covid) and was empirically treated with aggressive fluid resuscitation and broad-spectrum antibiotics. A TEE was negative for vegetations. The patient’s ICU course was complicated by pulmonary edema, and cardiogenic shock secondary to an NSTEMI. PCI found 80% right coronary artery occlusion and the vessel was subsequently stented. The patient was given a new diagnosis of HFrEF (EF 30%). The day following the stent placement, the patient experienced acute persistent complete loss of vision in his right eye, blurriness of the left eye, and eye pain bilaterally. Labs following the vision loss were notable for Na 131, a positive Coxsackie B antibody titer, Hba1c 6.3, and ESR 26. The patient was evaluated by Ophthalmology who identified bilateral optic disc edema (worse on the right side). The patient was treated with doxycycline and ceftriaxone for empiric treatment of an atypical infection and started on an empiric heparin drip for treatment of Central Retinal Artery Occlusion. Repeat TTE did not show any evidence of new vegetations however notably it did show the EF had recovered to 54%. Carotid artery duplex did not demonstrate narrowing or significant plaques. MRI orbit with and without contrast did not demonstrate any pathology. Further ophthalmologic exams demonstrated evidence most consistent with damage from an ischemic state. The heparin drip, ceftriaxone and doxycycline were discontinued. The patient eventually underwent temporal artery biopsy which did not demonstrate evidence of GCA.

Conclusion

There are a multitude of diagnoses associated with acute vision loss. In this gentleman who was over 50 years of age, and in the post-operative period, the diagnoses of anterior ischemic optic neuropathy (AION), posterior ischemic optic neuropathy (PION), and retinal vascular occlusion should be high on the differential. In this case, the bilateral findings (decreased visual acuity and optic nerve head swelling), severity of the vision loss in the right eye, and history of shock were more suggestive of a hypoperfusion-related ischemia (AION and PION) as opposed to an occlusion-related ischemia (Retinal Vascular Occlusion). Notably the patient experienced two consecutive ischemic states given that he initially was in septic shock followed by cardiogenic shock. In this patient, whose fundoscopic examination showed optic nerve head swelling, AION was the most likely diagnosis. Non-arteritic AION has a wide variety of causes; one of these causes is systemic hypotension (as seen in this patient). Given the history of multiple episodes of shock, the absence of GCA findings (polymyalgia rheumatica, jaw claudication, and facial pain), the absence of premonitory visual disturbances, and the negative temporal artery biopsy, the diagnosis given to this patient was shock-induced anterior ischemic optic neuropathy (SIAION). This is one of the first documented cases of AION secondary to a cardiogenic shock due to acute coronary syndrome, and also one of the first documented cases of AION following two consecutive shock states.

References