CASE REPORT

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Hyperbaric therapy for bilateral visual loss during hemodialysis

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Abstract
Bilateral vision loss during hemodialysis is a rare but devastating entity, with grim prognosis for sight. The etiologies are diverse but share ischemia as a common mechanism. This is a report of a patient with bilateral sight loss during hemodialysis, with early hyperbaric treatment and return of visual acuity to baseline. Hyperbaric treatment should be considered, where early administration is possible, for bilateral blindness during hemodialysis.

Key words Hemodialysis · Hyperbaric oxygen · Sudden blindness

Introduction

Sudden bilateral blindness during hemodialysis has previously been described in a number of case reports and small case series. Although it is a rare occurrence during hemodialysis, the consequences of bilateral blindness are devastating. Several syndromes culminating in loss of sight have been described, including anterior ischemic optic neuropathy, bilateral occipital infarcts, Ponscher’s-like retinopathy, and uremic amaurosis.

We present a case of bilateral blindness during hemodialysis treated by hyperbaric oxygen, with the return of the patient’s visual acuity to its predialysis level.

Case report

The patient was a 69-year-old man with diabetes mellitus, hypertension, and end-organ damage including background diabetic retinopathy, ischemic heart disease, and a history of cerebrovascular accident 7 years before his present illness. He was suffering from endstage renal disease and had been treated with hemodialysis for several years; he was not taking sildenafil.

During a hemodialysis session, bilateral loss of sight occurred. There were no documented hypotensive episodes during treatment. Visual acuity was limited to finger counting from a distance of 0.5m bilaterally, with no improvement after 4.5h. There was no relative afferent pupillary defect, and fundus examination revealed arterial narrowing, (A-V) crossing, silver wiring, and hard exudates, but no edema or cherry red spot. The suspected diagnosis was retinal artery occlusion, considered an indication for hyperbaric therapy; the patient was referred to the hyperbaric unit. Computerised tomography (CT) was performed and revealed an old occipital infarction.

The patient was treated with hyperbaric oxygen (HBO) at 2.8 atmospheres absolute (ATA) of oxygen according to United States Navy Table 6 (which entails cycles of oxygen breathing followed by intervals of air breathing to avoid oxygen toxicity), commenced within 4h of presentation, and a repeat HBO treatment at 2.5 ATA on the following day. Seven minutes after the initiation of therapy, the patient’s sight began to improve, continuing to do so during the subsequent HBO session. Vision remained stable, close to baseline function of 6/15, recorded before the hemodialysis session, and HBO was not continued. The patient declined to undergo further evaluation, including contrast echocardiography and follow-up CT. On follow-up 3 months after treatment his sight remained at baseline level and no further episodes occurred.

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Discussion

Sudden blindness during hemodialysis is a rare event with a limited differential diagnosis, including retinal artery occlusion, uremic amaurosis, Purtcher's retinopathy, anterior ischemic optic neuropathy, and occipital cortical ischemia. Cardiovascular pathologies, often present in the diabetic and hypertensive patient also suffering from endstage kidney disease, carry a high risk for retinal artery occlusion. Retinal artery occlusion occurs unilaterally. Most cases are of embolic etiology, originating in cardiac pathologies or atherosclerosis of the large vessels. The presentation is of sudden blindness, almost invariably accompanied by a relative afferent pupillary defect and fundoscopic findings, which include retinal ischemic edema, usually evident within 5-10 min. The arteries appear thin, veins appear darker than normal, and the retina reaches maximal whiteness 70 min after the occlusion. The fovea stands out as a red-brown spot due to preserved nutrition from the choroid, leading to the appearance of a "cherry red spot." With conventional treatment modalities, this entity carries a grim prognosis for vision. The bilateral presentation and the absence of the above signs in our patient made the diagnosis of retinal artery occlusion unlikely.

The syndrome of uremic amaurosis has been related to edema of the occipital cortex, and the visual loss in this syndrome resolves with prompt dialysis. The appearance of blindness during hemodialysis, as observed in our patient, is not compatible with this diagnosis.

Purtcher's retinopathy was initially described in trauma patients, but the list of etiologies has since been expanded to include acute pancreatitis and autoimmune diseases, among others. Purtcher's-like retinopathy has also been described in chronic renal failure. It has been suggested that complement-induced granulocyte aggregation and leukoembolisation underlie this syndrome. However, in the absence of the typical retinal appearance, with edema and hemorrhages in the posterior section of the retina, this diagnosis can be excluded.

Anterior ischemic optic neuropathy is caused by ischemia and infarction of the optic nerves, presenting as sudden, painless loss of vision that carries a poor prognosis for sight. Hypotension is a known major cause of ischemic optic neuropathy. Twenty-five percent of hemodialysis sessions are complicated by hypotension, which is considered to be the most common systemic complication of this procedure. The disease usually affects one eye; nevertheless, sequential bilateral involvement has been reported. Although anterior ischemic optic neuropathy cannot be ruled out in the presented patient, the lack of hypotensive events during hemodialysis and the simultaneous bilateral presentation of blindness decrease the probability of this diagnosis.

Ischemia of the occipital cortex can lead to bilateral visual loss, albeit with preservation of pupillary reactions and normal fundus examination. Our patient was diabetic, with known ischemic heart disease, a history of cerebrovascular accident, having CT evidence of an old occipital infarct. Fundus examination revealed combined diabetic and hypertensive retinopathy, with no further findings supporting an alternative diagnosis. The bilateral blindness favors the above diagnosis of occipital cortical ischemia, which might have been the result of preexisting cerebrovascular disease combined with transient hypoperfusion brought about by hemodialysis.

An alternative explanation for sudden occipital ischemia might be arterial gas embolism. The presence of air bubbles in cerebral arteries causes an abrupt decrease in blood flow in areas supplied by the occluded vessels. Air bubbles may be introduced into the arterial side as a result of barotrauma related to diving, as well as a number of iatrogenic causes, including cardiac surgery and catheterisation, head and neck surgery, lung biopsy, and open central lines.

Hemodialysis may be complicated by gas embolism. The gas bubbles introduced into the venous circulation are usually removed by the pulmonary filter, but large amounts might overwhelm the pulmonary filter and cause leakage of some gas through intrapulmonary shunts. Alternatively, the rise in pulmonary artery pressure may cause elevation of pressure in the right heart, forcing bubbles through a probe-patent septal defect, a condition present in 30% of the adult population. HBO is a specific and well-established treatment for gas embolism, and should be administered as soon as possible. The role of HBO in vascular stroke is still under consideration.

The rapid improvement of visual acuity during treatment in our patient supports an ischemic etiology, by one of the mechanisms described above. Potential benefits of HBO for this purpose include the following:

1. HBO increases the amount of dissolved oxygen in the plasma, supporting the aerobic processes of threatened cells and salvaging non-functioning tissue which may still be viable.
2. HBO causes vasoconstriction of normal cerebral vessels, augmenting the blood flow to ischemic regions.
3. The enhanced tissue survival reduces edema formation.
4. In the case of arterial gas embolism, HBO results in immediate reduction of bubble size with relief of mechanical obstruction.

The early commencement of hyperbaric treatment in our patient, and its proximity to an event that occurred in hospital, provided a unique opportunity to salvage the marginally perfused cells in the ischemic penumbra and to restore vision to near the pretreatment level.

Hyperbaric therapy is not free of side effects, which include barotrauma, oxygen toxicity, and claustrophobia. However, we believe that HBO should be considered an important adjunct in the treatment of sudden blindness during hemodialysis, given the dire consequences of this rare complication.

References