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CASE REPORT Open Access

Posterior ischemic optic neuropathy following continuous renal replacement therapy: a case report

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Abstract

Background Posterior ischemic optic neuropathy (PION) is a rare cause of acute vision loss in intensive care unit patients. PION following continuous renal replacement therapy (CRRT) hemodialysis has not ever been reported. Here, we report a case of bilateral nonarteritic PION following the initiation of CRRT.

Case presentation A 52-year-old man with hypertension and stage 4 chronic kidney disease was admitted due to metabolic acidosis, hyperkalemia, and acute exacerbation of CKD. CRRT caused transient hypotension upon initiation but corrected the metabolic acidosis and hyperkalemia six hours after initiation. Therefore, CRRT was stopped. However, several hours after the cessation of CRRT, the patient experienced sudden, painless vision loss in both eyes. Assessment of his visual acuity revealed the inability to perceive light in both eyes. There were no symptoms or signs of giant cell arteritis. An ophthalmological examination revealed no abnormalities. Magnetic resonance imaging of the brain revealed no compressive lesions or acute stroke, but magnetic resonance angiography revealed stenosis of both the bilateral carotid artery and the right middle cerebral artery. Administration of a high dose of corticosteroids did not reverse his vision loss. Thus, nonarteritic PION following CRRT was diagnosed.

Conclusions PION should be considered if a patient with multiple vascular risk factors complains of sudden painless vision loss without signs of optic disk edema after the initiation of CRRT. Preventing blood pressure drops during the initiation of CRRT in patients with multiple vascular risk factors may prevent PION.

Keywords Continuous renal replacement therapy, Posterior ischemic optic neuropathy, Visual loss

Introduction

Posterior ischemic optic neuropathy (PION) is caused by vascular compromise affecting the circulation of the posterior (retrolaminar) portion of the optic nerve [1–3]. PION is a rare cause of visual impairment, and its accurate incidence remains unclear [4]. PION is often classified into three subtypes on the basis of the clinical scenario and associated conditions: perioperative, arteritic, and nonarteritic [5, 6]. Perioperative PION typically occurs in patients with hypotension and severe anemia during the perioperative period. Arteritic PION is caused by systemic vasculitis, most commonly giant cell arteritis (GCA). Nonarteritic PION occurs in patients at risk for arteriosclerosis, often in association with hemodialysis, hypotension, or anemia [7]. However, PION following continuous renal replacement therapy (CRRT) has not been reported. Here, we report a case of bilateral

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nonarteritic PION following the initiation of CRRT in a patient with a high risk of arteriosclerosis.

Case presentation

A 52-year-old man with a history of uncontrolled hypertension and stage 4 chronic kidney disease (CKD) due to renal sclerosis presented to our emergency department because of a one-week history of appetite loss and weakness. He reported no headache, transient vision loss, jaw claudication, myalgia, constitutional symptoms, motor symptoms or sensory symptoms. He had a history of dyslipidemia and renal anemia, but no history of prior trauma or ophthalmologic diseases. His medications included antihypertensive drugs and loop diuretics. He did not take any drugs or substances that could cause ocular toxicity. Upon presentation, he was alert and oriented. His temperature was 36.0 °C, his heart rate was 103 beats per minute, his blood pressure was 140/60 mmHg, his respiratory rate was 36 breaths per minute, and his oxygen saturation was 99% while breathing ambient air. On physical examination, his lungs were clear on auscultation, and there was no abdominal tenderness on palpation. There was also no conjunctival congestion, eyelid swelling, proptosis, tenderness over the bilateral temporal region, or carotid bruit on either side. On neurological examination, there were no significant findings. His laboratory tests revealed anemia, renal failure, hyperkalemia, and high anion gap metabolic acidosis (Table 1). Chest X-ray revealed no abnormalities. Electrocardiography revealed sinus tachycardia and tented T waves, but no other abnormalities. Cardiac ultrasound revealed no cardiac structural or functional abnormalities. Metabolic acidosis and hyperkalemia due to acute exacerbation of CKD triggered by insufficient intake were diagnosed, and the patient was admitted to the intensive care unit. A vascular catheter was placed, and CRRT with a blood flow rate of 200 mL/min was initiated. During CRRT, the highest recorded arterial blood pressure was 160/60 mmHg, and the lowest was 90/40 mmHg. Six hours after the initiation of CRRT, metabolic acidosis and hyperkalemia improved, thus CRRT was discontinued. After discontinuing CRRT, no hypotension was observed. At this time, the patient did not report any visual symptoms. However, several hours later, his vision became blurred. The patient's blood pressure was maintained at 119/56 mmHg. Ten hours later, the patient reported painless bilateral vision loss without any other symptoms, such as headache. He exhibited hand-motion visual acuity in both eyes with fully dilated pupils. There was no relative afferent pupillary defect. Computed tomography (CT) and magnetic resonance imaging (MRI) of the head revealed no acute ischemic lesions or compressive lesions. Magnetic resonance angiography (MRA) revealed stenosis of both the bilateral internal carotid artery and the right middle cerebral artery, but the ophthalmic artery remained patent (Fig. 1). An ophthalmologic evaluation revealed that the anterior segments of his eyes, ocular motility, and intraocular pressures were normal. The optic nerve heads in the fundus of both eyes were normal, with no edema or pallor (Fig. 2). The electroretinogram results were normal, and the visual evoked response was unrecordable. The laboratory tests revealed that the erythrocyte sedimentation rate was 58 mm/hour (reference range: 0 to 13 mm/hr). The tests also yielded negative results for antinuclear antibodies, anti-neutrophil cytoplasmic antibodies, and anti-aquaporin-4 antibodies. Cerebrospinal fluid analysis showed no elevation in cell count or protein levels. The ankle-brachial index was 0.47 on the right and 0.52 on the left. Carotid ultrasonography revealed no stenosis of extracranial portion of internal carotid artery on either side. High doses of corticosteroids were intravenously administered for three days. Nonetheless, his vision did not improve despite this therapy. Thus, bilateral nonarteritic PION was diagnosed. After correction of the metabolic acidosis and hyperkalemia and antibiotic treatment for a urinary tract infection, his appetite improved. Nonetheless, his renal function did not improve, so he started regular hemodialysis after an arteriovenous shunt was created. During the 10-week

Table 1 Comparison of clinical features according to the subtypes of PION

	Perioperative[a]	Arteritic	Nonarteritic
Age	20 to 50 years	> 70 years	> 50 years
Onset	Sudden	Sudden	Sudden
Eye pain	Uncommon	Present	Uncommon
Accompanying symptoms or signs	Absent	Headache, jaw claudication, myalgia, and arthralgia	Absent
Bilateral or unilateral involvement	Often bilateral	Often bilateral, but not simultaneous onset	Typically unilateral
Cardiovascular risk factors	Present	Common	Present
Treatment	Not established	Corticosteroids	Not established
Prognosis[b]	Often stable	Often stable	Worse

[a]The most common surgical procedures are cardiac surgery and spine surgeries

[b]Visual course after initial vision

PION: posterior ischemic optic neuropathy

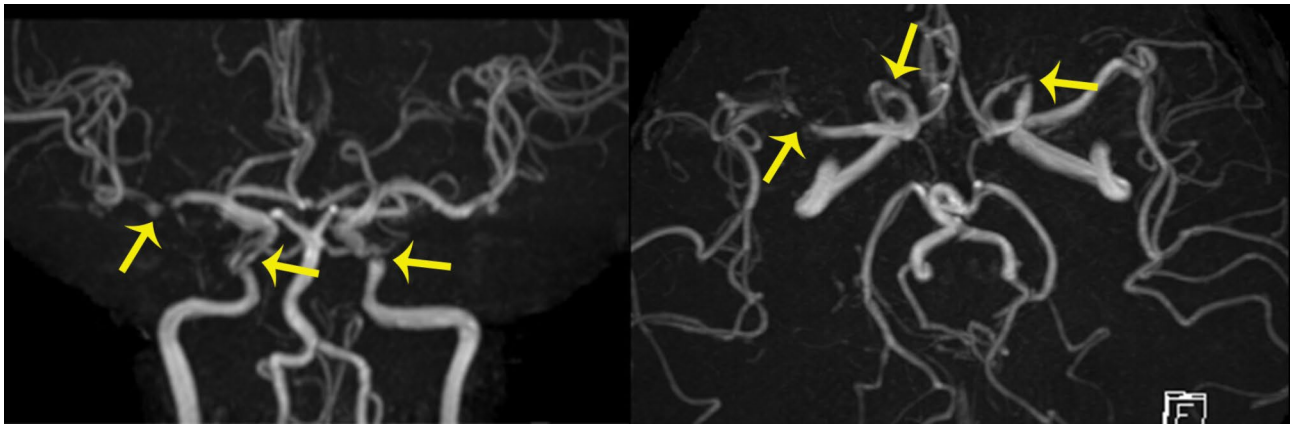

Fig. 1 Magnetic resonance angiography of the brain revealed stenosis of the bilateral internal carotid artery and right middle cerebral artery (yellow arrows)

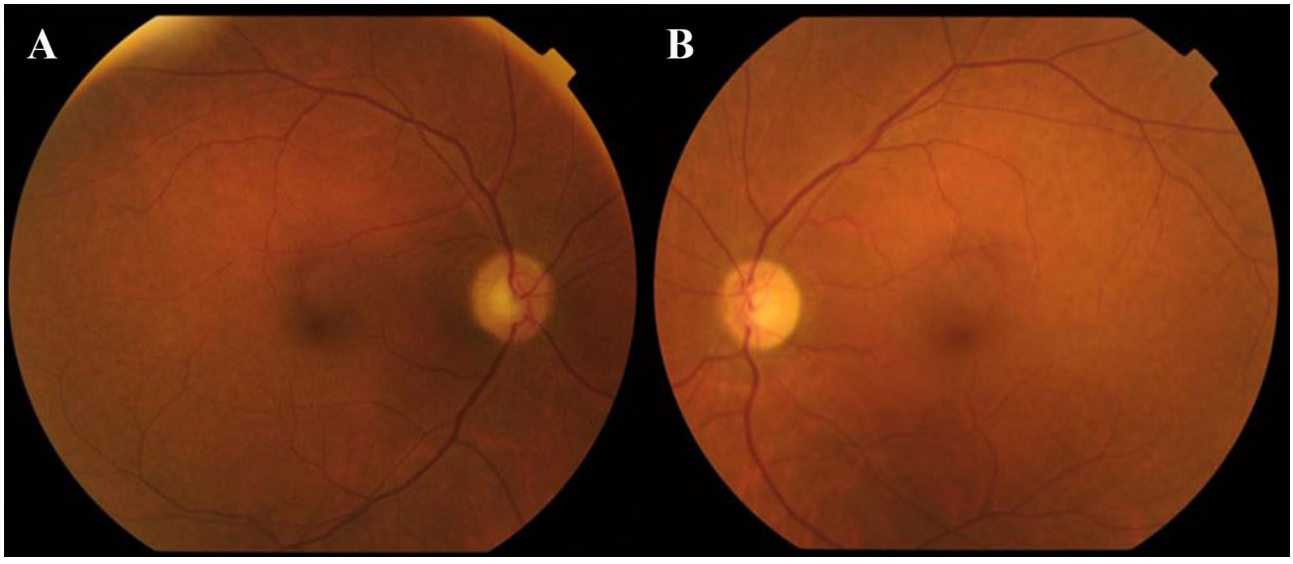

Fig. 2 Funduscopic examination of both the right (**A**) and left (**B**) sides revealed no optic disc edema or pallor

hospital stay, there was no improvement in his visual acuity. He was transferred to the rehabilitation facility.

Discussion

To our knowledge, this is the first case of PION following CRRT. We concluded that PION should be considered a differential diagnosis for patients with vision loss following CRRT. Moreover, it is important to avoid hypotension during CRRT in patients with multiple vascular risk factors to prevent PION.

Vision loss is a less common ophthalmologic complication in ICU patients [8]. The causes of vision loss in ICU patients include endophthalmitis, acute glaucoma, anterior ischemic optic neuropathy (AION), PION, retinal vascular occlusions, and retrobulbar optic neuritis [8]. To determine the cause of vision loss, ocular examinations, including funduscopy by ophthalmologists, should be routinely performed [8]. Although PION is a rare cause of vision loss in ICU patients, it should be considered as differential diagnosis if there are no abnormal findings on ocular funduscopy. Although differentiation between PION and retrobulbar optic neuritis is challenging, they can be differentiated based mainly on clinical features. PION is characterized by sudden-onset vision loss without pain, whereas retrobulbar optic neuritis develops over several hours to days and is accompanied by eye movement pain in most cases [6, 9]. Our patient presented with the clinical features of PION. Moreover, brain imaging should be conducted to rule out compressive lesions, inflammatory changes, and ischemic stroke because the exclusion of other disease associated with vision loss is necessary for the diagnosis of PION [7]. The proposed diagnostic criteria are as follows: an acute deficit in visual acuity, visual field, or both; an ipsilateral relative afferent pupillary defect, unless there is bilateral symmetrical involvement or preexisting contralateral

Table 2 Laboratory data at admission

Variables	At admission	Reference range
Hemoglobin (g/dl)	8.8	12.0–16.0
Hematocrit (%)	26.7	37.0–47.0
White-cell count (per μl)	23,900	4800–10,800
Platelet count (per μL)	280,000	150,000–450,000
Alanine aminotransferase (U/liter)	17	10–40
Aspartate aminotransferase (U/liter)	6	10–55
Alkaline phosphatase (U/liter)	54	129–417
Total bilirubin (mg/dL)	0.1	0.0–1.0
Lactate dehydrogenase (U/liter)	207	313–618
Lactate (mmol/liter)	2.2	0.7–2.1
Total protein (g/dl)	7.3	6.0–8.3
Albumin (g/dl)	2.4	3.3–5.0
Urea nitrogen (mg/dl)	197	8–25
Creatinine (mg/dl)	20.5	0.6–1.5
Estimated glomerular filtration rate (ml/min/1.73m^2)	2.2	> 59
Sodium (mmol/liter)	133	135–145
Potassium (mmol/liter)	7.1	3.4–5.0
Chloride (mmol/liter)	89	98–108
Calcium (mg/dl)	6.7	8.5–10.5
Glucose (g/dl)	136	70–110
Hemoglobin A1c (%)	5.9	4.6–6.2
C-reactive protein (mg/liter)	12.1	< 3.0
High density lipoprotein cholesterol (mg/dl)	45	40–86
Low density lipoprotein cholesterol (mg/dl)	62	70–139
Venous blood gas analysis		
pH	6.92	7.31–7.41
Partial pressure of carbon dioxide (mmHg)	17.1	41–51
Bicarbonate (mmol/liter)	3.4	23.0–32.0
Anion gap (mmol/liter)	43.1	8.0–16.0

optic neuropathy and pupils that are sluggish or nonreactive to light; documentation of a normal optic disc at the onset of the visual deficit; exclusion of other identifiable causes of visual deficit, such as retinal vascular occlusion; exclusion of other causes of optic neuropathy, such as compression, demyelination, or inflammation with neuroimaging; abnormal visual evoked response; normal electroretinogram; and development of optic nerve pallor within 4 to 8 weeks of the onset of vision loss [7]. Although the development of optic nerve pallor was not confirmed, our patient met al.l the other diagnostic criteria.

If PION is diagnosed, it is important to classify three subtypes of PION (Table 2). In particular, differentiation of arteritic PION from the other subtypes of PION is crucial because arteritic PION can be treated with glucocorticoids. Given that arteritic PION is caused by most commonly GCA, screening at-risk patients for signs and symptoms of GCA is important. In our case, there were no signs and symptoms associated with GCA. Moreover, there was no response to treatment with high-dose corticosteroids.

Most nonarteritic PION patients have one or more vascular risk factors, including hypertension, diabetes mellitus, hypercholesterolemia, cardiac disease, and cerebrovascular disease [5]. Hypotension, hypovolemia, and anemia can cause PION in patients with these risk factors [4, 5, 10, 11]. In the present case, the patient had a history of hypertension, dyslipidemia, CKD, and chronic anemia. Moreover, intracranial arteriosclerosis was newly identified after the index admission. Therefore, it is possible that CRRT-associated hypotension might result in PION in patients with these risk factors. Avoiding hypotension and correcting anemia and hypovolemia in patients with these risk factors are crucial to prevent PION.

To our knowledge, there have been no reports of nonarteritic PION following CRRT. However, Laurence LM et al. reported nonarteritic PION following the first session of hemodialysis in a stable patient with chronic renal failure [7]. This is the first reported case of PION following hemodialysis [7]. They speculated that the autoregulation of blood flow at the posterior optic nerve could not compensate for the small drop in blood pressure due to initiation of hemodialysis and might result in PION [7]. Therefore, hypotension during CRRT may have resulted

in PION in our patient. Given that CRRT-related hypotension or instability are common at the time of initiation [12], avoiding hypotension at the time of initiation of CRRT by increasing the blood flow rate gradually, particularly for patients with multiple vascular risk factors, may be crucial for preventing PION.

Conclusions

PION should be considered if a patient with a risk of arteriosclerosis complains of sudden painless visual impairment without signs of optic disk edema after the initiation of CRRT. Preventing blood pressure drops during the initiation of CRRT in patients with multiple vascular risk factors might prevent PION.

Abbreviations

CKD	Chronic kidney disease
CRRT	Continuous renal replacement therapy
CT	Computed tomography
GCA	Giant cell arteritis
MRA	Magnetic resonance angiography
MRI	Magnetic resonance imaging
ICU	Intensive care unit
PION	Posterior ischemic optic neuropathy

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Author contributions

HT wrote a draft of the manuscript. JK provided to the critical revision of the manuscript. All authors contributed to the revision of the manuscript and have read and approved the final manuscript.

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Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

We obtained informed consent from the patient.

Competing interests

The authors declare no competing interests.

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