Bilateral ischemic optic neuropathy due to hypotension during kidney dialysis

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Abstract

Nonarteritic anterior ischemic optic neuropathy (NAION) is a potentially sight-threatening condition. Simultaneous bilateral NAION, such as this case, are extremely rare, but have been shown to be associated with hypotension induced by kidney dialysis.
Patient Demographics

A 43 year old African American female is referred for central scotoma of the left eye and bilateral optic nerve head edema. The patient states she awoke 5 days ago with “scrambled vision.” She went to her kidney dialysis session and reports that her vision improved momentarily, but worsened again 2 days ago. She complains that the inferior two-thirds of her vision is gone in her left eye. She reports that her right eye seems fine.

The patient’s ocular history is unremarkable with no spectacle or contact lens prescription. She is currently on dialysis three times a week due to kidney failure secondary to preeclampsia during pregnancy in 1991. She has been on dialysis since 1993 and has had several kidney surgeries. Current medications include Renagel, Coumadin, Ranitidine, and Oxycodone.

Pertinent Findings

Uncorrected visual acuities were 20/30 in the right eye and hand motion in the left eye, with no improvement upon pinhole. Confrontation visual fields revealed a superior defect in the right eye and inferior defect in the left eye. Extra-ocular motility was normal. Pupils were recorded as nonreactive. Intraocular pressures were normal at 10mmHg in both eyes at 1:18pm. A blood pressure reading in-office measured 89/61. Anterior slit lamp examination was normal. Optic nerve evaluation revealed a cup-to-disc ratio of 0.0 in each eye, with inferior segmental edema of the optic nerve head of the right eye and superior edema/pallor of the left eye. Macula and retinal periphery was normal. (Fundus photos available for poster / presentation)

Differential Diagnosis

Optic nerve head edema occurs when there is a blockage of axoplasmic transport at the lamina cribrosa. This can result due to compression, ischemia, inflammation, metabolic dysfunction, or toxic damage. Clinical signs of optic nerve head edema may include blurred disc margins, loss of spontaneous venous pulsation, and hemorrhages or infarcts within the nerve fiber layer. The optic nerve usually becomes a pale color, a sign of dead axons, after the swelling has resolved.

True optic disc swelling can be cause by several different conditions and etiologies. Papilledema is defined as disc swelling in patients with increased intracranial pressure. Patients with papilledema typically complain of headaches and/or nausea, pulsatile tinnitus, and transient vision obstructions. The visual field defect in these patients can vary, but usually shows an enlarged blind spot.

Optic nerve swelling can be due to inflammation, called optic neuritis or papillitis. This is characterized by sudden vision loss and pain upon eye movement. Optic neuritis is typically associated with demyelinating conditions, such as multiple sclerosis, or inflammatory reactions due to syphilis, Lyme
disease, sarcoidosis, or cat-scratch disease. In most cases, the vision will remain diminished for several days and then gradually improve over several weeks.

Vascular dysfunction can cause ischemia to the optic nerve to produce optic disc swelling. This condition is called anterior ischemic optic neuropathy (AION). The patient typically complains of sudden, painless loss of vision. The condition is also characterized by the presence of a relative afferent pupillary defect and altitudinal visual field defect. The loss of vision develops over several hours and, unfortunately, usually remains stable. This condition is most common in individuals over the age of 50, and is usually associated with hypertension, diabetes, or giant cell arteritis.

Compression of the optic nerve caused by a tumor, or infiltration of the nerve due to leukemia or lymphoma may also produce optic nerve swelling. The signs and symptoms of these conditions may vary depending on the severity and extent of the disease.

Toxic and metabolic disorders may cause a mild, bilateral optic nerve swelling. Vision is usually diminished with abnormal color vision. Visual field defects tend to be central or cecocentral. Visual acuity can typically be recovered by eliminating the toxin or correcting the metabolic abnormality.

Optic disc swelling can also be caused by hypotony. This can be caused by blunt trauma, penetrating injury, or intraocular operation with an insecure wound. The vision usually improves as the hypotony resolves.

Some conditions, such as optic disc drusen and crowded discs, may mimic optic nerve swelling. However, these patients typically remain asymptomatic with normal visual acuity. (1)

**Diagnosis and Discussion**

Given the patient’s history of renal failure and subsequent dialysis, and presentation of sudden, painless vision loss consistent with an altitudinal field defect, a diagnosis of bilateral non-arteritic ischemic optic neuropathy was made. Below is a review of the classification, pathogenesis, clinical presentation, and management of anterior ischemic optic neuropathy. Also presented is a review of the current documentation on the association between NAION and hypotension during dialysis.

Ischemic optic neuropathy (ION) is a group of disorders in which blood supply to the optic nerve has been compromised. ION is the most prevalent non-glaucomatous optic neuropathy in patients over 50 years of age. The annual incidence of NAION is 2.3-10.2 per 100,000 individuals in this age group, 95% of these being Caucasian. (1) ION can be classified as anterior (AION), which is characterized by a relative pupillary defect, visual field defect, and optic disc edema – or posterior (PION) which does not present with disc edema. AION can also be termed arteritic (AAION), marked with severe optic disc edema and associated with temporal or giant cell arteritis – or nonarteritic (NAION), in which optic disc edema tends to be sectoral. (2) (3)
The pathogenesis of NAION involves the interference of blood supply to the optic nerve. This may be caused by systemic conditions such as hypertension, atherosclerosis, or anemia. Other predisposing factors may be local, such as crowded optic disc or presence of optic nerve head drusen. Unfortunately, NAION is a multifactorial condition, with no single factor explaining the incident. (2) (3) A precipitating factor may also be present; one that is not the underlying cause but contributes to the occurrence of optic nerve ischemia, such as nocturnal hypotension. (2)

Patients typically present with sudden, painless loss of vision. The complaint may be blurred central vision, loss of part of their visual field, or both. Visual acuity can vary from 20/20 to no light perception. Color vision is usually diminished on the affected side. Altitudinal visual field defects are the most common pattern, usually located inferiorly. An afferent papillary defect is usually present. Optic nerve evaluation will show a swollen optic disc, either diffuse or segmental corresponding to the altitudinal field defect. Flame-shaped hemorrhages may be seen near the disc margin. As the edema resolves, the affected portion of the disc will become pale. The visual outcome is highly variable. (1) (2)

Blood tests can be done to identify possible risk factors and/or etiology. Elevated erythrocyte sedimentation rate and C reactive protein are helpful in diagnosing AAION, with a definitive diagnosis made with temporal biopsy. Magnetic resonance imaging (MRI) can be used to rule out demyelinating diseases or tumors. (2)

The treatment and management of NAION has been of recent controversy. Optic nerve sheath decompression has been evaluated; however, a multicenter clinical trial has shown that this procedure is not effective and may even be harmful. Aspirin therapy was thought to prevent the development of ION in the fellow eye. This has also been disproven, as NAION is not a thromboembolic disorder, but a hypotensive issue. Recently, there have been studies to prove that systemic corticosteroid therapy is helpful in patients with NAION. They have shown that treatment may improve visual outcome and visual field defects, and shows that optic disc edema resolves significantly faster. However, the study's reliability is under scrutiny. Further studies are being done to examine the place for intravitreal injections, such as triamcinolone and bevacizumab, in NAION therapy. Regardless of therapy disputes, because NAION is a multifactorial condition, the best strategy is to evaluate and reduce as many predisposing risk factors as possible. (4)

Although fluid removal is the major goal of dialysis, the amount/rate of fluid removed often leaves the patient with hypotension and muscle cramps. (5) A decrease in blood pressure is one of the most frequent complications during and after dialysis. This is due to the excessive ultrafiltration and inadequate vascular filling. (6)

A significant decrease in blood pressure, during kidney dialysis for example, causes a decline in optic nerve head perfusion, leading to optic nerve ischemia. Several cases of NAION due to hypotension during renal dialysis have been reported. Extremely rare, however, are the simultaneous, bilateral events. These cases appear to be nearly exclusive to chronic dialysis patients, as they suffer from severe hypotension. (7) (8) In the few documented cases of NAION with dialysis patients, most cases share a
common feature of hypotension. (9) Nocturnal hypotension can be a significant factor in patients already susceptible to optic nerve damage. The decrease in blood pressure at night can result in NAION, explaining the fact that most patients discover vision loss upon wakening. (10) The overnight drop in blood pressure may not be appreciated with routine blood pressure measurements in the office. Therefore, 24-hour ambulatory blood pressure monitoring may be of significant benefit in these patients. (11)

**Treatment and Management**

The impression is: 1) bilateral non-arteritic ischemic optic neuropathy, possibly due to hypotension during dialysis; 2) visual field defect, left eye greater than right eye. The patient was asked to return in one week for a repeat dilated fundus exam and Humphrey visual field, with a note to carefully examine pupils prior to dilation. A phone call was made to the patient’s nephrologist regarding the concern of severe hypotension during and after dialysis. The doctor stated that measures can be taken to manage the patient’s hypotension during dialysis in the future.

The patient returns in one week, stating her vision has not changed since last visit. Visual acuities remained fairly stable at 20/30 in the right eye and count fingers in the left eye. Pupil examination showed a sluggish reaction in both eyes, with no relative afferent pupillary defect. Blood pressure was measured to be 113/75. A Humphrey visual field, 30-2 threshold, was performed on each eye. The right eye field shows a dense superior altitudinal defect, and the left shows a more diffuse inferior > superior altitudinal defect. There was great reliability on the right eye, but excessive false negative errors for the left eye, with a note that she could not fixate on the central target. These visual field patterns are consistent with the patient’s optic nerve evaluation, showing again inferior edema of the right disc and superior edema of the left disc. The impression remains to be ischemic optic neuropathy and visual field defect of both eyes. She was asked to return in 2-3 months for a progress check and repeat visual field. (Visual fields available for poster / presentation)

The patient returned for her overdue follow-up 4 months later. She again reports no changes. Visual acuities are 20/25 in the right eye and count fingers in the left eye. Visual field results are stable with a superior defect in the right eye and inferior defect in the left eye. The defect in the right eye shows a small central island of vision, through which the patient is able to see 20/25. The optic disc edema is resolved in both eyes, resulting in pallor of the affected areas. At this point, we do not expect the patient to have any significant improvements in either eye, and asked her to return in 4-6 months for a progress check.

**Conclusion**

The risk of developing NAION due to systemic conditions is significant, but coupled with the severe hypotension that patients experience on dialysis, the results can be devastating. Ischemic optic
neuropathy should be a primary consideration in any dialysis patient that presents with acute vision loss. With the increasing number of patients in need of renal replacement therapy, awareness is becoming more important. Effective treatment of NAION is not yet established; therefore prevention is critical. Blood pressure should be monitored closely, with the implementation of 24-hour ambulatory monitoring if necessary. Encouraging results have been found with systemic corticosteroid therapy. Thus, it seems reasonable to immediately correct hypotension and begin steroid treatments in a dialysis patient with NAION.
Works Cited


