

CASE REPORT

Transient Visual Loss May Anticipate Occipital Infarction From Hemodialysis

Michael Wells and Rod Foroozan, MD

• **Background:** The aim of this study was to report on a patient who had occipital infarction from intradialytic hypotension and review the literature on hemodialysis and visual loss. **Methods:** Neuro-ophthalmologic examination and magnetic resonance imaging (MRI) of the brain were conducted. **Results:** A 61-year-old woman had acute onset of a right homonymous superior quadrantic visual field defect during dialysis after several episodes of transient visual loss associated with symptomatic intradialytic hypotension. MRI showed acute infarction in the left occipital lobe. **Conclusion:** Visual loss is an uncommon complication of hemodialysis. The mechanism usually is related to hypotension and is usually from anterior ischemic optic neuropathy. Although cortical blindness secondary to stroke has been reported as a complication after hemodialysis, we were unable to find another report of visual loss secondary to stroke attributed to intradialytic hypotension with preceding episodes of transient visual loss. Transient visual loss may be a risk factor for stroke during hemodialysis. *Am J Kidney Dis* 43:E23.

© 2004 by the National Kidney Foundation, Inc.

INDEX WORDS: Hemodialysis (HD); visual loss; stroke; intradialytic hypotension.

VISUAL LOSS is an uncommon complication of hemodialysis. The mechanism is frequently related to hypotension and is usually from anterior ischemic optic neuropathy. We report the case of a 61-year-old woman who, after several episodes of transient visual loss associated with symptomatic intradialytic hypotension, had acute onset of a right homonymous superior quadrantic visual field defect during dialysis. Symptomatic intradialytic hypotension is a common complication of hemodialysis, and associated transient visual loss may be a risk factor for stroke during hemodialysis.

CASE REPORT

A 61-year-old woman with a history of renal cell carcinoma, status post-right nephrectomy in 1998, and end-stage renal disease secondary to diabetes mellitus, began hemodialysis 2½ years before presentation. She had a prior history of myocardial infarction, stroke, and hypertension. The patient reported that during dialysis in the weeks before presentation, she had repeated episodes of light headedness with visual disturbances, which she described as “black spots” in the visual field, lasting 20 to 30 minutes, which resolved spontaneously and completely. During a dialysis session the week before presentation, she lost consciousness when her blood pressure went “very low.” After regaining consciousness, she noted “spots” in both eyes off to the right side of her visual field. The patient reported that her blood pressure was 79/45 mm Hg on the monitor at that time. She also reported that the “spots” cleared somewhat after the episode. During dialysis later that week, a similar event occurred, but the patient reported that her vision subsequently cleared back to its baseline. In the period surrounding these events, the patient’s mean blood pressure before dialysis sessions was

156/78 mm Hg, and after dialysis sessions was 150/76 mm Hg while sitting, with systolic and diastolic blood pressures ranging from 102 to 214 mm Hg and 49 to 118 mm Hg, respectively. She had experienced several episodes of intradialytic hypotension, which responded to 100 mL bolus normal saline. Her hemoglobin was 13.3 g/dL (133 g/L) and hematocrit was 39.7%.

Neuro-ophthalmologic examination found a visual acuity of 20/25+2 in the right eye and 20/25-2 in the left eye. Her pupils reacted briskly without a relative afferent pupillary defect. Automated perimetry found a right homonymous superior quadrantic defect (Fig 1). She had mild nuclear sclerotic cataracts consistent with her visual acuity in each eye. Funduscopy showed normal optic nerves and background diabetic retinopathy.

Magnetic resonance imaging of the brain showed abnormal signal in the left medial occipital lobe consistent with acute infarction without hemorrhage (Fig 2), a probable subcortical infarction in the right cerebellar hemisphere, and minimal diffuse microvascular ischemic changes.

Her dialysis center was contacted and the treating physician notified that intradialytic hypotension was the likely cause of her visual field defect. The dialysis center changed the treatment protocol to decrease the amount of fluid removed during each session and to decrease the rate at which it was removed. The patient also was instructed not to

From the Neuro-Ophthalmology Service, Cullen Eye Institute, Baylor College of Medicine, Houston, TX.

Received October 22, 2003; accepted in revised form January 20, 2004.

Address reprint requests to Rod Foroozan, MD, Neuro-Ophthalmology Service, Cullen Eye Institute, Baylor College of Medicine, 6565 Fannin NC-205, Houston, TX 77030. E-mail: foroozan@bcm.tmc.edu

© 2004 by the National Kidney Foundation, Inc.

0272-6386/04/4305-0026\$30.00/0

doi:10.1053/j.ajkd.2004.01.017

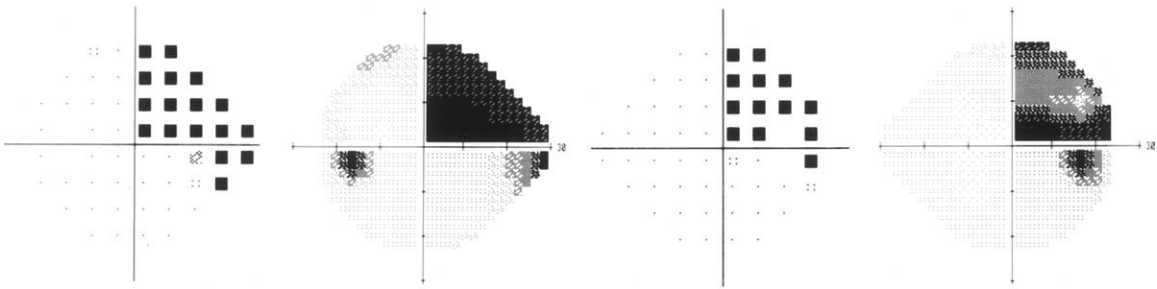


Fig 1. Automated perimetry (Humphrey 24-2 with pattern deviation and gray scale) shows a right homonymous superior quadrantic defect.

take her antihypertensive medications on the days of hemodialysis. She subsequently reported that the episodes of symptomatic intradialytic hypotension ceased, and she had no new visual symptoms.

Five months later, the patient reported a subjective improvement in her visual fields, and automated perimetry showed an improvement but persistence of the right homonymous superior quadrantic defect. The remainder of the examination remained unchanged.

DISCUSSION

Visual loss as a complication of hemodialysis is uncommon, is usually caused by ischemic optic neuropathy, and often occurs in the setting of intradialytic hypotension.¹⁻⁷ We reviewed the literature (www.pubmed.com, September 2003, using the following terms: “blindness,” “visual

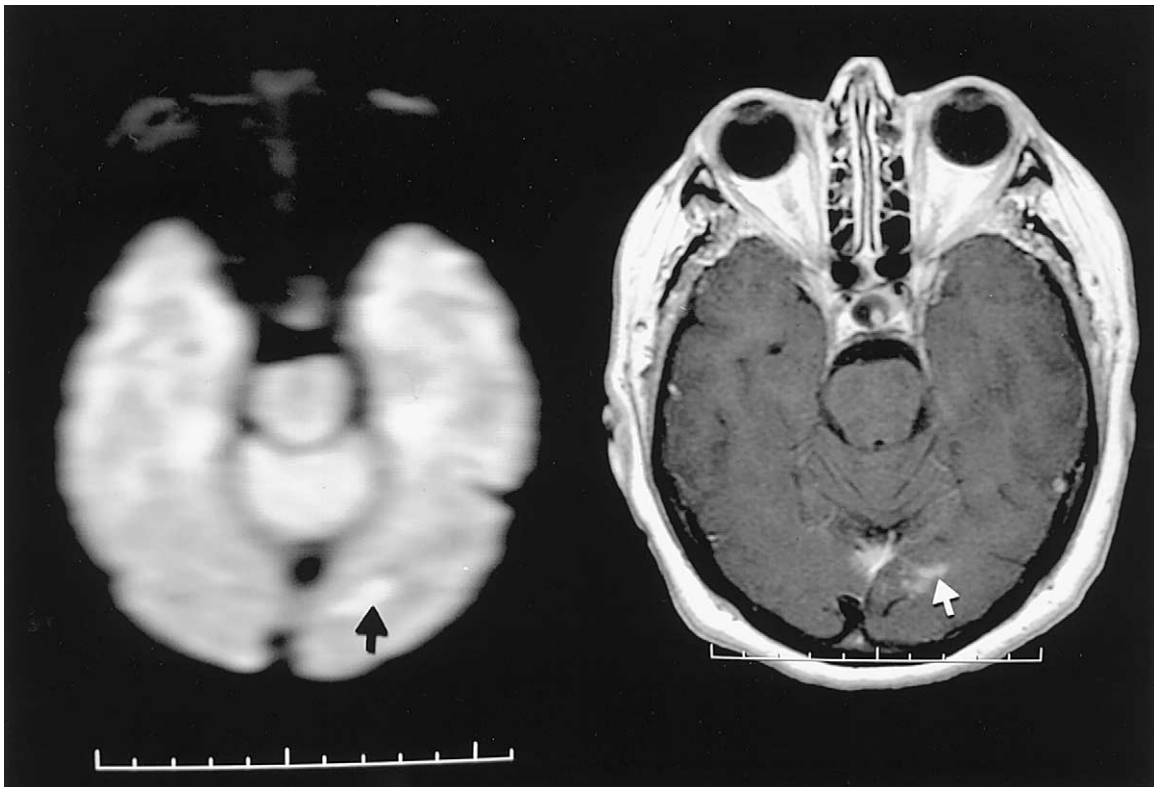


Fig 2. Axial magnetic resonance imaging scans, diffusion-weighted image (left) and contrast-enhanced T₁-weighted image (right), of the brain shows abnormal signal in the left medial occipital lobe (arrows) consistent with acute infarction without hemorrhage.

Table 1. Prior Reports of Visual Loss Related to Hemodialysis

Study	Age/Sex	Cause of Visual Loss	Timing of Visual Loss	Visual Acuity	Visual Field
1. Servilla and Groggel ¹	30/M	AION	Some sensation of "lights" and difficulty focusing after a hemodialysis session that included an episode of intradialytic hypotension; visual loss reported the next morning	20/30 OD 20/20 OS	Peripheral constriction OD; loss of superior field OS
2. Haider et al ²	53/M	AION	Rapidly progressive blurred vision of inferior visual field OS; the week before presentation, several episodes of intradialytic hypotension	6/5 OD 6/6 OS	Generalized constriction with inferior arcuate OS
3. Connolly et al ³	39/M	AION	Blurred vision OS immediately following hemodialysis with intradialytic hypotension	20/20 OD 20/800 OS	*
4. Koos and Munteanu ⁴	22/M	AION	Rapidly progressive visual loss OU during hemodialysis session	"Significant visual loss"	*
5. Sabeel et al ⁵	40/F	AION	Decreased vision OD after a hemodialysis session with intradialytic hypotension; decreased vision OS within 24 h	NLP OU	*
6. Basile et al ⁶	49/M	AION	Upon awakening in the morning OS; subsequent AION OD 9 mo later	LP OU	*
7. Buono et al ⁷	42/M	PION	Several hours after a hemodialysis session with intradialytic hypotension	NLP OU	*
8. Al-Falki ⁸	60/M	Cortical stroke	After a hemodialysis session without hypotension	LP OU	*
9. Moel and Kwun ⁹	5/M	Cortical ischemia	After intradialytic seizure; resolved fully over 2 mo	NLP OU	*
10. Arora et al ¹⁰	39/F	Purtscher's-like retinopathy owing to leukoembolization secondary to complement activation as a consequence of hemodialysis procedure	After hemodialysis	20/400 OU initially; improved to 20/50 OD and 20/200 OS over 7 mo	*
11. Hutter et al ¹¹	7 patients (25-65 years; 5 women)	Site of visual loss not reported, although felt to be an adverse reaction to aged cellulose acetate membranes	Within 48 h of hemodialysis	Varied (CF, 20/40, HM, CF, LP, CF, NLP)	*

Abbreviations: AION, anterior ischemic optic neuropathy; PION, posterior ischemic optic neuropathy; OD, right eye; OS, left eye; OU, both eyes; CF, count fingers; HM, hand motion; LP, light perception; NLP, no light perception.

*Visual fields were either not performed or not reported.

loss," "vision loss," "intradialytic," "hypotension," "dialysis," and "hemodiafiltration" and the Medical Subject Headings [MeSH] equivalents in various combinations) and were unable to find another report of fixed visual loss resulting from occipital infarction attributed to intradialytic hypotension. Hypotension was the preceding factor in a majority of the prior reports of visual loss associated with hemodialysis (Table 1), but none noted preceding episodes of transient visual loss. Hemodialysis-related visual loss was not attributed to hypotension in 4 reports, which included visual loss attributed to cerebral ischemia and infarction either after hemodialysis⁸ or owing to the disequilibrium syndrome,⁹ Purtscher's-like retinopathy,¹⁰ and toxic effects from aged dialyzers.¹¹

The patterns of visual loss caused by ischemia can be accounted for by the vascular anatomy supplying the visual pathway. The central retinal artery supplies the inner one third of the retina, whereas the choroidal vasculature supplies the remaining two thirds. Occlusion of the central retinal artery (or its source, the ophthalmic and internal carotid arteries) can result in monocular visual loss. The optic nerve has anterior and posterior circulations, which arise from the ciliary and ophthalmic arteries, respectively, and compromise of either vascular supply results in anterior ischemic optic neuropathy or posterior ischemic optic neuropathy, respectively. In the cerebral cortex, the optic radiations are usually supplied by the middle cerebral artery, whereas the blood supply of the visual cortex (calcarine fissure) in the occipital lobe is derived principally from the posterior cerebral arteries, with some collateral supply from the middle and anterior cerebral arteries. Infarcts in this area tend to cause quadrantanopias if only the superior or inferior portion of the calcarine fissure is involved unilaterally, or hemianopias when both superior and inferior portions are involved.

Symptomatic intradialytic hypotension is a common complication of hemodialysis, occurring in approximately 10% to 30% of all patients, and this overall rate has remained relatively unchanged despite improvements in dialysis techniques.^{12,13} Although the pathophysiology is complex, the most important factor appears to be from an ultrafiltration-induced reduction in blood volume.^{12,14,15} Proposed guidelines to prevent

episodes of intradialytic hypotension and decrease its associated morbidity include, but are not limited to, identifying the patient at risk for intradialytic hypotension, using morning or fasting dialysis, using sympathomimetic drugs before hemodialysis, avoiding high rates of ultrafiltration, and avoiding antihypertensive medications before dialysis.^{12,16}

The mechanism of the initial transient visual disturbances in our patient was likely caused by temporary occipital hypoperfusion during episodes of intradialytic hypotension, similar to a transient ischemic attack. The loss of consciousness at the time of the development of the visual deficit suggests that she had a more severe episode of hypoperfusion, which resulted in the left inferior occipital lobe infarct and the fixed visual field defect. Recognition of the transient visual disturbances and treatment of the associated intradialytic hypotension may have prevented the occipital stroke in our patient. After our patient's occipital stroke, her episodes of symptomatic intradialytic hypotension were alleviated by decreasing the rate and total volume of fluid removed and by avoiding antihypertensive medications before hemodialysis.

Transient visual loss may anticipate occipital stroke, especially in the setting of intradialytic hypotension. Treatment of intradialytic hypotension may avoid potentially devastating visual complications.

REFERENCES

1. Servilla KS, Groggel GC: Anterior ischemic optic neuropathy as a complication of hemodialysis. *Am J Kidney Dis* 8:61-63, 1986
2. Haider S, Astbury NJ, Hamilton DV: Optic neuropathy in uraemic patients on dialysis. *Eye* 7:148-151, 1993
3. Connolly SE, Gordon KB, Horton JC: Salvage of vision after hypotension-induced ischemic optic neuropathy. *Am J Ophthalmol* 117:235-242, 1994
4. Koos MJ, Munteanu G: Anterior ischemic optic neuropathy after systemic hypotension and anemia. *Oftalmologia* 44:79-83, 1998
5. Sabeel A, Al-Hazzaa SA, Alfurayh O, Mikkonen P: The dialysed patient who turned blind during a haemodialysis session. *Nephrol Dial Transplant* 13:2957-2958, 1998
6. Basile C, Addabbo G, Montanaro A: Anterior ischemic optic neuropathy and dialysis: Role of hypotension and anemia. *J Nephrol* 14:420-423, 2001
7. Buono LM, Foroozan R, Savino PJ, Danesh-Meyer HV, Stanescu D: Posterior ischemic optic neuropathy after hemodialysis. *Ophthalmology* 110:1216-1218, 2003

8. Al-Falki YH: Bilateral occipital infarcts in a hemodialysis patient. *Am J Nephrol* 21:413-414, 2001
9. Moel DI, Kwun YA: Cortical blindness as a complication of hemodialysis. *J Pediatr* 93:890-891, 1978
10. Arora N, Lambrou FH, Stewart MW, Vidrine-Parks L, Sandroni S: Sudden blindness associated with central nervous symptoms in a hemodialysis patient. *Nephron* 59:490-492, 1991
11. Hutter JC, Kuehnert MJ, Wallis RR, et al: Acute onset of decreased vision and hearing traced to hemodialysis treatment with aged dialyzers. *JAMA* 283:2128-2134, 2000
12. Zucchelli P, Santoro A: Dialysis-induced hypotension: A fresh look at pathophysiology. *Blood Purif* 11:85-98, 1993
13. Zucchelli P, Santoro A: The management of hypotension in dialyzed patients. *Miner Electrolyte Metab* 25:105-108, 1999
14. Sherman R: Intradialytic hypotension: An overview of recent, unresolved and overlooked issues. *Semin Dial* 15:141-143, 2002
15. Andrulli S, Colzani S, Mascia F, et al: The role of blood volume reduction in the genesis of intradialytic hypotension. *Am J Kidney Dis* 40:1244-1254, 2002
16. Passauer J, Büssemaker E, Gross P: Dialysis hypotension: Do we see light at the end of the tunnel? *Nephrol Dial Transplant* 13:3024-3029, 1998