Reminder of important clinical lesson

Non-arteritic ischaemic optic neuropathy after first return to haemodialysis

Sarah Bartlett, Andrew Cai, Hugh Cairns

Renal Medicine, Kings College Hospital NHS Foundation Trust, Denmark Hill, London, United Kingdom

Correspondence to Miss Sarah Bartlett, sarah.bartlett@kcl.ac.uk**Summary**

The authors present an interesting case of non-arteritic anterior ischaemic optic neuropathy (NAION) after 2 h of haemodialysis, in a 42-year-old Caucasian woman with anaemia (haemoglobin 7.1 g/dl) uraemia (56.3 mmol/l), spurious hypotension (42/26 mm Hg) and previous history of anterior ischaemic optic neuropathy (AION) in the other eye. Thirteen cases of haemodialysis related NAION in adults have been reported to date. NAION is produced when oxygen delivery to the optic nerve is compromised resulting in hypoxic swelling, nerve compression in the optic canal and ischaemia of the optic nerve head. It typically presents as a sudden, unilateral and painless inferior visual field defect with a fixed unreactive pupil after a period of relative hypotension. Clinicians should be aware of this unusual complication when considering and explaining dialysis options, particularly in patients with other risk factors such as hypotension, anaemia, significant uraemia and previous history of AION

BACKGROUND

We present an interesting case of non-arteritic anterior ischaemic optic neuropathy (NAION) after haemodialysis. Visual loss due to NAION is a rare but important complication of haemodialysis, with 13 cases reported in these patients.^{1–11} Therefore, this condition may be more common, than traditionally thought in patients on renal replacement therapy.

The aetiology of this condition is complex and frequently misunderstood.¹³ In the dialysis-dependent population, hypotension, anaemia and uraemia are known to be important risk factors^{1 12 13}

CASE PRESENTATION

A 42-year-old woman was admitted with abdominal pain due to peritoneal dialysis (PD)-related peritonitis while on holiday, on a background of ischaemic heart disease and end-stage renal disease due to immunoglobulin A nephropathy (ESRD). Over the past 20 years, she had been on all forms of renal replacement therapy, including three failed renal transplants and haemodialysis.

She had been switched to PD, 2 months previously, after haemodialysis had failed and further vascular access had been deemed impossible. Three months prior to admission, she lost all her vision in her right eye and was noted to have a swollen right optic disc. She had also suffered a haemorrhage from a duodenal ulcer 2 weeks prior to this admission, which required management with sclerosant injection by endoscopy as an inpatient.

On admission to the renal unit, her blood pressure was recorded as 46/26 mm Hg. However, this was thought to be spurious due to her history of failed fistula formations and known vascular occlusions. Her blood tests showed her to have anaemia (Hb 7.1 g/dl) uraemia (56.3 mmol/l) and a high serum creatinine (1079 µmol/l), in keeping with her renal disease.

The PD catheter was removed because of the peritonitis and extrusion of the external PD catheter cuff. The patient was haemodialysed for 2 h via a temporary femoral haemodialysis catheter (VasCath; Teleflex Medical, Athlone,

Ireland) one day after admission. She was hypotensive throughout her treatment, with a postdialysis blood pressure of 60/30 mm Hg (again possibly spurious). She also received 2 units of packed red cells during this haemodialysis session. However, she remained asymptomatic throughout her treatment and well in herself. That night, she complained of clouding of vision in her left (good) eye, but only reported loss of visual acuity to the ward staff the next morning, 12-h postdialysis.

INVESTIGATIONS

On examination, her visual acuity in the left eye was found to be reduced to hand waving and absent to light reflex in the right eye. Both the pupils were dilated and unreactive. Intraocular pressure was 8 mm Hg in both the eyes. On funduscopy and retinal imaging, the right optic disc was pale and the left disc was white and swollen with two flame haemorrhages at 9 and 10 o'clock (figure 1) positions. There was no evidence of cupped discs. Extraocular movements were normal and the remainder of the neurological exam was unremarkable. Temporal arteries were non-palpable and non-tender.

Repeat blood tests showed that serum urea concentration had fallen postdialysis to 40.6 mmol/l. An optical coherent tomography scan showed thinning of the retinal nerve fibre layer of the right eye, consistent with long-standing atrophy of the optic nerve. There was thickening of the retinal nerve fibre layer in the left eye, consistent with acute optic neuritis (figure 2). MRI of the optic nerves showed that the right intraorbital segment of the optic nerve was atrophic; there were no other significant abnormalities.

Figure 1 Retinal photographs showing pallor of the right optic disc (OD) and oedema and flame haemorrhages of the left oculus sinister (OS).

Figure 2 Optic coherent tomography of right (OD) and left (OS) retinal nerve fibre layers (RNFL). Thinning on the right shown. Left RNFL is hypertrophied. The black trace corresponds to the thickness recorded, the green zone shows the normal range and the red area indicates abnormal thinning of the RNFL.

DIFFERENTIAL DIAGNOSIS

In light of the clinical findings and risk factors, glaucoma and arteritic AION were excluded and a diagnosis of NAION was made.

TREATMENT

She was managed conservatively on advice from the ophthalmology team with correction of her hypotension and a gradual reduction in her serum urea concentration. After extensive discussion with the patient, she continued on haemodialysis with a view to returning to PD at a later stage. She was not started on a trial of steroids due to the lack of current evidence.

OUTCOME AND FOLLOW-UP

She was discharged from the hospital 3 weeks later and returned to the care of her local team. Her vision was unchanged on discharge.

DISCUSSION

NAION is a debilitating complication that may be associated with haemodialysis. It typically presents as a painless unilateral loss of vision with inferior altitudinal visual field defects and a fixed dilated pupil.¹³

NAION is thought to be caused by ischaemia of the optic nerve head, frequently produced by hypoperfusion of the posterior ciliary arteries. Hypoxia of the nerve fibres

results in axonal hypoxic swelling. As the optic nerve is contained within the bony optic canal, any increase in volume compresses the microvasculature and further compromises perfusion. Factors that reduce the oxygen-carrying capacity of the blood, such as anaemia, will exacerbate hypoxia in underperfused tissues.¹³

It is not all or nothing – the degree of visual loss is dependent on the severity and duration of the hypoperfusion and is typically less severe than that of arteritic neuropathy.¹³ Of the cases that we reviewed, include reports of visual disturbances ranged from blurring⁸ to no light perception.⁷ These cases occurred in long standing dialysis patients who had been on haemodialysis for many years.

In this case, our patient demonstrated a number of risk factors for this condition. She was anaemic, hypotensive and significantly uraemic and her hypotension was probably exacerbated during haemodialysis.

Hypotension is well described in association with NAION; there have been 13 cases published of non-arteritic AION in the adult haemodialysis population, of which 12 patients were hypotensive.^{1–11} The range of blood pressures reported across the hypotensive cases is from 52/38 mm Hg¹⁰ to 90/60 mm Hg.⁹ Paradoxically, NAION has also been described in association with sudden relative hypotension during dialysis, even when blood pressure may not fall below the traditionally-accepted normal range.⁴ Sudden relative hypotension is probably the most important causative factor in both dialysis-dependent patients^{2–4 12} and the general population.¹³ Intradialytic hypotension is a common complication of dialysis. During a sudden change in perfusion, autoregulatory mechanisms do not have time to respond and perfusion pressure is unable to overcome the extraneural pressure exerted by the intraocular pressure.¹³ Rapid fluctuations in blood pressure are more common during haemodialysis than in both the general population and patients on PD and therefore NAION is more common in this group.

Most of the cases described patients on an established dialysis modality, the average being 10 years (range: 3–20 years).^{1–11} To the best of our knowledge, there are no reports of a patient losing vision after the first session of haemodialysis. Although our patient had been on haemodialysis in the past, at the time of the report, she was well established on PD and had felt confident enough to go on a holiday overseas.

In 73% of cases, visual loss is reported on waking in the morning.¹³ Twenty-four hours ambulatory blood pressure monitoring has shown marked nocturnal hypotension in cases of AION discovered in the morning.¹³ This is in keeping with our case as our patient's symptoms were only reported in the morning after dialysis. Her recorded blood pressure was again noted to be 61/26 mm Hg and the medical team felt that the readings continued to be

spurious and not in keeping with the complete clinical picture.

Anaemia is common in patients with ESRD, the association of NAION with hypotension and anaemia was further demonstrated in a longitudinal study of 60 dialysis-dependent patients, of which four developed AION. The reported AION was attributed to hypotension in one case, anaemia in one case and atherosclerosis in the other two.¹²

Recovery has been shown to be related to the severity and duration of ischaemia and is usually unpredictable.¹³ The management is limited to alleviating the precipitating factors and minimising the predisposing ones as far as possible.

Learning points

- ▶ NAION is a rare occurrence in haemodialysis
- ▶ Risk factors are hypotension, anaemia, significant uraemia and previous history of AION
- ▶ Clinicians should be aware of this unusual complication when considering and explaining dialysis options, particularly in patients with multiple risk factors.

Competing interests None.

Patient consent Obtained.

REFERENCES

1. **Sabeel A**, Al-Hazzaa SA, Alfurayh O, *et al*. The dialysed patient who turned blind during a haemodialysis session. *Nephrol Dial Transplant* 1998;**13**:2957–8.
2. **Servilla KS**, Groggel GC. Anterior ischemic optic neuropathy as a complication of hemodialysis. *Am J Kidney Dis* 1986;**8**:61–3.
3. **Korzets A**, Marashek I, Schwartz A, *et al*. Ischemic optic neuropathy in dialyzed patients: a previously unrecognized manifestation of calcific uremic arteriolopathy. *Am J Kidney Dis* 2004;**44**:e93–7.
4. **Connolly SE**, Gordon KB, Horton JC. Salvage of vision after hypotension-induced ischemic optic neuropathy. *Am J Ophthalmol* 1994;**117**:235–42.
5. **Basri NA**, Shaheen FA. Visual loss in uremic patients on dialysis: a case report and review of literature. *Saudi J Kidney Dis Transpl* 2002;**13**:45–9.
6. **Koos MJ**, Munteanu G. Anterior ischemic optic neuropathy after systemic hypotension and anemia. *Ophthalmologia* 1998;**44**:79–83.
7. **Nieto J**, Zapata MA. Bilateral anterior ischemic optic neuropathy in patients on dialysis: A report of two cases. *Indian J Nephrol* 2010;**20**:48–50.
8. **Basile C**, Addabbo G, Montanaro A. Anterior ischemic optic neuropathy and dialysis: role of hypotension and anemia. *J Nephrol* 2001;**14**:420–3.
9. **León L**, Marinero C. Anterior ischemic optic neuropathy in haemodialysis. *Nefrologia* 2009;**29**:614–16.
10. **Michaelson C**, Behrens M, Odel J. Bilateral anterior ischaemic optic neuropathy associated with optic disc drusen and systemic hypotension. *Br J Ophthalmol* 1989;**73**:762–4.
11. **Cuxart M**, Matas M, Picazo M, *et al*. Acute bilateral visual loss in a hemodialysed patient. *Nefrologia* 2005;**25**:703–5.
12. **Haider S**, Astbury NJ, Hamilton DV. Optic neuropathy in uraemic patients on dialysis. *Eye (Lond)* 1993;**7**(Pt 1):148–51.
13. **Hayreh SS**. Ischemic optic neuropathy. *Prog Retin Eye Res* 2009;**28**:34–62.

This pdf has been created automatically from the final edited text and images.

Copyright 2011 BMJ Publishing Group. All rights reserved. For permission to reuse any of this content visit <http://group.bmj.com/group/rights-licensing/permissions>.
BMJ Case Report Fellows may re-use this article for personal use and teaching without any further permission.

Please cite this article as follows (you will need to access the article online to obtain the date of publication).

Bartlett S, Cai A, Cairns H. Non-arteritic ischaemic optic neuropathy after first return to haemodialysis. *BMJ Case Reports* 2011; 10.1136/bcr.04.2011.4072, date of publication

Become a Fellow of BMJ Case Reports today and you can:

- ▶ Submit as many cases as you like
- ▶ Enjoy fast sympathetic peer review and rapid publication of accepted articles
- ▶ Access all the published articles
- ▶ Re-use any of the published material for personal use and teaching without further permission

For information on Institutional Fellowships contact consortiasales@bmjgroup.com

Visit casereports.bmj.com for more articles like this and to become a Fellow