Case Report

Improvement in open-angle glaucoma by nocturnal home haemodialysis

Huseyin Kocak¹, Joseph Ly² and Christopher T. Chan²

¹Department of Medicine, Division of Nephrology, Akdeniz University School of Medicine Hospital, Antalya, Turkey and ²Department of Medicine, Division of Nephrology, Toronto General Hospital – University Health Network, University of Toronto, Toronto, Canada

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Introduction

Open-angle glaucoma (OAG) is a common ocular abnormality seen in patients with end-stage renal disease (ESRD) [1]. Although the pathogenesis of OAG is not yet fully understood, lowering intraocular pressure is the accepted treatment goal. Without adequate treatment, glaucoma can lead to visual disability and eventual blindness [2].

From a simplified viewpoint, intraocular pressure is determined by the balance between aqueous production from the ciliary body and drainage of the aqueous humour through the trabecular meshwork. Normal intraocular pressure (IOP) ranges from 10 to 21 mmHg. IOP of 20–30 mmHg usually results in chronic optic nerve damage. In cases of significant intraocular hypertension (40–50 mmHg), rapid visual loss and retinovascular occlusion may occur.

Intradialytic alteration of IOP by conventional haemodialysis (CHD) has been well-documented [3,4]. It is generally accepted that there are two opposing forces, which may impact on IOP during haemodialysis: ultrafiltration and solute removal. Tokuyama et al. [3] documented a fall in IOP which was inversely correlated with the increase in plasma oncotic pressure caused by ultrafiltration. In contrast, other authors speculated that the rapid removal of uraemic toxins via CHD may lower plasma osmolality at a rate in excess of changes in ocular osmolality resulting in an acute rise in IOP [5].

Nocturnal haemodialysis (NHD), which provides 8–10 h of renal replacement therapy during sleep, 5–7 nights per week, is a form of intensive haemodialysis which approximates most closely to normal physiology [6]. Here, we report our index case in which the conversion from CHD to NHD is associated with a restoration in IOP. We will further examine the potential interactions between augmentation of uraemia clearance, enhanced volume control and changes in total peripheral resistance in the treatment of glaucoma with NHD in ESRD patients.

Case history

A 59-year-old woman with ESRD secondary to type 2 diabetes mellitus was referred for conversion to NHD because of hypertension and intradialytic haemodynamic instability.

Her past medical history was significant for refractory hypertension, hyperthyroidism, diabetic retinopathy and OAG. The patient was originally initiated on CHD in 2000. While on CHD, the patient required: lisinopril 10 mg/day, metoprolol 100 mg/day for blood pressure control, calcium carbonate 4 g/day for phosphate binding, and multi-dose insulin regimen for glycaemic control. Diagnosis of bilateral OAG was made 2 years prior to the initiation of her renal replacement therapy. From 1998 to 2001, her IOP was repeatedly documented to be >30 mmHg in both eyes despite the compliant use of timolol ophthalmic solution.

CHD was performed using F80 polysulfone dialysers (Fresenius Medical Care, Lexington, MA, USA). The dialysate composition was as follows: Na⁺ 140 mM, K⁺ 1–3 mM, Ca²⁺ 1.25–1.5 mM, and HCO₃⁻ 40 mM. A blood flow rate of 400 ml/min and a dialysate flow rate of 500–750 ml/min were used.

Due to intolerance of CHD and poor blood pressure control, the patient was converted to NHD therapy in 2001. Dialysate composition was similar to that used for CHD therapy. Dialysate flow rate was 350 ml/min; blood flow rate was 300 ml/min and a Polyflux-17 polyamide dialyser (Gambro Inc, Hechnigen, Germany) was used. Expectedly, there was an increase in uraemia clearance after conversion to NHD. Serum phosphate concentration and blood pressure also fell
Despite the withdrawal of calcium-based phosphate binders and anti-hypertensive therapies (Table 1). After 3 months of NHD, her IOP decreased to 21 mmHg in both eyes. Six months after conversion to NHD, her IOP has returned to normal bilaterally (11 mmHg) and has remained normal to present. Concomitantly, her visual acuity improved without the need for pharmacotherapy.

**Discussion**

OAG is a common ocular abnormality seen in ESRD patients. Without adequate treatment, OAG may lead to visual loss. To our knowledge, this is the first reported case of restoration of normal IOP with the use of intensive haemodialysis modality such as NHD. By increasing the dialysis frequency and duration of therapy, NHD offers superior management of solute and fluid control in comparison with standard CHD [7]. In addition, NHD has been previously shown to improve flow-mediated dilation [8] and peripheral vascular disease [9] in ESRD patients. We speculate that all the three factors, namely: (i) augmentation of uraemia clearance, (ii) enhanced volume control and (iii) changes in total peripheral resistance are responsible for the observed improvement in IOP seen in our patient. Each of these hypotheses will be discussed in turn.

Rapid removal of small solutes has been associated with a rise in IOP [10]. In fact, Tovbin et al. [11] studied 19 chronic stable CHD patients and correlated post-dialysis urea rebound with changes in IOP. These investigators found that post-dialysis urea rebound was positively correlated to mean intra-dialytic changes in IOP [11]. This observation suggests that fast removal of small solutes causes a detrimental rapid shift in small molecules in comparison with intraocular osmolality, which results in an intra-dialytic gain in fluid in the ocular compartment and thus a higher IOP, especially towards the end of dialysis. In contrast, by increasing dialysis frequency and duration, NHD minimizes the potential for small molecular rebound. We observed a sustained decrease in pre- and post-dialysis plasma urea concentrations after conversion to NHD (Table 1). It is tempting to speculate that an increase in solute clearance by NHD will decrease the potential to develop intraocular hypertension by minimizing rapid small molecular shifts.

Ultrafiltration is noted to decrease IOP during CHD sessions [10]. Leiba and colleagues [10] examined 30 non-glaucomatous patients with ESRD and demonstrated that IOP decreased significantly after ultrafiltration alone in comparison with a rise after CHD. It is reasonable to propose that we were able to remove additional extracellular fluid from our patient while on NHD in comparison with her CHD regimen (Table 1). An improved time-averaged volume control may ultimately lead to a beneficial modification of IOP.

However, alterations in fluid shifts in the ocular compartment during dialysis are not likely by themselves sufficient to explain the restoration of IOP and visual acuity observed in our patient. Several studies have linked the impairment in vascular function with the development of OAG [12,13]. It was postulated that due to abnormal resistance vessels in the eyes, patients are more susceptible to develop OAG because of an inability of the optic resistance vessels to dilate, resulting in a functional outflow obstruction of the aqueous humour. It is interesting to note that NHD has been documented to have several vascular advantages in comparison with CHD, including: (i) augmentation in flow-mediated dilation [8], (ii) restoration of total peripheral resistance [8] and (iii) improvement in peripheral Doppler flow [9]. It is reasonable to propose that amelioration in optic resistance arteries responsiveness via augmentation of uraemia clearance may lead to an improvement in the aqueous humour outflow, resulting in a fall in IOP.

Previous modifications of dialytic procedures [14,15] have been used to prevent further increase in IOP during renal replacement therapy. Our report is unique in describing a potential therapeutic use of NHD to restore IOP in a highly susceptible patient population. In contrast, renal transplantation, due to the use of

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**Table 1. Changes in biochemical and clinical characteristics before and after conversion to nocturnal haemodialysis**

<table>
<thead>
<tr>
<th>Variables</th>
<th>CHD</th>
<th>NHD (3 months)</th>
<th>NHD (6 months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dialysis (h)</td>
<td>4</td>
<td>7.5</td>
<td>7.5</td>
</tr>
<tr>
<td>Frequency of dialysis (treatments/week)</td>
<td>3</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Equilibrated Kt/V (daily/weekly)</td>
<td>1.32/3.95</td>
<td>2.5/12.5</td>
<td>2.24/11.2</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>73.8</td>
<td>70.6</td>
<td>68</td>
</tr>
<tr>
<td>Pre-dialysis/post-dialysis plasma creatinine concentrations (µmol/l)</td>
<td>718/304</td>
<td>444/111</td>
<td>382/103</td>
</tr>
<tr>
<td>Pre-dialysis/post-dialysis plasma urea concentrations (µmol/l)</td>
<td>22.4/7.7</td>
<td>14.5/2.2</td>
<td>14.3/2.5</td>
</tr>
<tr>
<td>Pre-dialysis/post-dialysis plasma phosphate concentrations (mmol/l)</td>
<td>1.25/0.46</td>
<td>0.8/0.52</td>
<td>1.0/0.6</td>
</tr>
<tr>
<td>Serum parathyroid hormone (pmol/l)</td>
<td>1.5</td>
<td>1.4</td>
<td>1.3</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>157</td>
<td>132</td>
<td>134</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>99</td>
<td>80</td>
<td>84</td>
</tr>
<tr>
<td>Antihypertensive agents</td>
<td>2</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Intraocular pressure (mmHg)</td>
<td>&gt;30</td>
<td>21</td>
<td>11</td>
</tr>
</tbody>
</table>

CHD, conventional haemodialysis; NHD, nocturnal haemodialysis.
steroid, has been associated with the development of glaucoma [16]. Further mechanistic and clinical studies are required to fully understand the impact of intensive haemodialysis on the treatment of OAG in ESRD patients.

Conflict of interest statement. None declared.

References

2. Weinreb RN, Khaw PT. Primary open-angle glaucoma. Lancet 2004; 363: 1711–1720

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