Effect of Hemodialysis on Intraocular Pressure and Ocular Perfusion Pressure

Jennifer Hu, MD; Kelly M. Bui, MD; Kevin H. Patel, MD; Hajwa Kim, MA, MS; Jose A. L. Arruda, MD; Jacob T. Wilensky, MD; Thasarat S. Vajaranant, MD

IMPORTANCE Elevated intraocular pressure (IOP) and decreased ocular perfusion pressure (OPP) are risk factors for glaucoma development and progression. Unrecognized significant IOP elevation or OPP reduction during hemodialysis (HD) could lead to glaucomatous optic nerve damage and subsequent visual loss.

OBJECTIVE To evaluate changes in IOP and OPP during HD.

DESIGN, SETTING, AND PARTICIPANTS A cross-sectional observational study was conducted in patients undergoing HD at an ambulatory care clinic at the University of Illinois at Chicago.

EXPOSURES Forty-nine patients (97 eyes) undergoing HD were enrolled. Exclusion criteria included preexisting corneal abnormalities, history of corneal surgery, allergy to topical anesthetic agents, and current eye infection. Nine patients had previous diagnoses of open-angle glaucoma (OAG) or suspected glaucoma. At 3 time points, IOP was measured with the patient in a seated position approximately 15 minutes before starting HD (T1), approximately 2 hours after starting HD (T2), and approximately 15 minutes after ending HD (T3). Mean arterial pressure (MAP) and OPP (systolic, diastolic, and mean OPP) were calculated.

MAIN OUTCOMES AND MEASURES Intraocular pressure and OPP.

RESULTS From T1 to T3, IOP significantly increased by 3.1 mm Hg (both eyes, \( P < .001 \)), MAP significantly decreased by 5.8 mm Hg (\( P = .05 \)), and all OPP measures significantly decreased from baseline (all \( P \leq .02 \)). Using previously reported thresholds of increased glaucoma development and progression risk, 53% of the right eyes (26 of 49) and 46% of the left eyes (22 of 48) had a systolic OPP of 101 mm Hg or less, 71% of the right eyes (35 of 49) and 73% of the left eyes (35 of 48) had a diastolic OPP of 55 mm Hg or less, and 63% of the right eyes (31 of 49) and 65% of the left eyes (31 of 48) had a mean OPP of 42 mm Hg or less.

CONCLUSIONS AND RELEVANCE Significantly increased IOP and decreased OPP occur during HD, bringing both to levels that increase the risk of glaucoma development and progression. Clinicians should consider HD history in patients who have glaucoma progression, even when IOP has been well controlled. Such patients may benefit from IOP and blood pressure monitoring during HD sessions to minimize OPP changes resulting from IOP spikes and/or suboptimal blood pressure.
According to the National Institutes of Health, \(^2\) 871,000 Americans received treatment for end-stage renal disease in 2009, with 26% of these patients older than 60 years. Given that age is a risk factor for glaucoma, \(^2\) older patients with end-stage renal disease who are undergoing hemodialysis (HD) would be more likely to have glaucoma. Intraocular pressure (IOP) is a major risk factor for development and progression of glaucomatous disease, and transient increases in IOP have been reported during HD in patients with and without glaucoma. \(^3\)–\(^12\) The meaning of significant but transient IOP elevation during HD sessions, in terms of risk of glaucoma development and progression, is not known.

Adequate oxygenation of ocular tissues depends on maintenance of ocular perfusion pressure (OPP) through systemic regulation of blood pressure (BP) and local regulation of IOP. It has been proposed \(^13\)–\(^15\) that vascular dysregulation leads to abnormal ocular perfusion and thus optic nerve ischemia, serving as an underlying cause of glaucomatous damage. In several animal models of glaucoma, \(^16\)–\(^18\) researchers found that optic nerve hypoxia occurs when IOP increases to above 40 mm Hg or OPP decreases to below 50 mm Hg, when autoregulation can no longer compensate. Indeed, Stefánsson et al \(^16\) observed that the optic nerve can tolerate OPPs that are above 50 mm Hg, but in a variety of animal \(^17\)–\(^18\) and clinical \(^19\) studies, hypoxia ensued when OPP fell to below 30 mm Hg.

In humans, numerous population-based epidemiologic studies have shown that low OPP is associated with open-angle glaucoma (OAG). \(^15\)–\(^19\) The Barbados Eye Studies \(^24\)–\(^25\) showed that patients with a low baseline OPP had a higher risk for OAG than did patients with a higher baseline OPP 4 and 9 years later. Patients with a systolic OPP (SOPP) less than 101 mm Hg, a diastolic OPP (DOPP) less than 55 mm Hg, and a mean OPP (MOPP) less than 42 mm Hg were 2.6, 3.2, and 3.1 times more likely to have OAG, respectively. Similarly, Early Manifest Glaucoma Trial \(^26\) results indicated that a low baseline systolic perfusion pressure increased the hazard ratio for glaucoma progression to 1.42. All of these epidemiologic studies referred to stable and consistently low OPPs; HD potentially produces only a transient change in OPP. However, given the data indicating that low OPP is a risk factor for glaucoma development and progression, we chose to examine IOP and OPP changes occurring during individual HD sessions. We further investigated whether OPP levels reached thresholds associated with optic nerve damage.

Methods

The institutional review board of the University of Illinois at Chicago reviewed and approved this prospective study. Study participants provided written informed consent and were not given a stipend for study participation. The study was compliant with the Health Insurance Portability and Accountability Act and adhered to the tenets of the Declaration of Helsinki.

Forty-nine patients with end-stage renal disease who were undergoing maintenance HD at the University of Illinois at Chicago were enrolled in this prospective, cross-sectional, observational study. Dialysis was performed 3 times a week, with session durations of 3 to 5 hours. High-performance dialyzers (polysulfone) were used at a blood flow rate of 500 to 600 mL/min, and the systemic circulation was accessed through an arteriovenous fistula. Patients were excluded from participation if they had a preexisting corneal abnormality, a history of corneal surgery, an allergy to topical anesthetic agents, or a current eye infection.

Intracocular pressure was measured twice at each time point using a pneumotonometer (Model 30 Classic, Mentor; Reichart Technologies). The 2 measurements had a deviation of less than 0.5 mm Hg and were averaged into a single data point for all analyses. The IOP was measured with the patient in a seated position at each of the following time points: T1, approximately 15 minutes before starting HD; T2, approximately 2 hours after starting HD; and T3, approximately 15 minutes after ending HD.

The following formula was used to calculate mean arterial BP (MAP) at each time point: 

\[
MAP = \frac{1}{3} (systolic BP + diastolic BP)
\]

Systolic and diastolic BP were measured with an automated sphygmomanometer on the upper arm over the brachial artery with the patient sitting upright. The OPP at each time point was then calculated as 

\[
OPP = MAP - IOP.
\]

The SOPP, DOPP, and MOPP levels were also calculated for each time point using the following equations:

\[
SOPP = Systolic BP - IOP,
\]

\[
DOPP = Diastolic BP - IOP,
\]

\[
MOPP = \frac{1}{3} (MAP - IOP).
\]

Pre-HD and post-HD plasma osmolarity, colloid osmotic pressure, and body weight were also analyzed. Plasma osmolarity was calculated as: 

\[
\text{plasma osmolarity} = 2\left(\frac{\text{Na}}{\text{mM}}\right) + 0.16\left(\frac{\text{glucose}}{\text{mM}}\right),
\]

where Na indicates plasma sodium ion concentration (in millimoles per liter), glucose indicates plasma glucose concentration (in milligrams per deciliter), and SUN indicates levels of serum urea nitrogen (in milligrams per deciliter). Conversion of sodium to meq equivalents per liter is 1:1; to convert glucose and SUN to millimoles per liter, multiply by 0.0555 and 0.357, respectively.

Colloid osmotic pressure was calculated using the amount of total protein (TP) in the plasma (in grams per deciliter; to convert to grams per liter, multiply by 10) using the following formula: 

\[
\text{colloid osmotic pressure} = 2.1(\text{TP}) + 0.16(\text{TP})^3 + 0.009(\text{TP})^4.
\]

Finally, serum osmolarity rate of change was calculated in the following manner: (post-HD serum osmolarity – pre-HD serum osmolarity)/(HD duration).

Statistical Analysis

Comparisons between mean IOP, MAP, and OPP at T1, T2, and T3 were made using paired 2-tailed t tests. The Pearson correlation coefficient was calculated and used to determine the statistical significance of linear correlations. Commercial software (SAS, version 9.2; SAS Institute Inc) was used to perform all statistical analyses, and statistical significance was defined as \(P < .05\).
Results

This study included 97 eyes (49 right eyes and 48 left eyes) from 49 patients. Table 1 summarizes baseline characteristics. Five patients had previous diagnoses of OAG and 4 patients had suspected glaucoma. No patient experienced any significant adverse events during HD.

Table 1. Baseline Characteristics of 49 Participants

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean (SD) [range], y</td>
<td>56.7 (13.6) [26-86]</td>
</tr>
<tr>
<td>Sex</td>
<td></td>
</tr>
<tr>
<td>Male</td>
<td>28 (57)</td>
</tr>
<tr>
<td>Female</td>
<td>21 (43)</td>
</tr>
<tr>
<td>Race/ethnicity</td>
<td></td>
</tr>
<tr>
<td>African American</td>
<td>40 (82)</td>
</tr>
<tr>
<td>White</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Asian</td>
<td>1 (2)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>2 (4)</td>
</tr>
<tr>
<td>Unknown</td>
<td>5 (10)</td>
</tr>
<tr>
<td>Cause of chronic kidney disease</td>
<td></td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>16 (33)</td>
</tr>
<tr>
<td>Hypertension</td>
<td>15 (31)</td>
</tr>
<tr>
<td>Focal segmental glomerulosclerosis</td>
<td>5 (10)</td>
</tr>
<tr>
<td>Other</td>
<td>13 (26)</td>
</tr>
<tr>
<td>Glaucoma/suspected glaucoma</td>
<td>9 (18)</td>
</tr>
<tr>
<td>Diagnosed glaucoma</td>
<td>5 (10)</td>
</tr>
<tr>
<td>Suspected glaucoma</td>
<td>4 (8)</td>
</tr>
</tbody>
</table>

Table 2. Pressure Measurements and Calculations During Hemodialysis in All Participants Examined*

<table>
<thead>
<tr>
<th>Measurement, mm Hg</th>
<th>T1 Mean (SD)</th>
<th>T2 Mean (SD)</th>
<th>T3 Mean (SD)</th>
<th>Change From T1 to T2</th>
<th>Change From T2 to T3</th>
<th>Change From T1 to T3</th>
</tr>
</thead>
<tbody>
<tr>
<td>IOP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right eye</td>
<td>19.1 (6.1)</td>
<td>21.0 (5.5)</td>
<td>22.2 (5.6)</td>
<td>1.9 (4.1)</td>
<td>1.2 (3.4)</td>
<td>3.2 (3.8)</td>
</tr>
<tr>
<td>Left eye</td>
<td>17.7 (4.7)</td>
<td>19.2 (4.2)</td>
<td>20.8 (4.2)</td>
<td>1.4 (4.5)</td>
<td>1.6 (3.2)</td>
<td>3.1 (3.7)</td>
</tr>
<tr>
<td>MAP</td>
<td>100.9 (21.7)</td>
<td>95.8 (18.3)</td>
<td>95.1 (17.3)</td>
<td>−5.1 (17.3)</td>
<td>−0.7 (11.4)</td>
<td>−5.8 (20.1)</td>
</tr>
<tr>
<td>OPP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right eye</td>
<td>81.9 (21.2)</td>
<td>74.8 (18.9)</td>
<td>72.9 (17.5)</td>
<td>−7.1 (16.4)</td>
<td>−1.9 (11.5)</td>
<td>−8.9 (19.2)</td>
</tr>
<tr>
<td>Left eye</td>
<td>83.1 (20.1)</td>
<td>76.6 (18.2)</td>
<td>74.4 (16.7)</td>
<td>−6.5 (16.8)</td>
<td>−2.2 (11.8)</td>
<td>−8.7 (19.8)</td>
</tr>
<tr>
<td>SOPP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right eye</td>
<td>124.7 (28.8)</td>
<td>114.5 (27.8)</td>
<td>113.3 (25.7)</td>
<td>−10.2 (24.9)</td>
<td>−1.2 (18.1)</td>
<td>−11.4 (30.7)</td>
</tr>
<tr>
<td>Left eye</td>
<td>125.7 (27.8)</td>
<td>115.9 (26.9)</td>
<td>114.6 (25.4)</td>
<td>−9.7 (25.5)</td>
<td>−1.3 (18.5)</td>
<td>−11.0 (31.4)</td>
</tr>
<tr>
<td>DOPP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right eye</td>
<td>60.4 (19.7)</td>
<td>54.9 (16.5)</td>
<td>52.7 (15.5)</td>
<td>−5.5 (14.4)</td>
<td>−2.2 (10.0)</td>
<td>−7.7 (15.1)</td>
</tr>
<tr>
<td>Left eye</td>
<td>61.8 (18.5)</td>
<td>56.9 (16.0)</td>
<td>54.3 (14.5)</td>
<td>−4.9 (14.6)</td>
<td>−2.6 (10.2)</td>
<td>−7.5 (15.7)</td>
</tr>
<tr>
<td>MOPP</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Right eye</td>
<td>48.2 (14.4)</td>
<td>42.9 (13.2)</td>
<td>41.2 (12.1)</td>
<td>−5.4 (10.9)</td>
<td>−1.7 (8.0)</td>
<td>7.0 (12.6)</td>
</tr>
<tr>
<td>Left eye</td>
<td>49.5 (13.1)</td>
<td>44.6 (12.3)</td>
<td>42.7 (11.1)</td>
<td>−4.8 (11.3)</td>
<td>−2.0 (8.2)</td>
<td>−6.8 (13.2)</td>
</tr>
</tbody>
</table>

Abbreviations: DOPP, diastolic ocular perfusion pressure (OPP); IOP, intraocular pressure; MAP, mean arterial pressure; MOPP, mean OPP; SOPP, systolic OPP.

* Measurement time points included T1: seated, 15 minutes before starting hemodialysis; T2: seated, 2 hours after starting hemodialysis; and T3: seated, 15 minutes after ending hemodialysis.

Average IOP, MAP, and OPP all significantly changed during the HD session (Figure 1). From T1 to T3, IOP significantly increased by 3.1 mm Hg in both eyes (P < .001), MAP significantly decreased by 5.8 mm Hg (P = .05), and all categories of perfusion pressures were significantly lower than they were at baseline. These results are reported in Table 2.

Applying the thresholds used in the Barbados Eye Studies24,25 for evaluating relative risk of OAG in our data...
(Table 3), 26 of 49 right eyes (53%) and 22 of 48 left eyes (46%) had a SOPP that fell to 101 mm Hg or less. Similarly, 35 of 49 right eyes (71%) and 35 of 48 left eyes (73%) had a DOPP of 55 mm Hg or less, and 31 of 49 right eyes (63%) and 31 of 48 left eyes (65%) had a MOPP of 42 mm Hg or less. A higher percentage of eyes in the glaucoma group met these thresholds in all perfusion pressure categories compared with normal eyes (Table 3). Figure 2 illustrates the average OPP of both eyes at each measurement time during HD in glaucomatous and non-glaucomatous eyes. Looking at HD-related factors, we noted a significant decrease in mean (SD) plasma osmolarity of 15.9 (6.9) mOsm/L ($P < .001$) when comparing pre-HD with post-HD values. The rate of change in plasma osmolarity averaged −4.5 (1.8) mOsm/L/h. There was also a significant increase in colloid osmotic pressure of 2.8 (6.1) mm Hg ($P = .004$) across the HD sessions. However, there were no significant correlations

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Ocular Perfusion Pressure, No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Systolic ≤ 101 mm Hg</td>
</tr>
<tr>
<td>All eyes</td>
<td></td>
</tr>
<tr>
<td>Right (n = 49)</td>
<td>26 (53)</td>
</tr>
<tr>
<td>Left (n = 48)</td>
<td>22 (46)</td>
</tr>
<tr>
<td>Glaucomatous eyes</td>
<td></td>
</tr>
<tr>
<td>Right (n = 9)</td>
<td>6 (67)</td>
</tr>
<tr>
<td>Left (n = 9)</td>
<td>6 (67)</td>
</tr>
</tbody>
</table>

Figure 2. Average Ocular Pressure Readings During Hemodialysis in Glaucomatous and Nonglaucomatous Eyes

Average ocular perfusion pressure (OPP) (A), mean OPP (MOPP) (B), systolic OPP (SOPP) (C), and diastolic OPP (DOPP) (D) of both eyes at each point of measurement during hemodialysis in glaucomatous and nonglaucomatous eyes. All measurements were taken with the patient in the seated position 15 minutes before starting hemodialysis (T1), 2 hours after starting hemodialysis (T2), and 15 minutes after ending hemodialysis (T3). L indicates left eye; and R, right eye.
between changes in IOP or OPP and systemic HD factors (changes in plasma osmolarity, rate of change in plasma osmolarity, change in colloid osmotic pressure, dialysis rate, volume of ultrafiltrated fluid, and change in body weight).

There was a negative correlation between change in body weight and change in osmotic pressure \( (r = -0.34; P = 0.02) \), as well as a negative correlation between body weight change and ultrafiltrated fluid volume \( (r = -0.93; P < 0.001) \). A positive correlation was observed between the change in colloid osmotic pressure and the volume of ultrafiltrated fluid \( (r = 0.46; P = 0.001) \).

### Discussion

The effect of HD on OPP has not been well studied. In contrast, the effect of HD on IOP has been extensively studied, but results are conflicting and no concrete answer has emerged. Our study demonstrates a statistically significant increase in IOP during HD, with a concomitant decrease in all measures of perfusion pressure. The OPP reached levels that increase the risk of both glaucoma development and progression. Interestingly, more glaucoma and glaucoma-suspect eyes reached threatening levels than did normal eyes.

The earliest studies\(^3\)-\(^6\) examining the effect of HD on IOP predominantly reported a rise in IOP, which was explained as an effect secondary to a rapid decrease in plasma osmolarity and/or a relative increase in intracellular, compared with extracellular, urea concentration. The rapid change results in a gradient between plasma and ocular compartments, inducing a shift of extracellular fluid from the blood to the anterior chamber. Other studies\(^27\)-\(^29\) reported no correlation between serum osmolarity and IOP. More recent studies\(^29\)-\(^34\) demonstrated no significant change in IOP with HD or a decrease in IOP. Because rapidity of plasma osmolarity decreases during HD may also play a role, one theory suggests that the reversal in IOP change between earlier and later studies is attributable to newer HD techniques using a slower dialysis process, resulting in slower plasma osmolarity changes. Indeed, Sitprija et al\(^33\) demonstrated a rise in IOP when the plasma osmolarity rate of change was \(-11\) mOsm/L/h, but not when it was \(-8\) mOsm/L/h. Other groups found no significant change in IOP for plasma osmolarity rates of change of \(-6\),\(^28\)\(-4.1\),\(^29\) and \(-2.9\)\(^33\) mOsm/L/h.

Additionally, fluid removal during ultrafiltration without concomitant albumin removal increases colloid osmotic pressure, leading to a fluid shift from the aqueous humor to the plasma and a decrease in IOP.\(^29\) Tokuyama et al\(^29\) found a correlation between change in IOP and colloid osmotic pressure, but not plasma osmolarity. Doshiro et al\(^39\) also found that IOP changes were negatively correlated with colloid osmotic pressure changes. The theoretical relationships between these various factors affecting IOP during HD are illustrated in Figure 3.

**Figure 3. Flowchart**

Various factors affecting intraocular pressure (IOP) fluctuation during hemodialysis.

Despite multiple reports with conclusions varying from an increase to no change to a decrease in IOP during HD, there is a tendency for IOP to increase in patients with abnormal aqueous outflow, as shown in patients with narrow angles.\(^\text{8,26}\) Taiwar et al\(^7\) reported an IOP increase in eyes with compromised aqueous outflow. Doshiro et al\(^39\) reported a significant IOP decrease during HD in the general study group, but an IOP increase in patients with glaucoma. This IOP difference was not statistically significant, likely because of the small numbers of patients with glaucoma. Furthermore, several case reports\(^6\)-\(^12\) described acute IOP spikes after HD in patients with diagnoses varying from neovascular to exfoliative glaucoma. Similar to previous studies, our investigation found significant changes in IOP, with greater magnitude in patients with a diagnosis of glaucoma or suspected glaucoma when compared with IOP changes in patients without a history of glaucoma (Figure 2).

Recent studies showed no change or a decrease in IOP\(^27\)-\(^30\)\(^33\) and no change in OPP\(^33\) during HD. These observations contrast with our findings, which showed a significant IOP increase and significant OPP decrease during HD. Our HD technique did not differ from the one used in these other studies enough to explain the varying results. As discussed above, a decrease in plasma osmolarity may lead to an increase in IOP, and an increase in colloid osmotic pressure tends to lead to a decrease in IOP. Interestingly, our study showed both a significant decrease in plasma osmolarity and a significant increase in colloid osmotic pressure, neither of which was correlated with IOP changes.

Our study is unique in that, unlike other trials that recruited predominately white patients without glaucoma, most of our participants were African American. Given the higher prevalence of glaucoma among African Americans compared with other races, our patient population may be at high risk for underlying glaucomatous disease and for increased IOP with decreased OPP after HD even in patients reporting no history
of glaucoma. Possible racial differences in IOP and OPP changes during HD need to be confirmed in a larger study.

Autoregulation, the ability of a vascular bed to change vascular resistance in response to perfusion pressure changes to maintain a relatively constant blood flow,\(^{37}\) plays a crucial role in maintaining blood flow to the optic nerve. Patients with glaucoma may have autoregulation impairment, with abnormal responses to changes in OPP. For instance, when healthy participants moved from the sitting to the supine position, the central retinal artery resistive index increased, but no change occurred in patients with glaucoma.\(^{38}\) Furthermore, experimentally increasing IOP (via suction cup) in individuals with and without primary OAG (POAG) resulted in a more pronounced retinal vessel dilatation in healthy participants than in those with POAG, indicating a possible abnormal autoregulatory response in the POAG group.\(^{39}\)

Evidence is increasing that IOP and BP instability may be associated with glaucoma. Thus, OPP variations due to IOP and/or BP fluctuations may play a role in the disease process. This idea is supported by 2 observations. First, patients with otherwise well-controlled IOP but progressive POAG or normal-tension glaucoma had a significant reduction in systolic BP during the 24-hour period.\(^{40}\) Second, compared with healthy individuals, patients with POAG had not only significantly lower MOPP but also greater diurnal MOPP variations.\(^{41}\) Furthermore, other studies demonstrated that MOPP fluctuations during a 24-hour period might be a risk factor in the development and progression of normal-tension glaucoma.\(^{42,43}\) Combined, these studies show that “passive” OPP fluctuations are greater in patients with glaucoma than in healthy people. Our study showed fluctuations in OPP during the “active” process of HD, which, even in relatively healthy patients, is known to be accompanied by BP changes secondary to fluid shifts. Accordingly, the HD patients might have additional active IOP and/or OPP fluctuations during frequent sessions of HD, which may subsequently increase their risk for glaucoma development and progression.

In our study, the MOPP was consistently lower than the DOPP. This is because the calculation of MOPP includes an adjustment (arbitrarily defined in the literature as \(\frac{2}{3}\)) for the mean ophthalmic arterial pressure being lower than the mean brachial arterial pressure. Yet the calculated OPP, SOPP, and DOPP do not contain this correction. Thus, although there are inconsistencies in calculating the various perfusion pressures given the numerous conclusions from previous publications based on these methods of data analysis, we have chosen to define our results as such for better comparison with other studies.

Our results are particularly worrisome for our patient population, which undergoes long-term, frequent HD sessions, each lasting several hours. Survival rates of HD patients have been improving, with those aged 65 to 74 years experiencing a 23% improvement in survival between 1990-1994 and 1995-1999. Additionally, life expectancy between 1995 and 1999 of HD patients aged 65 to 69 years was 4.6 years after HD initiation.\(^{44}\) As the Barbados Eye Studies\(^{24,25}\) demonstrated, the increased risk for glaucoma development at 4 and 9 years in patients with low baseline OPP, and the longer survival time of HD patients potentially exposes them to recurrent optic nerve ischemic damage.

The present study has strengths and limitations. The primary strength of our study is that most of our participants were African American (82%), unlike in previous reports, and a significant proportion (18%) of HD patients had a diagnosis of glaucoma or suspected glaucoma. The first limitations of the present study are the small number of patients with glaucoma and that the glaucoma diagnosis was based on patient reports and/or reviews of available medical records. Nevertheless, our subgroup analysis showed that, compared with patients without glaucoma, patients with glaucoma had higher percentages of OPP variables reaching levels reported to increase the risk of glaucoma development and progression. Second, because IOP and OPP were measured in a single session, it is not known whether the observed elevated IOP and decreased OPP would be reproducible for each patient during other HD sessions. A future study with a larger sample size that includes African Americans and has repeated measurements during multiple HD sessions is needed to confirm our findings. Third, the role of such transient increases in IOP and decreases in OPP is unknown. Several patients with glaucoma likely experience a significant reduction of OPP every night owing to the decrease in systolic and diastolic BPs that occur normally during sleep. In the study conducted by Choi et al,\(^{47}\) 42% of patients with normal-tension glaucoma had a greater than 10% reduction in nocturnal BP. Thus, the changes in OPP found in our study, occurring during a period of 5 hours, 3 times a week, may be insignificant in terms of risk of glaucoma progression. A prospective study looking specifically at the risk of glaucoma progression after HD with evaluation of visual field changes is needed to fully address this.

In conclusion, it may be prudent to monitor IOP during HD sessions, perhaps lowering IOP or permitting higher BP as needed. This would be especially valuable for patients with glaucoma, who are at an increased risk for significant OPP decreases and, at baseline, are more vulnerable to ischemic insult because of a diminished optic nerve reserve. In clinical practice, clinicians should inquire about HD history in patients who have glaucoma progression despite tight IOP control. Ophthalmologists and nephrologists should collaborate to ensure sufficient BP and OPP during HD in high-risk patients, such as those with advanced glaucoma. Such patients may especially benefit from IOP and BP monitoring during HD sessions to avoid OPP changes from IOP spikes and/or suboptimal BP.
Hemodialysis Effect on Ocular Pressure

Analysis and interpretation of data: Hu, Kim, Vajaranant.
Drafting of the manuscript: Hu, Kim.
Critical revision of the manuscript for important intellectual content: Hu, Bui, Patel, Arruda, Wilensky, Vajaranant.
Statistical analysis: Hu, Patel, Kim.
Obtained funding: Hu, Vajaranant.
Administrative, technical, and material support: Hu, Bui, Wilensky.
Study supervision: Arruda, Wilensky, Vajaranant.
Conflict of Interest Disclosures: None reported.

Funding/Support: This study was funded by grant K2DH055892 (Dr Vajaranant) from the National Institute of Child Health and Human Development and the Office of Research on Women’s Health, the Komarek-Hyde-McQueen Foundation, and the Illinois Society for the Prevention of Blindness.

Role of the Sponsor: The funders had no role in the design and conduct of the study; collection, management, analysis, and interpretation of the data; preparation, review, or approval of the manuscript; and decision to submit the manuscript for publication.

Additional Contributions: We thank the American Glaucoma Society for the 2012 Bernard Schwartz Award.

REFERENCES