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The effect of hemodialysis on intraocular pressure and ocular perfusion pressure: an interdisciplinary review

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HIGHLIGHTS

Hemodialysis may destabilize intraocular pressure and ocular perfusion, supporting ophthalmic monitoring in high-risk patients.

ABSTRACT

Hemodialysis, as the most commonly used method of renal replacement therapy, may induce changes in intraocular pressure. A hemodialysis-associated increase in intraocular pressure has been reported primarily in patients with impaired aqueous humor outflow, including those with narrow or closed iridocorneal angles and those with primary or secondary glaucoma, especially neovascular or pseudoexfoliative glaucoma. One of the main mechanisms underlying intraocular pressure elevation is considered to be a non-synchronous decrease in blood and intraocular fluid osmolality, leading to a transient osmotic gradient and fluid shift into the eyeball. Hemodialysis may also affect ocular perfusion pressure, and its reduction may increase optic nerve susceptibility to damage even when intraocular pressure remains within normal limits. Fluctuations in both intraocular pressure and ocular perfusion pressure represent a clinically significant but under-recognized concern in patients at risk. This review highlights the importance of interdisciplinary collaboration between nephrologists and ophthalmologists, while existing knowledge gaps justify further research in this area.

Key words: intraocular pressure, ocular perfusion pressure, hemodialysis, end-stage renal disease, renal replacement therapy

INTRODUCTION

Chronic kidney disease is a growing clinical problem, and in its most severe stage, end-stage renal disease, hemodialysis (HD) remains one of the most widely used methods of renal replacement therapy.

The significance of this issue for ophthalmology stems from the fact that HD can affect both intraocular pressure (IOP) and ocular perfusion pressure (OPP) – parameters that are crucial for assessing the risk of glaucomatous optic nerve damage. Elevated IOP remains the primary modifiable risk factor for glaucoma; however, a decrease in OPP may increase optic nerve susceptibility to damage even when IOP is well controlled [1].

The first preliminary observations regarding changes in IOP during HD were presented by Sitprija and Holmes as early as 1962 [2], and 2 years later, Sitprija et al. published a classic paper on this topic [3]. Despite more than 6 decades of research, no clear consensus has been reached regarding the direction, magnitude, and predictors of IOP changes during HD. The literature has described increases, decreases, and no significant changes in IOP during or after HD [4, 5]. The particular clinical significance are cases of increased IOP in patients with impaired aqueous humor outflow, especially in eyes with a narrow or closed iridocorneal angle [4, 6]. Symptomatic episodes of increased IOP have also been described in patients with neovascular or pseudoexfoliative glaucoma [7, 8].

The clinical relevance of this issue is also supported by studies assessing ocular perfusion and glaucoma risk in dialysis patients. In their 2013 study, Hu et al. demonstrated that HD may affect not only IOP but also OPP, which justifies a more comprehensive assessment of optic nerve perfusion in dialysis patients [1]. Population-based studies, in turn, suggest a possible association between dialysis therapy and the risk of glaucoma, although they do not conclusively establish a causal mechanism [9]. Therefore, dialysis patients, especially those with diagnosed glaucoma or a predisposition to aqueous humor outflow disorders, require special attention from both ophthalmologists and nephrologists.

Recent studies from 2026 indicate that a single hemodialysis session can affect numerous parameters of the anterior and posterior segments of the eye [10], and a prospective study by Lee et al. demonstrated a transient increase in IOP during HD and a decrease in mean OPP [11].

The purpose of this review is to:

1. Summarize and critically review current data on the impact of HD on IOP and OPP.
2. Discuss the possible pathophysiological mechanisms underlying these changes.
3. Identify patient groups at particular risk for ophthalmic complications of HD.

MATERIALS AND METHODS

A narrative literature review was conducted using PubMed/MEDLINE and Google Scholar. The search covered publications from January 1962 to May 2026. The following Medical Subject Headings (MeSH) and free-text terms were used in various combinations: *hemodialysis, haemodialysis, dialysis, intraocular pressure, IOP, ocular perfusion pressure, OPP, glaucoma, ocular hypertension, aqueous humor outflow, ocular dialysis disequilibrium, anterior chamber angle, osmolality, end-stage renal disease, chronic kidney disease*. Corresponding free-text terms in Polish and Russian were also used where applicable. Articles published in English, Polish and Russian were taken into account. The reference lists of identified review articles were also searched manually to identify additional relevant publications. Case reports were included when they illustrated a specific pathophysiological mechanism or management strategy not adequately represented in larger studies.

RESULTS

Effect of hemodialysis on intraocular pressure

The available literature indicates 3 major patterns of IOP response to hemodialysis: an increase, a decrease, or no significant change. This heterogeneity reflects differences in study populations, dialysis techniques, and methods used to assess IOP [4].

Early studies more frequently reported an increase in IOP during HD, which was attributed, among other factors, to rapid changes in plasma osmolality and to the dialysis techniques used at that time, particularly HD with acetate-based dialysate. Sitprija et al. reported a mean IOP increase of approximately 5.9 mmHg during the first hours of dialysis [3]. Burn described an increase in IOP in a subset of patients undergoing long-term HD [12]. Ramsell et al., however, demonstrated that IOP changes during HD may follow a dynamic course and do not necessarily correspond to a simple, linear increase in pressure despite a decrease in plasma osmolality [13].

With development in dialysis techniques, this pattern has changed. Leiba et al. observed only minimal IOP changes during HD and a decrease in IOP during isolated ultrafiltration, a procedure involving removal of excess fluid without simultaneous diffusion of solutes, thereby helping maintain relatively stable plasma osmolality [14]. Subsequent studies demonstrated that a clinically relevant IOP increase occurs mainly in eyes with impaired aqueous humor outflow. Tawara et al. found a significant increase in IOP in eyes with reduced outflow facility, whereas no significant changes were observed in eyes with proper outflow [6].

In their 2018 study, Kilavuzoglu et al. demonstrated a significant decrease in IOP during contemporary HD: from 16.71

(±2.51) mmHg before the procedure to 15.52 (±3.18) mmHg at the end of HD and 15.23 (±2.73) mmHg 30 min after the procedure, with p = 0.001 in repeated-measures analysis [15]. The authors attributed this finding, among other mechanisms, to slower changes in osmolality and the effect of plasma colloid osmotic pressure. A decrease in IOP during HD was also reported by Tokuyama et al., Doshiro et al., Chelala et al., and Dinc et al., suggesting a possible role of changes in plasma colloid osmotic pressure, systemic parameters, and ocular morphometric changes [16–19]. Other analyses reported no significant change in IOP, further supporting the dependence of study findings on the examined population, dialysis technique, and measurement methodology [20, 21].

The meta-analysis by Chen et al., which included 53 studies, did not demonstrate a significant overall increase in IOP during HD [5]. However, subgroup analysis indicated that the direction of IOP change depended on the publication period and type of dialysate. In studies published before 1986, in which acetate-based dialysate was most commonly used, IOP increases were observed more frequently. Between 1986 and 2005, no significant changes were found, whereas after 2005, with the use of bicarbonate-based dialysate, a small but statistically significant decrease in IOP was reported. In meta-regression, dialysate type remained a significant moderator of IOP change, while glaucoma, a narrow anterior chamber angle, and impaired aqueous humor outflow were identified as factors predisposing to IOP elevation during HD [5].

Nowadays, the studies also highlight the importance of anatomic factors. Wang et al. demonstrated that an increase in IOP during HD occurs primarily in eyes with a narrow anterior chamber angle [22]. Sarr et al., in turn, reported a significant postdialysis increase in IOP in their study population, emphasizing the persistent variability of the clinical response [23].

The recent 2026 study by Roskal-Wałek et al., which included a comprehensive assessment of ophthalmic parameters after a single hemodialysis session, did not demonstrate a significant change in IOP but showed that HD affected multiple parameters of both the anterior and posterior segments of the eye [10]. These findings support the interpretation that the absence of a significant IOP change does not exclude other peridialytic morphometric and hemodynamic alterations within the eye.

The total available data indicate that the direction of IOP changes during hemodialysis is determined primarily by the status of the aqueous humor outflow system and by the nature of osmotic changes occurring during the procedure. This substantially limits the generalizability of findings across patient populations. Consequently, studies combining IOP measurement with assessment of anterior segment structure, systemic parameters, and ocular perfusion pressure are becoming increasingly important.

Ocular perfusion pressure during hemodialysis

In studies evaluating HD, OPP is most commonly calculated indirectly on the basis of mean arterial pressure (MAP) and IOP. Depending on the adopted methodology, different simplified formulas are used, including OPP = ⅔ × MAP - IOP or mean OPP = ⅔ × (MAP - IOP), which means that OPP values reported across individual studies are not always directly comparable. This parameter reflects the effective pressure gradient for ocular blood flow, accounting for the opposing effect of IOP; therefore, a decrease in OPP may be relevant to optic nerve head perfusion, particularly in patients with glaucoma or susceptibility to glaucomatous optic nerve damage [1].

During HD, MAP may decrease as a result of ultrafiltration and reduction in intravascular volume. If this is accompanied by an increase in IOP, the combined effect may lead to a significant decrease in OPP. In 2013, Hu et al., in a study including 49 patients and 97 eyes, demonstrated an increase in IOP with a concomitant decrease in MAP, resulting in a significant reduction in the 3 analyzed OPP parameters: systolic, diastolic, and mean OPP [1]. In some of the analyzed eyes, OPP values decreased to levels considered in the literature to be associated with an increased risk of glaucoma development or progression [1]. No significant correlation was found between changes in IOP or OPP and osmolality parameters, suggesting that osmotic changes alone do not fully explain the observed alterations [1].

Sarr et al. reported in 2024 an increase in IOP after an HD session without significant changes in OPP [23], emphasizing that the effect of HD on ocular parameters is heterogeneous and depends on the balance between changes in IOP, arterial pressure, and compensatory mechanisms.

In the 2025 clinical study by Hendricks et al., patients with glaucoma exhibited higher IOP variability and larger decreases in OPP during HD than individuals without glaucoma; these changes were often asymptomatic [24]. Described findings indicate that the risk of transient deterioration in optic nerve perfusion conditions in patients with glaucoma may be underestimated if ophthalmic assessment is limited to measurements performed outside dialysis sessions.

In a recent prospective study published in 2026, Lee et al. showed that an increase in IOP during HD was accompanied by a decrease in mean OPP, further supporting the assessment of pressure-related parameters in conjunction with perfusion-related parameters in patients undergoing dialysis [11].

The total available data indicate that HD may destabilize not only IOP but also OPP, creating a complex hemodynamic environment that may potentially promote optic nerve damage in high-risk patients. This justifies consideration of ophthalmic monitoring in selected hemodialysis patients, especially those with glaucoma, progression of

neuropathy despite controlled IOP, or other risk factors for impaired ocular perfusion.

Pathophysiological mechanisms and ocular dialysis disequilibrium

The term ocular dialysis disequilibrium describes a transient, sometimes symptomatic increase in IOP during HD, associated with rapid and asynchronous changes in the osmolality of blood and intraocular fluids. This concept has been discussed more extensively in recent reports, including by Maja et al. in 2022 [7].

Rapid removal of urea and other osmotically active solutes from plasma during HD may lead to a decrease in plasma osmolality. Because of the limited permeability of the blood–aqueous barrier, equilibration of solute concentrations between plasma and aqueous humor may be delayed, promoting the formation of a transient osmotic gradient. As a result, fluid may shift toward the anterior chamber, increasing aqueous humor volume and potentially raising IOP, particularly when aqueous humor outflow is impaired [4, 6].

A similar mechanism has also been considered in relation to the vitreous humor, where elimination of urea and other osmotically active solutes may occur more slowly than in plasma. Persistence of an osmolality difference between plasma and the vitreous humor could promote the formation of an osmotic gradient and secondary IOP fluctuations. However, it should be emphasized that the significance of this mechanism remains less well documented and is based mainly on clinical observations and pathophysiological interpretations [4, 26].

The key factor appears to be not only the absolute change in osmolality but also the rate of its decline. In the classic observations by Sitprija et al., a faster decrease in plasma osmolality was associated with a larger increase in IOP, whereas slower osmotic changes resulted in less pronounced IOP fluctuations [3, 4]. This may partly explain why more recent studies conducted under more controlled HD conditions have more often reported no significant change or a decrease in IOP rather than a marked pressure increase [5, 15].

Mechanistic interpretations have also emphasized the importance of postdialysis urea rebound, which results from the movement of urea from tissues back into plasma after the completion of dialysis. This phenomenon, together with the degree of hemoconcentration related to ultrafiltration, may modify both the direction and magnitude of intradialytic IOP changes [27].

In addition, mechanisms analogous to cerebral dialysis disequilibrium syndrome have been considered, including the generation of so-called idiogenic osmoles and changes in intraocular pH. These hypotheses could explain the persistence of relative hyperosmolality of intraocular fluids, particularly aqueous humor, despite changes in plasma parameters. However, their role in the pathophysiology of IOP changes during HD remains unclear [4].

TABLE 1

Intraocular pressure and ocular perfusion pressure changes during hemodialysis in selected studies. Authors' own elaboration based on selected studies. The table presents selected representative studies and does not include all available reports.

Author (year)	Population	Change in IOP/OPP during HD	Factor differentiating the IOP/OPP response	Relevance for interpretation
Sitprija et al. (1964) [3]	uremic patients undergoing HD	increase IOP	rapid decrease in plasma osmolality	classical osmotic gradient hypothesis
De Marchi et al. (1989) [25]	55 patients	increase IOP in eyes with a narrow angle; no significant change/decrease in the remaining eyes	anterior chamber angle width / aqueous humor outflow	identification of a high-risk group
Leiba et al. (1990) [14]	30 patients; 58 eyes	minimal, nonsignificant increase IOP after HD; significant decrease IOP during isolated UF	change in osmolality during HD; stable osmolality during ultrafiltration	differentiation between the effects of osmolality changes and ultrafiltration on IOP
Hu et al. (2013) [1]	49 patients; 97 eyes	increase IOP; decrease OPP	decrease in MAP and reduction in OPP	importance of simultaneous assessment of IOP and OPP during HD
Kilavuzoglu et al. (2018) [15]	106 patients	decrease IOP	slower changes in osmolality / colloid osmotic pressure	confirmation that IOP may decrease during contemporary HD
Wang et al. (2022) [22]	52 patients	increase IOP in eyes with an extremely narrow or closed angle	anterior chamber angle configuration/anterior segment anatomy	confirmation of the importance of anterior segment anatomy
Sarr et al. (2024) [23]	29 patients; 58 eyes	increase IOP; no significant change OPP	increase in IOP without significant change in OPP	ambiguous relationship between IOP and OPP changes during HD
Lee et al. (2026) [11]	56 patients; 103 eyes	increase IOP; decrease mean OPP	baseline serum osmolality; phakic vs pseudophakic eyes	multifactorial determinants of IOP elevation and OPP changes during HD

IOP – intraocular pressure; HD – hemodialysis; MAP – mean arterial pressure; OPP – ocular perfusion pressure.

When aqueous humor outflow is within the norm, the mechanisms described above may not lead to a clinically significant increase in IOP, because excess fluid may be compensated for by efficient outflow. Conversely, when outflow impairment coexists – such as a narrow or closed anterior chamber angle, primary glaucoma, anterior synechiae, neovascular glaucoma, or other forms of secondary glaucoma – this compensatory mechanism may be insufficient. Observations by Tawara et al., based on assessment of aqueous humor outflow facility, indicate that significant IOP increases during HD occur primarily in eyes with impaired aqueous humor hydrodynamics [6].

Patient groups particularly vulnerable to ocular complications of hemodialysis

Patients with primary glaucoma

Patients with primary glaucoma – including primary open-angle glaucoma and primary angle-closure glaucoma – constitute a group requiring particular attention during HD. Impaired aqueous humor outflow limits the ability to compensate for transient osmotic and hemodynamic changes, which may be of minor importance in individuals without ocular pathology but may promote significant increases in IOP in patients with glaucoma [4]. At the same time, coexisting glaucomatous neuropathy increases the clinical relevance of reductions in OPP, because decreased ocular perfusion may further increase the risk of optic nerve damage [1].

In the 2025 clinical study by Hendricks et al., patients with glaucoma exhibited higher IOP variability and larger decreases in OPP during HD than individuals without glaucoma [24]. These findings suggest that measurements performed only outside HD sessions may not reflect the actual pressure-related and perfusion-related burden on the eye in patients with glaucoma.

Patients with secondary glaucoma (pseudoexfoliative and neovascular glaucoma)

Pseudoexfoliation syndrome leads to the deposition of fibrillar material within the trabecular meshwork of the anterior chamber angle, which may increase resistance to aqueous humor outflow. Consequently, pseudoexfoliative glaucoma may increase susceptibility to IOP elevation during HD. This is illustrated by the case report presented by Herman et al. in 2025, describing a patient with advanced pseudoexfoliative glaucoma who developed marked, recurrent IOP elevations during HD that required surgical intervention [8].

Neovascular glaucoma, which may develop in the course of proliferative diabetic retinopathy or central retinal vein occlusion, is associated with pathologic neovascularization of the anterior chamber angle and its secondary closure. In this group of patients, rapid osmotic and hemodynamic changes during HD may reveal limited aqueous humor outflow reserve, leading to substantial IOP elevations and ocular pain. Kozina et al. emphasized that neovascularization of the ocular drainage structures and glaucoma are important, although not undisputed, risk factors for painful dialysis-related ocular hypertension [26].

Patients with narrow and closed anterior chamber angle

An anatomically narrow or closed iridocorneal angle is a well-documented risk factor for significant IOP elevation during HD. Osmotically induced fluid movement into the eye may increase the load on the aqueous humor outflow system and, in eyes with limited anatomic reserve, lead to a clinically significant increase in IOP [4].

De Marchi et al. demonstrated that excessive IOP elevation occurred mainly in patients with a narrow anterior chamber angle, whereas in most patients with normal angle anatomy, IOP remained stable or decreased [25]. More recent anterior segment imaging studies indicate that HD may be associated with transient narrowing of angle parameters, and that baseline angle anatomy may modify both the direction and magnitude of IOP changes during the procedure [22, 28].

Patients with diabetes mellitus and its ocular complications

Diabetes mellitus may increase the risk of ophthalmic complications during HD primarily indirectly – through proliferative diabetic retinopathy, neovascularization of the anterior chamber angle, and the development of neovascular glaucoma. However, diabetes itself should not be regarded as an unequivocal predictor of IOP elevation during HD. Study findings remain heterogeneous: some authors have suggested an association between baseline glycemia or diabetes and the magnitude of IOP changes, whereas other studies have not confirmed a significant effect of diabetes or diabetic retinopathy on intradialytic IOP fluctuations [26].

Patients after ophthalmic surgery

Patients after glaucoma surgery or anterior segment surgery may represent a potential risk group, because anatomic changes within the anterior chamber angle and aqueous humor outflow pathways may modify the IOP response to HD. The literature has particularly highlighted patients after recent ocular surgery, in whom disturbances in aqueous humor dynamics during HD may have greater clinical relevance. However, the available data are limited, and the effect of HD on IOP in this group remains difficult to predict [4].

Patients undergoing repeated hemodialysis sessions

In patients undergoing regular HD sessions, not only isolated episodes of IOP elevation but also repeated diurnal fluctuations in this parameter may be clinically relevant.

Panagiotou et al. showed that on the day of HD, peak IOP values and the amplitude of diurnal IOP fluctuations were higher than on a nondialysis day [29]. It means that single IOP measurements performed outside HD sessions may not reflect the actual pressure variability in dialysis patients. However, these data do not prove that regular HD itself leads to progression of glaucomatous neuropathy; the long-term significance of such fluctuations requires further investigation [4].

CONCLUSIONS

HD affects IOP and OPP through complex osmotic, hydrodynamic, and hemodynamic mechanisms. The direction and magnitude of these changes are heterogeneous; however, aqueous humor outflow status, anterior chamber angle anatomy, the rate of osmolality changes, hemodynamic parameters, and the characteristics of the dialysis procedure appear to be of key importance [4, 5].

Patients with glaucoma, narrow or closed iridocorneal angles, ocular complications of diabetes, and those undergoing repeated hemodialysis sessions require particular attention. In these groups, fluctuations in IOP and OPP may create unfavorable conditions for the optic nerve and may contribute to the progression of glaucomatous neuropathy, particularly when they remain undetected during routine ophthalmic follow-up [1, 4, 26].

Routine measurements performed outside HD sessions may not reflect the actual variability of IOP and OPP in dialysis patients. The pain in the eye, headache, or transient visual disturbances during HD should prompt exclusion of symptomatic IOP elevation [7, 30]. Therefore, targeted ophthalmic monitoring is justified in at-risk patients, especially in cases of glaucoma, progression of neuropathy despite controlled IOP, or ocular symptoms occurring during HD [24, 29, 30].

Consideration of OPP in ophthalmic risk assessment and individualization of HD parameters should form part of the care of at-risk patients. Management should include consideration of dialysis parameter modification, ophthalmic treatment, and urgent ophthalmic consultation in cases of symptomatic IOP elevation during HD [4, 15, 26].

The complexity of these phenomena requires close collaboration between ophthalmologists and nephrologists. Further prospective long-term studies are needed to assess the impact of repeated peridialytic fluctuations in IOP and OPP on optic nerve structure and function, particularly in patients with glaucoma and risk factors for impaired ocular perfusion [4, 24]. It also remains necessary to identify the highest-risk groups, standardize ophthalmic monitoring protocols during HD, and determine which dialysis parameters and ophthalmic-nephrological interventions may reduce the adverse ophthalmic consequences of renal replacement therapy [4, 5, 15].

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