Intraocular Pressure Changes During Hemodialysis and the Role of Blood Glucose

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Ocular problems have been reported to exist in patients with end-stage renal disease (ESRD). The most common ocular complaint is red eye which may be associated with an increase in calcium-phosphate product. Other ocular conditions such as macular edema, ischemic optic neuropathy, retinal detachment, retinal hemorrhage, and elevated intraocular pressure have also been reported. The uremic milieu and its treatment with hemodialysis may affect ocular diseases.[1] Changes in intraocular pressure (IOP) are an eye problem among hemodialysis patients, about which there is controversy in the literature. Most studies have shown an increase or no changes in IOP during hemodialysis,[2-5] but some describe a decrease in IOP during hemodialysis.[6,7]

The question is what the relationship between IOP and dialysis is and what mechanisms are involved. The aim of hemodialysis is to correct the composition and volume of body fluids. The body fluid composition is corrected by elimination of uremic toxins and changes in plasma osmolality during dialysis. Some studies suggest that a decrease in serum osmolality during hemodialysis leads to imbalance in osmolality between the ocular chamber and blood; therefore, there is an influx of volume into the posterior chamber, and IOP increases after dialysis. In animal studies, a decline of 11 mOsm/kg H_2O/h in plasma osmolality is associated with an increase in IOP; however, this does not happen with lower decline in plasma osmolality (8.5 mOsm/kg H_2O/h). This suggests that a gradual change in serum osmolality does not affect IOP.[8] However, osmotic gradient may be a possible factor influencing IOP changes in some patients.

Another mechanism involves plasma colloid osmotic pressure. In some patients, serum osmolality is stable during dialysis and an increase in plasma colloid osmotic pressure due to increase in ultrafiltration leads to a decrease in IOP.[9] Recent studies have evaluated the relationship between central corneal thickness and IOP in dialysis patients. Using the Goldmann tonometery for measuring corneal thickness, Ozdek and colleagues showed a decline in IOP after dialysis with a decrease in corneal thickness, but there was no correlation between them.[10] Thus, changes in corneal thickness associated with IOP in hemodialysis patients is unclear.[10] The Singapore Malay Eye Study suggests that lower estimated glomerular filtration rate is associated with higher levels of IOP after adjusting for diabetes mellitus and glaucoma. It is due to trabecular meshwork impairment, and its mechanism is an increased oxidative stress which may cause cellular damage and dysfunction.[11] The epithelium of the ciliary body is responsible for aqueous humor formation and it transports ions and nutrients from circulation to posterior chamber. The osmotic gradient from this solute leads to water drag. In addition, pressure gradient between the ciliary body arterioles and the posterior chamber is resulted from ultrafiltration of interstitial fluid.[12] Thus, there are two opposite forces during hemodialysis that alter IOP: filtration and solute clearance.

Ultrafiltration leads to a gradual reduction of extracellular fluid and this reduction is intensified by fluid removal in patients with intradialytic weight gain. Oncotic pressure increases due to ultrafiltration because large proteins such as albumin and globulin cannot cross the membranes, so there will be a colloid gradient between plasma and interstitial fluid and water moves from the interstitial and aqueous humor to plasma fluid. The rate of increase in colloid pressure is slow until the rate of water movement from plasma by hemodialysis corrects the fluid accumulation

in plasma. These changes in oncotic pressure and ultrafiltration lead to decline in IOP at the end of dialysis. The change in IOP is related to the degree of ultrafiltration.[7]

Another theory is increasing IOP during dialysis.[13-15] During dialysis, uremic toxins and solutes are removed from vascular content, and this can lead to a lower serum osmolality compared to ocular osmolality. This osmotic gradient leads to water movement into the aqueous humor and increase in IOP. A high prevalence rate of glaucoma is shown in ESRD patients.[16] Tovbin and colleagues reported that increasing IOP correlated with rapid solute removal and high postdialysis urea rebound in dialysis patients, and they supported the role of solute clearance in the pathogenesis of IOP.[2]

Elevated IOP at baseline and its increase during dialysis lead to symptomatic intraocular hypertension, headache, orbital pain, or visual changes. Chronic elevation in IOP leads to optic nerve damage, glaucoma, and blindness. Normal IOP ranges from 10 mm HG to 21 mm Hg. An IOP of 20 mm Hg to 30 mm Hg usually results in chronic optic nerve damage and an IOP of 40 mm Hg to 50 mm Hg leads to rapid visual loss and retinovascular occlusion. Therefore, we should modify dialytic therapy for decreasing the risk of ocular complications. Using β-blockers, α2-adrenergic agonists, and acetazolamide is beneficial for decreasing the production of aqueous humor. Using prostaglandin agonists and mitotic agents leads to increased filtration and drainage of aqueous humor. In addition to medical therapy, surgical trabeculectomy or argon laser trabeculoplasty can be used. Another approach is modification of dialytic prescription, such as increasing the time of dialysis to reduce concentration gradients between blood and ocular chambers. Increasing ultrafiltration also has beneficial effects. Finally, we can use hypertonic solutions such as manitol or 3% saline to enhance serum osmolality and reduce IOP during dialysis.[2,17] In addition, the use of intensive hemodialysis modality such as nocturnal home hemodialysis can improve IOP in open-angle glaucoma. This improvement is related to three factors in dialysis: (1) enhancement of uremic clearance, (2) enhanced volume control, and (3) improvement in peripheral resistance.[18,19]

In the current issue of the *Iranian Journal of Kidney Diseases*, Afshar and coworkers report that IOP decreases during the first and second hours of hemodialysis, while blood sugar level is increased.[20] They report that there is a correlation between blood glucose changes and decrease in IOP, because high blood glucose leads to higher blood osmolality and enhances fluid movement from eyes to plasma. This study suggests that osmotic gradient is a dominant mechanism for IOP in conventional hemodialysis, and there is inverse correlation between plasma osmolality and IOP.

CONFLICT OF INTEREST

None declared.

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Primary Fibrosarcoma of Kidney

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Pathological features of renal masses can be classified as malignat, such as renal cell carcinoma (RCC), urothelial-based, sarcoma, and embryonic or pediatric tumor; benign, such as simple cyst, angiomyolipoma, oncocytoms, and renal adenoma; and inflamatorty, such as abscess, focal pyelonephritis, infected cyst, and tuberculosis.[1] Among malignant urologic tumors, RCC was the 3rd most prevalent after prostate carcinoma and transitional cell carcinoma (TCC) of the bladder.[2] Malignant tumors of the kidney in adults may arise from the renal parenchyma and renal palvis. Often, renal pelvic cancers are TCC type, comprising less than 10% of the microscopically confirmed kidney carcinomas. Renal cell carcinoma arise from renal parenchyma accounting for over 90% of kidney carcinomas.[3]

Sarcomas of the kidney are uncommon, 1% to 3%, as compared to other renal malignant tumors.[4,5] Histopathologically, renal sarcomas are malignant mesenchymal tumors of the kidney with a variety of histological types, among which fibosarcoma is the most frequent type. Other rarer sarcomas may occur and include leiomyosarcoma, rhabdomyosarcoma, osteogenic sarcoma, and liposarcoma. Fibrosarcoma of the kidney usually develops from the capsule of the kidney, which contains much fibrous and connective tissue with smooth muscle, and thus, it is thought to be the origin of mesenchymal tumors of the kidney.[5]

Microscopically, fibrosarcoma is composed of elongated spindel cells with delicate cytoplasm and long tapered nuclei. Cells were in broad fascicles, which focally produced herring bone pattern. Nuclear pleomorphism is not a feature, althogh mitoses are frequent, and necrosis is present focally. Immunohistochemical staining studies usually show positive staining for vimentin in tumor cells and negative for actin and S-100 stains.[5]

Fibrosarcoma of the kidney clinically occurs mainly in adults, and both genders are equally affected. The tumors are usually encapsulated and grow rapidly. About 40% of tumors are found to have renal vein invasion upon operation.[5]

Retroperitoneal sarcoma arises primarily from soft tissue of fibrous and adipose origin as well as muscle, nerve, and lymphatic tissue. These tissues are derived from primitive mesenchyme from the mesoderm with the same contribution from neuroectoderm. In classic reviews of these lesions, the tissue distribution in descending order is liposarcoma, leiomyosarcoma, and fibrosarcoma,