Can Toxic Anterior Segment Syndrome Cause a Severe Intraocular Pressure Elevation in a Hemodialysis Process?

Hemodiyaliz Sırasında Ciddi Göz İçi Basınç Yüksekliğinin Sebebi Geçirilmiş Toksik Anterior Segment Sendromu Olabilir mi?*

Ahmet ELBAY[1], Işıl KUTLUTÜRK[2], Alper DİNÇYILDIZ[1], Süleyman KUĞU[3], Ahmet ERGİN[4]

ABSTRACT

A 59-year-old diabetic male patient was admitted to our clinic with ocular pain and lacrimation in his right eye during a hemodialysis (HD) session. In the patient history he was treated for toxic anterior segment syndrome (TASS) occurring after phacoemulsification+intraocular lens implantation+pars plana vitrectomy (PHACO+IOL+PPV with no intraocular tamponades) for vitreous hemorrhage and epiretinal membranes. The diagnosis was acute angle closure glaucoma with pupil block. After receiving intravenous 20% mannitol and topical treatment, two peripheral laser iridotomies (LI) were performed on the right eye superiorly. In later weeks, it was seen that the LI sites were occluded and intraocular pressure (IOP) increases were seen. Surgical peripheral iridectomy was performed and the IOP stabilized. We thought that as a result of severe inflammation and destruction in the trabecular meshwork, the potential cause of the IOP elevation was fluid alteration in the vitreous cavity during a HD session and inadequate trabecular outflow response to this fluid excess. Secondary to inadequate outflow this fluid excess pushes the iris lens diaphragm anteriorly and this causes angle closure glaucoma with pupil block. Thus clinicians should be aware of this IOP elevation mechanism and surgical peripheral iridectomy can solve this IOP elevation mechanism.

Key Words: Angle closure, pressure, hemodialysis, toxic.

ÖZ

Ellidokuz yaşında diyabetik erkek hasta kliniğimize sağ gözünde hemodiyaliz (HD) sırasında gelişen ağrı ve sulanma şikayeti ile başvurdu. Hastanın anamnezinde, daha önce vitre içi hemoraji ve epiretinal membran nedeni ile fakoemülsifikasyon + göz içi lens implantasyonu + pars plana vitrektomi operasyonu ve sonrasında toksik anterior segment sendromu (TASS) nedeni ile tedavi öyküsü mevcuttu. Yapılan muayenede hastaya pupil bloklu açı kapanması glokomu tanısı konuldu. İntravenöz % 20 lik mannitol ve topikal glokom tedavisi sonrası sağ göze üst yerleşimli iki adet periferik lazer iridotomi (Lİ) açıldı. Takip eden haftalarda Lİ alanlarının kapanması ve HD sırasında tekrar eden göz içi basınç (GİB) artışlarının görülmesi üzerine cerrahi olarak periferik iridektomi uygulandı ve GİB'in HD sırasında stabil bir şekilde seyrettiği görüldü. TASS sırasında ortaya çıkan şiddetli inflamasyonun trabeküler ağda harabiyete yol açarak trabeküler dışa akımı bozduğu ve HD sırasında vitreus kavitesi içinde artan sıvı miktarına trabeküler dışa akım cevabını azalttığı düşünülmektedir. Azalan trabeküler dışa akım cevabına ikincil olarak artan vitreus içi sıvının iris lens diyaframını arkadan öne iterek pupil bloklu açı kapanması glokomuna yol açtığı sonucuna varılmaktadır. Bu nedenle klinisyenler TASS öyküsü olan HD hastalarında böyle bir GİB artış mekanizmasını göz önünde bulundurmalı ve cerrahi periferik iridektominin bu başınç artışını engellemede çözüm yolu olabileceğinin farkında olmalıdırlar.

Anahtar Kelimeler: Açı kapanması, basınç, hemodiyaliz, toksik.

Bu çalışma TOD 48 Ulusal Oftalmoloji Kongresi'nde sunulmuştur.

1- M.D. Pendik State Hospital, Eye Clinic, Istanbul/TURKEY
ELBAY A., draelbay@yahoo.com
DINCYILDIZ A., alperdincyildiz@gmail.com
2- M.D. Umraniye Training and Research Hospital Eye Clinic, Istanbul/TURKEY
KUTLUTURK I., slkutluturk@gmail.com
3- M.D. Dr Lutfi Kirdar Kartal Training and Research Hospital Eye Clinic, Istanbul/TURKEY
KUGU S., skugu@yahoo.com
4- M.D. Professor, Umraniye Training and Research Hospital Eye Clinic, Istanbul/TURKEY
ERGIN A., aergin60@yahoo.co.uk

Geliş Tarihi - Received: 16.09.2015
Kabul Tarihi - Accepted: 11.02.2016
Glo-Kat 2016;11:203-205

Yazışma Adresi / Correspondence Adress:
M.D. Ahmet ELBAY
Pendik State Hospital, Eye Clinic, Istanbul/TURKEY

Phone: +90 216 373 60 93
E-mail: draelbay@yahoo.com

INTRODUCTION

Using hemodialysis (HD) in patients with end-stage renal failure has undesirable non-ocular side effects. Besides these side effects, some ocular complications may arise such as intraocular pressure (IOP) changes, corneal and retinal diseases.[1]

In this case report we investigate an IOP elevation and poorly controlled IOP spikes during the HD process in a patient. The patient had experienced toxic anterior segment syndrome (TASS) in his right eye.

To our knowledge, this is the first case who had TASS previously and had an acute angle closure glaucoma during HD and treated with a surgical iridectomy procedure.

CASE REPORT

A 59-year-old end-stage diabetic nephropathic male patient was admitted to our clinic with ocular pain and lacrimation in his right eye during HD session.

He had been operated for vitreous hemorrhage and epiretinal membranes secondary to proliferative diabetic retinopathy two weeks ago. TASS had diagnosed in the right eye based on the existence of the findings including severe intraocular inflammation with fibrin reaction and hypopyon formation accompanied by mild corneal edema without pain on the first postoperative day. The patient was treated with subconjonctival dexamethasone (2 mg/0.5 ml) injenction, topical prednisolone acetate (1%) hourly, cyclopentolate hydrochloride (1%) three times daily and ofloxacin (0.3%) four times daily. The symptoms had improved substantially within ten days. He had been operated for vitreous hemorrhage and epiretinal membranes secondary to proliferative diabetic retinopathy two weeks ago. TASS had diagnosed in the right eye based on the existence of the findings including severe intraocular inflammation with fibrin reaction and hypopyon formation accompanied by mild corneal edema without pain on the first postoperative day. The patient was treated with subconjonctival dexamethasone (2 mg/0.5 ml) injenction, topical prednisolone acetate (1%) hourly, cyclopentolate hydrochloride (1%) three times daily and ofloxacin (0.3%) four times daily. The symptoms had improved substantially within ten days.

He had received surgery for vitreous hemorrhage and epiretinal membranes secondary to proliferative diabetic retinopathy two weeks before. TASS had been diagnosed in the right eye based on the existence of the findings including severe intraocular inflammation with fibrin reaction and hypopyon formation accompanied by mild corneal edema without pain on the first postoperative day. The patient had been treated with subconjonctival dexamethasone (2 mg/0.5 ml) injection, topical prednisolone acetate (1%) hourly, cyclopentolate hydrochloride (1%) three times daily and ofloxacin (0.3%) four times daily. The symptoms had improved substantially within ten days.

During the slit lamp examination, corneal edema, shallow anterior chamber, mid-dilated iris, centralized IOL with iris touch but no posterior synechia and elevated IOP (averaged~50 mmHg with Goldmann application tonometry) were observed in the right eye and senile nuclear cataract, normal iris and normal gonioscopic examination was observed in the left eye. The fundus examinations indicated dispersed hemorrhages and hard exudates, venous beadings and panretinal photocoagulation scars in the right and left eye. In the light of these findings the diagnosis of acute angle closure glaucoma with pupil block was made. After receiving intravenous 20% mannitol (2.5 -10 ml/kg) and topical beta blocker (Timolol, 0.5%) corneal edema was cleared and peripheral lazer iridotomies (LI) were performed on the right eye superiorly. Gonioscopy showed the presence of many anterior synechiae at various quadrants of angle (Figure 1). In later weeks, it was seen that LI sites were occluded and IOP spikes recurred during HD. Finally surgical peripheral iridectomy was performed and the IOP stabilized in the low-teens (range, 12-14 mmHg) without any antiglaucomatous therapy and no painful IOP spikes occurred during HD over the first postoperative year but the pupil remained mid-dilated (Figure 2).

DISCUSSION

Previously, different studies and case reports have suggested IOP changes related to HD.[2] Although many theories have been suggested to explain the mechanism of this event, the main cause has not been determined yet. But it is emphasized that the most possible mechanism causing the rise in IOP may be a decrease in serum osmolarity resulting in a gradient between plasma and ocular compartments. Subsequently this leads to fluid exchange from the extracellular area to the vitreous cavity and anterior chamber and increases the aqueous production.[2,3]

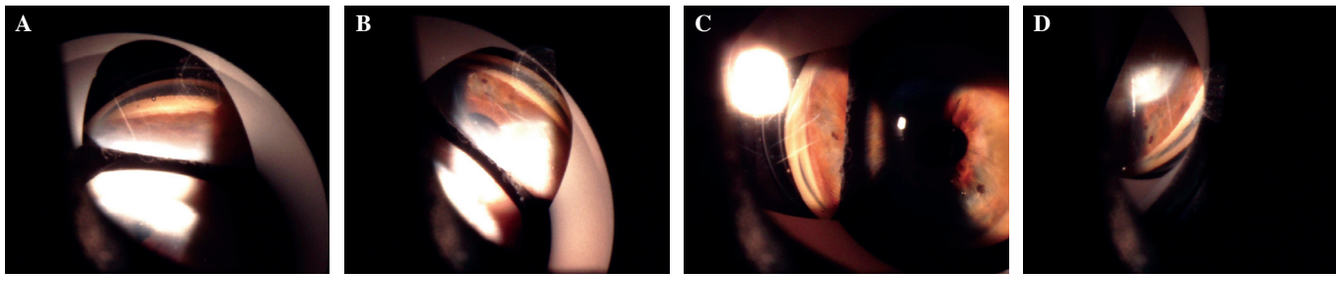

Figure 1a-d: *Gonioscopy demonstrates numerous anterior synechiae at a: inferior, b: inferotemporal, c: superotemporal, d: nasal quadrants of the angle of the right eye.*

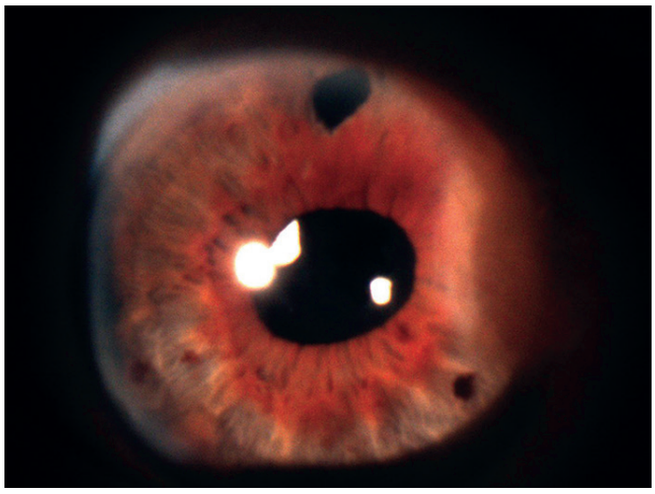

Figure 2: *Final anterior segment image of the right eye.*

In addition, as a result of this fluid exchange Burn et al. demonstrated that if the ocular outflow is healthy, there is a slight elevation of IOP or none but on the other hand, IOP in the eyes with outflow disturbance may rise during HD.[3]

Some cases with IOP elevation caused by outflow obstruction during HD were reported previously. Song et al. attributed the IOP elevation in neovascular glaucoma during HD to the occlusion of the angle with neovascularization and obstruction of the trabecular meshwork with red blood cells.[4] Masuda et al.,[5] suggested a pseudoexfoliative case with IOP elevation during HD as a result of accumulation of pseudoexfoliative material in the trabecular meshwork. Lim et al.,[6] demonstrated that uveitic patients may have IOP elevation during HD due to the increase in aqueous viscosity caused by inflammatory products and various cytokines, swelling or dysfunction of the trabecular meshwork, remodeling of the extracellular matrix, the obliteration of outflow channels, or synechial closure.

In our case, the patient had TASS treated with intense steroid therapy after PHACO+IOL+PPV operation and two weeks later he had IOP alteration during HD. We diagnosed angle closure with pupil block and we thought that the potential cause of the IOP elevation was fluid alteration in conjunction with fluid excess in the vitreus cavity and inadequate trabecular outflow response to this fluid excess. As a result of the elevated vitreus cavity pressure and inadequate outflow, anterior movement of the IOL and IOL-iris touch occurs. This causes pupil blocking and angle closure glaucoma. Outflow dysfunction was attributed to trabecular meshwork dysfunction, remodeling of the extracellular tissue, obliteration of outflow channels and deficient outflow resulting from inflammation due to TASS.

Presently to prevent IOP elevation during a session of HD, different treatment methods are being used for medical treatment. Topical and systemic carbonic anhydrase inhibitors, oral or intravenous hyperosmotic agents are usually preferred. However, medical therapy may cause electrolyte imbalance and metabolic acidosis, therefore surgical treatments are practiced, such as argon lazer trabeculoplasty, trabeculectomy with anti-metabolites and Ahmed valve implantation.[4-6] In our case, we preferred peripheral lazer iridotomy as our first choice because this was a closed angle IOP elevation with pupil block. But IOP elevations are not prevented during HD because of oclussion of the LI sites. Therefore we performed a large surgical iridectomy and as a result new IOP spikes were not seen during HD over the first postoperative year.

In conclusion, TASS may cause pupil block closed angle IOP elevation with a different mechanism during HD sessions as a result of severe inflammation in the trabecular mesh work. Clinicians should be aware of this IOP elevation mechanism and should bear in mind peripheral iridectomy as a surgical prevention method.

REFERENCES/KAYNAKLAR

1. Evans RD, Rosner M. Ocular abnormalities associated with advanced kidney disease and hemodialysis.Semin Dial. 2005;18:252-7.
2. Levy J, Tovbin D, Lifshitz T, et al. Intraocular pressure during haemodialysis: a review. Eye (Lond). 2005;19:1249-56.
3. Burn RA. Intraocular pressure during haemodialysis. Br J Ophthalmol. 1973;57:511-3.
4. Song WK, Ha SJ, Yeom HY, et al. Recurrent intraocular pressure elevation during hemodialysis in a patient with neovascular glaucoma. Korean J Ophthalmol. 2006;20:109-12.
5. Masuda H, Shibuya Y, Ohira A. Markedly increased unilateral intraocular pressure during hemodialysis in a patient with ipsilateral exfoliative glaucoma. Am J Ophthalmol 2000;129:534-6.
6. Lim SH, Son J, Cha SC. Recurrent symptomatic intraocular pressure spikes during hemodialysis in a patient with unilateral anterior uveitis. BMC Ophthalmol. 2013;13:3.