Eye (2017) 31, 1249–1252

www.nature.com/eye

Sir,
Immediate IOP elevation after transscleral cyclophotocoagulation

Transscleral cyclophotocoagulation (TSCPC) is used to treat patients with refractory glaucoma.[1,2] TSCPC is a destructive procedure of the ciliary processes leading to decreased aqueous humor production.[2] Because of complications such as hypotonia and phthisis bulbi, TSCPC is usually opted as a therapy for eyes with low visual potential and poor response to other medical or surgical interventions.[2] However, IOP changes in the very early postoperative period and its clinical implications have not been addressed yet. This study evaluates the immediate changes in intraocular pressure (IOP) after TSCPC.

In a prospective interventional design, we evaluated the changes of IOP after TSCPC in a series of 10 patients with refractory glaucoma. IOP was measured with a Tono-Pen Avira (Reichert, Buffalo, NY, USA) right before the procedure, immediately after TSCPC, 3 h, 1 day, 3 days, 7 days, and 28 days thereafter. The procedures were performed in the operating room under retrobulbar anesthesia using 2 ml of 2% lidocaine, and intravenous sedation with propofol and cardiopulmonary monitoring by anesthesia team. The starting laser parameters were power of 2000 mW and duration of 2000 ms. On the basis of the pop-sound response, power was adjusted on the steps of 250 mW.[3] All patients received subconjunctival injections of 4 mg dexamethasone at the conclusion of surgery. Those with immediate post-laser high IOP received intravenous 20% mannitol (1 g/kg). Postoperatively, all patients were instructed to use topical 1% prednisolone acetate 4 times daily (tapered over a 6 weeks period), and glaucoma medications were adjusted as required.

Table 1 summarizes the patients' demographic data, laser settings, and IOP changes after TSCPC. All but one patient showed a significant increase in IOP immediately after TSCPC (mean IOP, 31.4 ± 9.8 before *vs* 44.3 ± 14.3 mm Hg immediately after the procedure; $P = 0.012$, Wilcoxon-signed rank test). Immediately after the procedure, all patients had IOP ≥ 30 mm Hg, 5 (50%) had IOP ≥ 40 mm Hg, and 3 (30%) had IOP ≥ 50 mm Hg (Table 1). Three hours after the procedure, despite receiving the intravenous mannitol, six patients had IOP ≥ 30 mm Hg and two had IOP ≥ 50 mm Hg. However, on the first postoperative day, no patient had IOP ≥ 30 mm Hg. The pattern of IOP changes during the first month after TSCPC is depicted in Figure 1.

The main finding of the present study was a significant IOP elevation in almost all eyes immediately after TSCPC. This could be dangerous in patients with advanced glaucomatous optic nerve damage and may result in further optic nerve injury, temporary central retinal artery occlusion, and ischemia-reperfusion retinal injury.[4] The laser-induced coagulative necrosis and disruption of ciliary processes may increase intraocular volume by creating air bubbles (which are typically heard as 'pop-sound' during TSCPC)[2,5] and lead to immediate IOP rise, which could not be handled properly by the impaired trabecular meshwork. The IOP elevation also happened in two patients that received only 12 spots of laser therapy

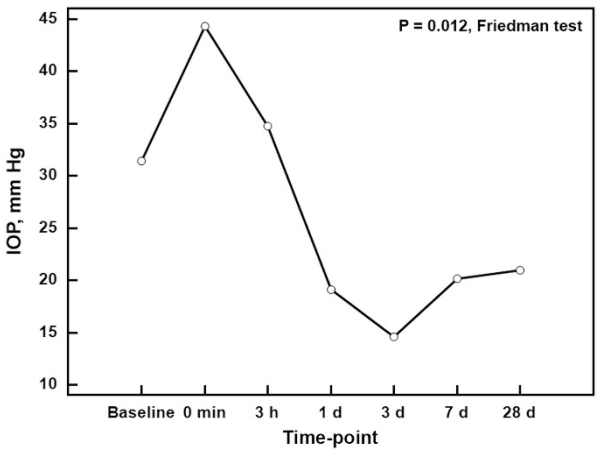

Figure 1 The intraocular pressure profile after transscleral cyclophotocoagulation.

Table 1 Data summary of 10 glaucoma patients who underwent transscleral cyclophotocoagulation

Case	*Age (years)*	*Sex*	*Eye*	*Lens status*	*Number of glaucoma medication before TSCPC*	*TSCPC*			*IOP (mm Hg)*		
						Number	*Power (mW)*	*Duration (ms)*	*Immediately before TSCPC*	*Immediately after TSCPC*	*3 h*
1	65	F	R	Pseudophakic	3	24	1500	2000	44	35	50
2	42	F	L	Pseudophakic	3	25	2000	3000	34	42	24
3	19	M	L	Phakic	3	24	2000	2500	22	35	30
4	12	F	L	Phakic	3	24	2000	2000	42	80	55
5	58	M	R	Apkakic	3	24	3000	4000	44	52	33
6	70	M	L	Apkakic	3	24	2000	2000	35	52	26
7	4	F	R	Phakic	4	14	2000	2000	23	39	32
8	70	F	R	Pseudophakic	3	26	3000	2750	20	32	25
9	1	F	L	Apkakic	4	12	2500	2800	20	36	13
10	4	M	L	Apkakic	3	12	2000	2000	30	40	35

Abbreviations: IOP, intraocular pressure; L, left; R, right; TSCPC, transscleral cyclophotocoagulation.

with a power and duration around 2000–2500 mW and 2000–2800 ms, respectively. Therefore, we believe that the IOP needs to be checked immediately after TSCPC and managed properly if it is elevated.

Conflict of interest

The authors declare no conflict of interest.

References

1 Hennis HL, Stewart WC. Semiconductor diode laser transscleral cyclophotocoagulation in patients with glaucoma. *Am J Ophthalmol* 1992; **113**(1): 81–85.
2 Amoozgar B, Phan EN, Lin SC, Han Y. Update on ciliary body laser procedures. *Curr Opin Ophthalmol* 2017; **28**(2): 181–186.
3 Alzuhairy S, Albahlal A, Aljadaan I, Owaidhah O, Al Shahwan S, Craven ER *et al*. Intraocular pressure outcomes following transscleral diode cyclophotocoagulation using long and short duration burns. *J Glaucoma* 2016; **25**(9): e782–e786.
4 Hartsock MJ, Cho H, Wu L, Chen WJ, Gong J, Duh EJ. A mouse model of retinal ischemia-reperfusion injury through elevation of intraocular pressure. *J Vis Exp* 2016; e-pub ahead of print 14 July 2016; doi:10.3791/54065.
5 Sivagnanavel V, Ortiz-Hurtado A, Williamson TH. Diode laser trans-scleral cyclophotocoagulation in the management of glaucoma in patients with long-term intravitreal silicone oil. *Eye* 2005; **19**(3): 253–257.

MR Razeghinejad[1,2], A Hamid[2] and MH Nowroozzadeh[2]

[1]Department of Ophthalmology, Truhlsen Eye Institute, University of Nebraska Medical Center, Omaha, NE, USA
[2]Department of Ophthalmology, Poostchi Eye Research Center, Shiraz University of Medical Sciences, Shiraz, Iran
E-mail: nowroozzadeh@hotmail.com

Eye (2017) **31,** 1249–1250; doi:10.1038/eye.2017.59; published online 7 April 2017

Sir,

Assessment of a three-generation pedigree with Fuchs endothelial corneal dystrophy with anticipation for expansion of the triplet repeat in the *TCF4* gene

Fuchs endothelial corneal dystrophy (FECD) has a significant genetic component to its pathogenesis and several genetic risk factors for FECD have been discovered. The first genetic risk factor for FECD, transcription factor 4 (*TCF4*), was detected by a genome-wide association study.[1] A trinucleotide repeat, $(CTG)_n$ also known as CTG18.1, is located within an intron of the *TCF4* gene.[2] Expansion of the *TCF4* trinucleotide repeat is associated with FECD and expansion to >40 repeats confers a hazards ratio of 1.64 for a corneal transplant.[3] Anticipation, earlier onset of disease with increasing severity in successive generations, occurs in diseases caused by trinucleotide repeat expansions (ie, Huntington's disease).[4]

Case report

We investigated the potential role of *TCF4* trinucleotide repeat expansion in a three-generation pedigree with a history suggestive of anticipation. FECD was diagnosed via slit lamp biomicroscopy by a board-certified ophthalmologist with cornea fellowship training, graded using a modified Krachmer scale of 0 (no disease)–6 (>5 mm of central confluent guttae with corneal edema),[5] and examined via confocal microscopy (ConfoScan 4, Nidek Technologies, Fremont, CA, USA) and corneal tomography (Pentacam HR, Oculus, Lynnwood, WA, USA).

Three family members were diagnosed with FECD[5] and displayed features of anticipation. The age at diagnosis occurred earlier in each successive generation, with the grandmother, mother, and daughter receiving the diagnosis of FECD at 63, 48, and 27 years of age, respectively (Figures 1 and 2). The severity of corneal endothelial disease was greater in each successive generation, with the grandmother, mother, and daughter displaying a modified Krachmer Grade of 2, 4, and 6, respectively, and central corneal thickness (right/left) of 535/537, 568/572, and 627/602 μm, respectively.

The size of the *TCF4* trinucleotide repeat was evaluated in members of the FECD pedigree to determine if an expansion of the repeat might be the source of anticipation. The *TCF4* trinucleotide repeat was amplified from family member DNA samples with the polymerase chain reaction, cloned, and sequenced using standard methods.[6] Each family member's genome has two copies of the *TCF4* gene. Analysis of these DNA sequences revealed the exact number of *TCF4* trinucleotide repeats in each family member's genome. The grandmother, mother, and

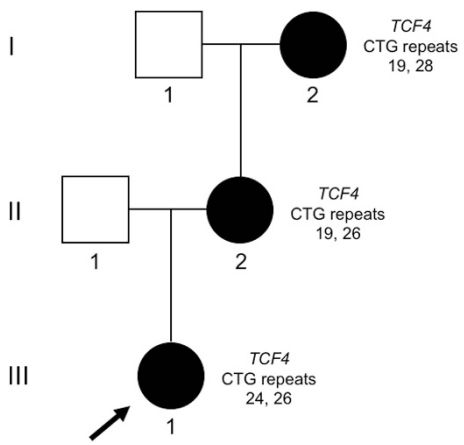

Figure 1 Three-generation FECD pedigree. The proband is indicated with an arrow and symbol III-1, while the proband's mother and grandmother are indicated by symbols II-2 and I-2, respectively. Family members diagnosed with FECD are indicated with symbols that are shaded black. The number of *TCF4* trinucleotide repeats in each family member's genome was determined by PCR amplification and DNA sequencing, and is indicated to the right of their pedigree symbol.