Anterior chamber depth and intraocular pressure following panretinal argon laser photocoagulation for diabetic retinopathy

To the Editor: Ocular photocoagulation uses heat produced through the absorption of light by ocular pigments. Absorption of light can take place either in the tissue to be photocoagulated or in a neighboring tissue, from which heat is then transferred to the tissue of interest by thermal conduction. Thermal damage is caused by chemical changes that result when the ocular tissues are heated to temperatures high enough to denature proteins or other large molecules.[1-3] A temperature increase of 10°C to 20°C is sufficient to produce the desired chemical changes. Photocoagulation is used in the management of retinal diseases such as diabetic retinopathy, diabetic maculopathy, subretinal neovascularization, retinal vascular abnormalities, and retinal breaks or tears of various types.[3] Panretinal argon photocoagulation (PRP) is commonly performed for the treatment of proliferative diabetic retinopathy, ischemic central retinal vein occlusion, and other causes of retinal ischemia.[4] Complications of PRP include thermal injury to the cornea, iris and lens, visual field loss, haemorrhage, macular edema, and elevated intraocular pressure with or without angle closure.[4-6] Many transient changes after PRP occur only when a large area of the retina is treated in one session or in closely spaced sessions.[5] In the study, we investigated the effects of PRP on anterior chamber depth changes and early and late period intraocular pressure.

We studied 170 eyes with diabetic retinopathy (Type 2 diabetes). All patients had proliferative or high-risk preproliferative retinopathy. PRP was applied to all eyes under topical anesthesia. Patients with closed angle, rubeosis iridis, open angle or neovascular glaucoma, and other intraocular disorders were excluded. Examination before and after PRP comprised the fundus, chambers angle, visual acuity, slit-lamp examination, fundus Fluorescein angiography and IOP. IOP was measured with a Goldman applanation tonometer. Eyes with IOP over 30 mm Hg were treated with timolol maleate 0.05%. Anterior chamber depth was evaluated by the same person using A-scan ultrasonography. IOP measurements and anterior chamber depth were measured after cycloplegia. Cycloplegia was applied using cyclopentolate hydrochloride 1%. The intraocular pressure of each eye was measured during the first examination before PRP and at the first hour, first day, and the first, third and sixth months after PRP. Anterior chamber depth was also measured before PRP, and after the first hour and first day after PRP. Patients with an IOP >30 mm Hg were treated by antiglaucomatous agents. The initial treatment protocol of PRP was 850 to 1200 burns, the intensity varied from 0.2 to 1.0 W, the duration of exposure varied from 0.1 to 0.2 second, and the spot size 200 to 500 µ. Additional photocoagulation was performed if deemed necessary by the treating ophthalmologist. The paired t-test was used in the statistical analysis.

The study included 85 patients with Type 2 diabetes mellitus, aged 38 to 77 years (mean, 62.0 years). In the first examination, we found high-risk nonproliferative diabetic retinopathy in 68 eyes and proliferative diabetic retinopathy in 102 eyes. While IOP was significantly elevated in the first hour after PRP, it was not significantly different in the following measurements (Table 1). IOP over 30 mm Hg was seen in four eyes in the first hour after PRP, and these eyes were treated with timolol maleate 0.05% . IOP decreased to normal levels on the first day after PRP in all these eyes. While anterior chamber depth was significantly shallow in the first hour after PRP, it was not statistically different from the first day after PRP (Table 2). None of the eyes developed neovascular glaucoma or rubeosis iridis during the observation period.

Table 1. Intraocular pressure values (mm Hg).

Before PRP	After PRP				
	First hour	First day	First month	Third month	Sixth month
16.40±0.43	17.70±0.44*	16.57±0.32	16.02±0.47	16.30±0.40	16.61±0.37

*$P<0.05$ (paired t-test)

Table 2. Values of anterior chamber depth (mm).

Before PRP	After PRP	
	First hour	First day
3.06±0.04	2.99±0.04*	3.04±0.03

*$P<0.05$ (paired t-test)

Many investigators have reported a transient elevation during the first few hours after panretinal coagulation;[7-9] one reported a decrease within the first six months.[10] In this study, we detected an elevation of IOP following panretinal photocoagulation within a few hours. However, IOP was statistically different on the first day compared with the first, third or sixth month after PRP. Many transient changes after PRP occur only when a large area of the retina is treated in one session or in closely spaced sessions. These changes are due to the exudation of fluid from the choroids and retina. The exudation of fluid into the posterior segment causes a forward displacement of the lens-iris diaphragm. The forward movement of the iris-lens diaphragm often is associated with a rise in the intraocular pressure. The pressure probably rises because exudation of fluid into the posterior segment occurs faster than aqueous fluid can leave the anterior chamber through the trabecular meshwork, and the outflow facility usually is decreased. Another possible pathogenic mechanism is compression of episcleral veins by the flange of the fundus contact lens used in delivering the photocoagulation treatment.[7] Kaufman et al studied 1742 treated and untreated eyes, and IOP was measured during the first five years after PRP. They reported that their findings were not consistent with the suggestion that PRP might cause a meaningful long-term reduction in IOP. However, they found that neovascular glaucoma occurred more frequently among the untreated eyes. In our study, no difference was found on the first day, and in the first, third, and sixth months after PRP in IOP.

One of the major ophthalmic complications of diabetes mellitus is neovascular glaucoma. Several publications have suggested that PRP could cause the regression of rubeosis iridis and angle vascularization.[11-13] According to Wand et al.[11] PRP reduced or eliminated the stimulus for new vessel formation in the posterior pole and they showed that PRP in eyes with proliferative retinopathy decreased the incidence of rubeosis iridis, angle neovascularization, and probably neovascular glaucoma. In our cases, neovascular glaucoma did not develop. We think that PRP can take a preventive role against neovascular glaucoma. Whatever the cause, it appears that PRP reduces or eliminates the stimulus for new vessel formation in the posterior pole.

In this study, we detected anterior chamber depth shallowness in the first few hours after PRP. However, any differences were not detected on the first day of PRP. Mensher[14] had studied anterior chamber depth and angle changes after PRP for diabetic retinopathy. All patients treated with argon laser had shallowing of the anterior chamber and narrowing of the angle with angle closure in 31%. Blondeau[9] reported intraocular pressure increased in 17 of 18 eyes treated for diabetic retinopathy with argon laser PRP. All eyes had open anterior chamber angles before treatment and closed angles developed in three eyes. In our study, angle closure developed in one patient and it was treated with a cycloplegic agent. These studies show that anterior chamber angle closure may develop after PRP in open angle eyes as well. Angle closure glaucoma following PRP is believed to be caused by anterior rotation of the ciliary body secondary to a ciliochoroidal fluid and detachment. This is postulated as a result of transudation of fluid from the choroidal vasculature into the choroid and suprachoroidal space secondary to a thermally induced choroiditis and choroidal vascular occlusions.[15,16] According to Mensher,[14] anterior chamber shallowing after retinal photocoagulation is a vascular phenomenon, and the choroidal system is most likely affected. Hayreh and Baines have shown that anterior chamber shallowing can be produced acutely by an obstruction in the vortex venous system.[9,14] It has been demonstrated that choroidal vascular occlusions may occur after photocoagulation. It is possible that anterior fluid transudation may play a role in altering vitreous volume dynamics causing a shallowing of the anterior chamber. Risk factors for the development of the ciliochoroidal effusion after PRP include a greater number of laser applications, shorter axial length, and a greater percentage of retinal surface area treatment.[15]

In conclusion, a shallowing in the anterior chamber and elevation of IOP may occur in the first few hours after PRP, but treatment is hardly needed. However, patients with borderline IOP before PRP should be followed carefully for an elevation in first few hours after PRP.

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