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ORIGINAL PAPER

Comparing Two Techniques of Panretinal Photocoagulation on Visual Acuity on Patients with Proliferative Diabetic Retinopathy

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e wanted to examine which of two panretinal photocoagulation (PRP) techniques, classical panretinal photocoagulation (CPRP) and modifield peripheral panretinal photocoagulation PPRP), causes less decline of visual acuity (VA) due to macular edema (ME) in patients with proliferative diabetic retinopathy (PRD). This clinical study includes 180 eyes with PDR with initial papillar neovascularization. The patients were divided into two groups according the RP. PPRP and CPRP showed the decline of VA in all patients, more pronounced in the CPRP group after one week. After three and six months, with CPRP and PPRP the values of VA was stabilized. The result suggests that eyes with PDR and starting epipapillar neovascularisation should be treated with PPRP with priority given to CPRP because it caused better VA. Key words: Techniques, panretinal photocoagulation, visual acuity, macular edema.

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1. INTRODUCTION

The data in world literature confirms that diabetic retinopathy (DR) is the most common cause of blindness in developed countries (1). Proliferative diabetic retinopathy (PDR) is associated with Macular Edema (ME) (2, 3, 4), with relatively good Visual Acuity (VA) (4, 5, 6). Argon laser panretinal photocoagulation (PRP) is the most used efficacious method for the PDR (7, 8, 9). The mode of action of PRP is probably the destruction of ischemic retina and the increasing of retinal oxygenation with a new choriretinal vascular shunt created in the laser's scors, the elimination of peripheral retina, metabolic and haemodinamic parameters are increased consequently (9, 10, 11).

Despite the panretinal photocoagulation (PRP) benefits, the therapy procedure has some harmful side-effects and complications, among then the most significant producing of ME (12, 13, 14, 15, 16, 17, 19), which leads to temporal or permanent VA decline (7, 8, 9, 16, 18, 19, 20, 21, 22, 23, 24).

2. AIM

The aim of this study is to compare the techniques of PRP in treatment of PDR and to compare which technique has fewer complications on ME and VA. The aim is to show which technique of panretinal photocoagulation is more effective and has fewer complications in in the decline of visual acuity.

3. PATIENTS AND METHODS

All patients in this study had diabetes and came to observation in the surgery for retinal diseases at the Eye Clinic, Clinical Hospital Split. One hundred and thirty type 1 diabetic patients with PDR and incipient papillary neovascularization (papillary neovasculates or within 1 papillary diameter from papillae), which is a high risk indicator for serious sight loss, were enrolled in this clinical prospective study. We used a blue-green argon laser and wide-angle Mainster's WF panfundoscope (that way spots were magnified by 200 μ), so 650 applied spots are equal as 900 spots applied using Goldmann's contact corneal lens, which is the top recommended laser therapy in one session. Panretinal (mild scatter) photocoagulation was performed in two different ways. Central Classical panretinal photocoagulation (CPRP) was performed with "mild-scatter" technique number 650 spots with 500µ, exposition 0.5 second, 2 optic disc diameter from macula to pre-equatorial (15° to 80°). With the peripheral panretinal photocoagulation (PPRP) technique, 650 spots, 500µ in diameter, exposition 0.5 second, 5 optic disc diameter from the centre of the macula (40°-105°) was performed. The patients were divided into two groups according to the type of laser treatment. The first group of 65 patients (87 eyes) was treated by PPRP, and the other group of 65 patients (93 eyes) CPRP. Fundus was examined by direct and indirect ophthalmoscopy, and the eventual presence of macular edema was examined with central part of Goldmann's contact corneal lens. VA assessment was done in regular time intervals; before treatment, one week, 3 months, and 6 months after laser therapy.

The mean age in the group of patients treated with PPRP was 37.7 years (SD \pm 12.4), range 20-69 years (Table 1). The mean age of patients treated with

CPRP was 41.6 years (SD \pm 14.5). The youngest patient $\overline{1}$ was 18, and the oldest 66 - years old (Table 1).

The duration of diabetes before treatment amounted to between 9-31 years. The mean value of diabetes duration for PPRP group was 16.3±5.23 years. The shortest duration of diabetes was 9 years, and the longest 28 years. The mean value of diabetes duration before treatment in CPRP group was 18.1±5.24 years. The shortest period of illness was 10 years, and the longest 31 years. There was no statistically significant difference between diabetes duration in the two groups (p=0.06), Table 1.

In total there was 66 (50.8%) men and 64 (49.2%) women, Table 2. There were some more men in the CPRP group, exactly 35

(53.8%), and more women in PPRP group, exactly 34 (52.3%). The difference between these two groups was not statistically significant (p=0.29).

ME was found in 45 (48.4%) eyes before being treated with CPRP, in 46 (52.9%) eyes being treated with PPRP. (Table 3)

VA before treatment for both groups was 0.2-1.0. In patients treated with CPRP, VA was 0.2-0,6 in 52(56%) eyes, and 0.7-1.0 in 41(44%) eyes. In patients treated with PPRP, VA was 0.2-0.6 in 47(54%) eyes, and 0.7-1.0 in 40(46%) eyes.

Statistical analysis of obtained results was performed on the computer program Statistica for Windows v 5.0 (StatSoft, Inc 1995), by calculating x^2 and **t**-test. The significant difference was understood if p > 0.05.

reatment	Number		Exis	ting macu	ılar edema at	fter treatme	nt
	eyes	1 v	veek	3 n	nonths	6 m	onths
	Ň	Ν	%	Ν	%	Ν	%
CPRP	93	48	51.6	18	19.4	17	18.3
PRP	87	27	32	12	13.8	9	10.3
$(\chi^2$ -test)		0.0	010	(0.389	0.1	66

Table 4. Macular edema in eyes treated with CPRP and PPRP. CPRP, classical panretinal photocoagulation, PPRP, peripheral panretinal photocoagulation

		X±SD(ran	ge)	
		Mean visual	acuity	
Treatment	Before	After	After	After
	therapy	1. weak	3 months	6 months
CPRP	0.75±0.22	0.60±0.29	0.69±0.27	0.7±0.26
	0.2-1.0	0.1-1.0	0.2-0.27	0.2-1.0
PPRP	0.72±0.22	0.67±0.21	0.73±0.22	0.71±0.26
	0.3-1.0	0.1-1.0	0.2-1.0	0.2-1.0
P (t-test)	0.025	0.081	0.749	0.71

Table 5. Mean visual acuity before and after PPRP and CPRP treatment. CPRP, classical panretinal photocoagulation, PPRP, peripheral panretinal photocoagulation

	Age		Duration of illness treatment		
	X± SD	range	$X \pm SD$	range	
CPRP	41.6±14.5	18-66	18.1±5.24*	10-31	
PPRP	37.7±12.4	20-69	16.3±5.23*	9-28	

Table 1. Age patients and diabetes mellitus duration in years before treated with CPRP and PPRP. *P = 0.06, CPRP, classical panretinal photocoagulation, PPRP, peripheral panretinal photocoagulation

Treatment	М	en	Won	nen	То	otal	
	N	%	Ν	%	Ν	%	
CPRP	35	53.8	30	46.2	65	50	
PPRP	31	47.7	34	52.3	65	50	
Total	66	50.7	64	49.2	130	100	

Table 2. Patients by gender. P = 0.29, CPRP, classical panretinal photocoagulation, PPRP, peripheral panretinal photocoagulation

		Ma	Macula edema before treatment				
		Y	Yes		No		
treatment	number eyes	Ν	%	Ν	%		
CPRP	93	45	48.4	48	51.6		
PPRP	87	46	52.9	41	47.1		

Table 3. Macular edema before treatment with CPRP and PPRP. $\chi 2 = 0.2 P = 0.5447$ CPRP, classical panretinal photocoagulation PPRP, peripheral panretinal photocoagulation

4. RESULTS

The statistically significant difference in the number of eyes with ME between each therapy after 1week, 3 and 6 months are presented on Table 4. After 1 week, ME is 1.6 more in the group treated with CPRP in relation to the group treated with PPRP. This difference in ME is statistically significant (p=0.010). After 3 and 6 months, treatment with CPRP and PPRP there differens are not statistically significant (there was no statistically significant difference between treatment with CPRP and PPRP) p=0.389; p=0.166.

The mean VA before treatment in the PPRP group after international optotipes was 0.72 ± 0.22 , and for the CPRP group it was 0.75 ± 0.22 and these values were statistically different (p=0.0249) Table 5. VA worsened a weak later after the therapy, the mean value for the CPRP group was 0.60 ± 0.29 , and for the PPRP group it was 0.67 ± 0.21 . There was no statistically significant difference (p=0.081). Mean VA after 3 months was still worsening, and for the PPRP group amounted to 0.73 ± 0.22 and for the CPRP group 0.69 ± 0.27 , and there was no statistically significant difference (p=0.749). Mean VA in the PPRP group, six months after laser therapy, amounted to 0.71 ± 0.26 , and was higher than in CPRP group which amounted to 0.7 ± 0.26 . Even though the values were different, there was no statistically significant difference (p=0.71).

5. DISCUSSION

Laser therapy is successful in treatment of DR, which was confirmed by several large randomized studies (7, 8). PRP is used as treatment of PDR. In despite of PRP advantages, the therapeutical procedure can have harmful side effects and complications, among which the most important is the creation and exacerbation of ME, which leads to transient or permanent loss of VA(16-24). Several days after treatment with PPRP and CPRP, ME is worsening, but 1,6 more than CPRP. After three and six months ME was less pronounced in patients treated with PPRP. Blankenship (25) finds different values, especially the worsening of ME with CPRP treatment of 18%, ameliorated 19% with PPRP. These differences can be explained by the different mode of treatment solely. Some authors find lessening of ME in 8-46% patients (26-30). These differences are probably caused by different selection of patients, status of DR, age and different mode of treatment. Our results show worsening of VA after 6 months of 2% (from 0.72 to 0.71) in the PPRP group, and of 6% (0,75-0,71) in CPRP group. Even though there is a slight difference between the two study groups, there was no statistically significant difference. One large multicentric prospective randomized study ETDRS (8) brought VA results 5 years after PRP therapy and they have reported VA worsening of 2.5% for early therapy and 3.7% for postponed PRP. Štriga et al. found worsening of VA after PRP that amounted to two or more rows of Snellens optotipe (18). Blankenship (25) found worsening of two or more rows, 24% after CPRP and 8% after PPRP, and McDonald (20) found worsening of 25% after PRP. Other groups of authors found bettering of VA from 8% to 89% depending on results evaluation (31-36). Their results are hardly comparable with the present study because they treated patients with different DR grade (16, 36, 37,38), and they did not use the same laser techniques (16, 28), more laser treatment (25, 29, 30, 32, 35, 36, 38) with larger groups of patients (16, 22, 24, 30, 32).

Although, at the beginning of the present study, the stadium of PDR in both study groups was similar in clinical and functional ways, we can deduce that the result differences are exclusively due to PRP technique. On the basis of these results, it can be stated that eyes with PDR and recent epipapillary neovascularizations treated with PPRP develop immediately after treatment less therapy induced ME and better VA.

6. CONCLUSIONS

VA was deteriorated one week after treatment with CPRP and PPRP due to preexisting or macular edema worsening. After 3-6 months of beginning of treatment, ME and VA was not changed significantly. VA was slightly better in patients treated with PPRP. Based on our results it can be concluded that eyes with PDR and epipapillary neovascularizations can be treated by modified PRP (PPRP), and that it should have priority to classical PRP (CPRP), because PPRP causes less ME and less VA loss.

REFERENCES

- Klein R, Klein BEK, Moss SE. Epidemiology of proliferative diabetic retinopathy. Diabetes Care. 1992; 15: 1875-1891.
- Klein R. Prevention of visual loss from diabetic retinopathy. SURV Ophthalmol. 2002; 47: 266-272.
- Gardner TW, Eller AW, Friberg TR, Reduction of severe macular edema in eyes with poor vision after panretinal photocoagulation for proliferative diabetic retinopathy. Graefes Arch Clin Exp Ophthalmol. 1991; 229: 323-328.
- Margolis R, Singh RP, Bhatnager P, Kaiser PK. Intravitreal triamcinolone as adjuction treatment to laser panretinal photocoagulation for concomitant proliferative diabetic retinopathy and clinically significant macular edema. Acta Ophthalmol. 2008; 86: 105-110.
- Maia O Jr, Akahasy Wy, Bonamoni MT, Marbach Rf, Kara-Jose N. Visual stability in diabetic rethinopathy treated by panretinal laser photocoagulation. Arq Bras Endocrinol Metabol. 2007; 51: 575-580.
- Shimura M, Yasuda K, Nakazawa T, Tamai M. Visual disfunction after panretinal photocoagulation in patients with severe diabetic retinopathy an good vision. Am J Ophthalmol. 2005; 140: 8-15.
- Diabetic Retinopathy Study Research Group. Photocoagulation treatment of proliferative diabetic retinopathy: Clinical application of Diabetic Retinopathy Study (DRS) findings, DRS report No. Invest Ophthalmol Vis Sci. 1981; 88: 583-600.
- Early treatment diabetic retinopathy study research group: Early photocoagulation for diabetic retinopathy. ETDRS report No 9. Ophthalmology. 1991; 98 (Suppl): 765-85.
 Čupak K, Laktić N, Padovan S. Primjena argonskog la-
- Čupak K, Laktić N, Padovan S. Primjena argonskog lasera u nekih vaskularnih bolesti mrežnice. In: Čupak K, Ed. Fotokoagulacija. Laser u oftalmologiji. Čakovec: Zrinjski, 1979: 40-49.
- Pournaras CJ, Tsacopoulos M, Strommer K, Gilodi N, Levenberger PM. Scatter photocoagulation restores tissue hypoxia in experimental vasoproliferative microangiopathy on miniatures pigs. Ophthalmology. 1990; 97: 1329-1333.

- American Academy of Ophthalmology. Retina and vitreus. Basic clinical science course 1993-4, section 12, San Francisco: AAO: 1993: 61.
- Zein WM, Noureddin BN, Jurdi FA, Schakal A, Bashshur ZF. Panretinal photocoagulation and intravitreal triamciolone acetonide for the management of proliferative diabetic retinopathy with macular edema. Retina. 2006; 26: 137-142.
- Lee CM, Olk RJ, Akduman L.Continued modifield grid and panretinal hotocoagulation for diabetic macular edema and proliferative diabetic retinopathy. Ophthalmic Surq Lasers. 2000; 31: 292-300.
- Wei ZY, Hu SH, Tang N, Wn J, Wang J. Effect of argon laser photocoagulation on diabetic retinopathy. Ci Yi Jun Da Xue Xue Bao. 2004; 24: 1313-1315.
- Walczynski M, Dzivqielewski K. Result of laser photocoagulation in patients with diabetic retinopathy developed as a complication of diabetes type 2. Klin Oczna. 2006; 108: 66-69.
- McDonald Hr, Schatz H. Macular edema following panretinal photocoagulation Retina. 1985; 5: 5-10.
 Davis MD. Proliferative diabetic retinopathy. U: Ryan
- Davis MD. Proliferative diabetic retinopathy. U: Ryan SJ, ur. Retina. 2. ed. St Luis: CV Mosby Co, 1994: 1319-1359.
- Štriga M, Katušić D. Traumatic lesion and complications after argon laser photocoagulation treatment of diabetic retinopathy. Diab Croat. 1990; 19: 87-98.
- ŠtrigaM, Katušić D, Ćurković T. Functional lesions and results following argon laser treatment for diabetic retinopathy. Diab Croat. 1990; 19: 99-108.
- McDonald Hr Schatz H. Visual loss following panretinal photocoagulation for roliferative diabetic retinopathy. Ophthalmology. 1985; 92: 388-393.
- Ferris FL, Podgor MJ, Davis MD. Macular edema in diabetic retinopathy study atients. Diabetic retinopathy study report number 12. Ophthalmology. 1987; 94: 754-760.
- Meyers SM. Macular edema after scatter photocoagulation for proliferative diabetic retinopathy. Am J Ophthalmol. 1980; 90: 210-216.
- Aylward GW, Pearson RV, Jagger JD, Hamilton AM. Extensive argon laser photocoagulation in treatment of proliferative diabetic retinopathy. Br J Ophthalmol. 1989; 73: 197-201.
- 24. Zhang CF. Clinical study on preproliferative and proliferative diabetic retinopathy. Chung Kuo I Hsueh Ko Hsueh Yuan Hseueh Pao. 1989; 11: 224-225.
- Blankenship GW. A clinical comparison of central and peripheral argon laser panretinal photocoagulation for proliferative diabetic retinopathy. Ophthalmology. 1988; 95: 170-177.
- Vine AK. The efficacy of additional argon laser photocoagulation for persistent, severe proliferative diabetic retinopathy. Ophthalmology. 1985; 92: 1532-1537.
- Wade Ec, Blankenship GW. The effect of short versus long exposure times of argon laser panretinal photocoagulation on proliferative diabetic retinopathy. Graefes Arch Clin Exp Ophthalmol. 1990; 228: 226-231.
- Odonoghue HN. Laser treatment in diabetic retinopathy. Trans Ophthalmol Soc U K. 1982; 102: 468-470.
- Zhang CF. Laser treatment for pre-proliferative and proliferative diabetic retinopathy. Chung Hua Yen Ko Tsa Chih. 1989; 24: 329-332.
- Prskavec FH, Fulmek R, Kleman C, Stelzer N. Changes in the visual field and dark adaptation following panretinal photocoagulation in diabetic retinopaphy. Klin Monatsbl Augenheilkd. 1986; 189: 385-387.
- Fine SL, Patz A. Ten years after Diabetic Retinopathy Study. Ophthalmology. 1987;94: 739-740.
- Doft BH, Blankenship GW. Retinopathy risk factor regression after laser photocoagulation for proliferative diabetic retinopathy. Ophthalmology. 1984; 91: 1453-1457.
- Asher R, Hunt S, Hamilton AM, Townsend C. Photocoagulation for optic disc new vessels in diabetic mellitus. Int Ophthalmol. 1981; 3: 79-85.
- Lim As, Khoo CY, Ang BC, Chiang C. Argon laser photocoagulation in diabetic retinopathy: five years review of 697 treated eyes. Ann Acad Med Singapore. 1985; 14: 252-260.
- Atmaca LS, Idil A, Gunduz K. Dye laser treatment in proliferative diabetic retinopathy and maculopathy. Acta Ophthalmol Scand. 1995; 73: 303-307.
- Bailey CC, Sparrow IM, Grey RH, Cheng H. The national diabetic retinopathy laser treatment audit. III. Clinical outcomes. Eye. 1999; 13: 151-159.
- Singer DE, Nathan DM, Fogel HA, Schachat AP. Screening for retinopathy. Ann Intern Med. 1992; 116: 660-671.
- Khosla PK, Gupta V, Tewari HK, Kumar A. Automated perimetric changesfollowing panretinal photocoagulation in diabetic retinopathy. Ophtalmic Surg 1993;24:256-61.