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The Role of Oxidative Stress after Retinal Laser Photocoagulation in Nonproliferative Diabetic Retinopathy

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A B S T R A C T

Diabetic retinopathy (DR) represents the most common chronic complication of diabetes, and it is the leading cause of new cases of blindness in patients between 20–74 years old in developed countries. Laser photocoagulation (LF) represents an efficacious approach to the treatment of DR. Oxidative factors, such as free radicals (FR), are continuously generated in aerobic organisms as a result of different metabolic processes. It is well known that oxidative stress plays a role in the development of DR. The aim of this study was to evaluate the thermal effects of the scatter retinal laser photocoagulation technique on the production of FR. A total of 90 patients were enrolled in this study. They were divided in 3 groups: 30 diabetic patients with DR, 30 diabetic patients without DR, and 30 control individuals without diabetes mellitus (DM). Full scatter retinal LF was performed in all patients with DR. We measured the concentrations of superoxide dismutase (SOD), glutathione peroxidase (GPOD), catalase, and total antioxidative status (TAS). Of the 30 DR patients, 13 showed the appearance or worsening of macular edema after LF, whereas the other 17 patients showed no change. Thirty days after LF, improvement in visual acuity was observed, but this change was not statistically significant. The mean plasma or erythrocyte lysate concentrations of various antioxidants were significantly lower in the diabetic patients without DR compared to the individuals without DM and in the diabetic patients with DR compared to the individuals without DM; the diabetic patients with DR did not show lower concentrations of the antioxidants compared to the diabetic patients without DR. The concentrations of SOD, GPOD, catalase, and TAS were significantly lower in the diabetic patients with DR after retinal scatter LF, which could be the consequence of retinal oxidative stress caused by the LF thermal effect.

Key words: *oxidative stress, diabetes mellitus, light coagulation, retina, Croatia*

Introduction

Diabetes mellitus (DM) is a complex disease with multiple influencing factors; it is a chronic metabolic disorder that is frequently associated with progressive vision loss.

Diabetic retinopathy (DR) represents the most common chronic complication of diabetes. It is the leading cause of new cases of blindness in patients between 20–74 years old in developed countries[1]. The mean incidence of DR in the USA and Europe is 40.3%[2]. The incidence of blindness is proportional to the degree of retinopathy and is related to age and the duration of diabetes; the relative risk of the progression to blindness in diabetic patients is 29 times higher than in non-diabetic patients[3].

Laser photocoagulation (LF) represents an efficacious approach to the treatment of DR[4,5], and its absolute indications are macular edema (ME) and severe nonproliferative diabetic retinopathy (NPDR). The procedure produces a 50% reduction in the risk of progression to the proliferative form of diabetic retinopathy (PDR)[6,7]. Complications of LF include the following: Bruch's membrane rupture[8], choroidal neovascularization[9], retinal pigment epithelium atrophy[10], and macular edema[11–15].

Oxidative factors, such as free radicals (FR), can be described as atoms and/or molecules with one or more unpaired electrons in the outer orbit or reactive oxygen compounds (O_2). These factors are continuously being

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generated in aerobic organisms as a result of different metabolic processes; they include the superoxide radical (O_2^-), hydrogen peroxide (H_2O_2), and the hydroxyl radical (OH). These highly reactive molecules can react with biomolecules such as lipids, proteins, nucleic acids, and carbohydrates[16,17]. These metabolites are responsible for the peroxidation of lipids (POL), which is defined as a set of reactions that involve cellular membrane polyunsaturated fatty acids (phospholipids, glycolipids, and sterols)[18].

Antioxidants are compounds that significantly decrease or prevent the oxidation of any substrate and are typically enzymes or small non-enzymatic protein molecules[19]. We may find a great number of enzymes with antioxidative effects; these include the following: superoxide dismutase (SOD) eliminates O_2 and glutathione peroxidase (GPOD) eliminates H_2O_2, hydroperoxide catalase, reduces H_2O_2, and vitamin C, and suppresses O_2^-.

It is well known that there is an imbalance between oxidative and antioxidative factors in diabetic patients[20–26].

The aim of this study was to evaluate the thermal effect of the scatter retinal LF technique on the production of FR and reactive oxygen species, which results in additional tissue damage. We also planned to evaluate the correlation between antioxidative status and the incidence of ME after scatter retinal laser photocoagulation. It is well known that the level of oxidative stress is high in diabetic patients with DR[27–31], what is also including in this study.

Materials and Methods

There were 90 patients enrolled in this study: 38 men and 52 women (45–60 years old). They were divided into 3 groups: 30 diabetic patients with DR, 30 diabetic patients without DR, and 30 individuals without DM. The DR group consisted of 30 male and female patients between 40 and 65 years old with type 1 and 2 diabetes who visited the Outpatient Department for Retinal Diseases of the Ophthalmology Department, Split University Hospital Center. All patients had a visual acuity of 20/100 (0.2) or better in each eye. Fundus examinations under mydriasis revealed severe NPDR with all the associated characteristics, which include numerous microaneurysms, intraretinal hemorrhages, vein abnormalities, hard lipid exudates, and cotton-wool exudates; some examinations also revealed macular edema. Patients with severe NPDR were selected using the EDTRS criteria. In particular, diabetic patients with level C (severe) and D (very severe) NPDR were included[32,33] based on previous studies that recommended the treatment of severe NPDR with scatter LF[4,6,7,34–36].The presence of macular edema was verified by fundoscopic exam, fundus photography, and fluorescein angiography (FA). The shortest duration of diabetes was 5 years. Scatter panretinal photocoagulation was performed in all patients. Patients with neovascularization of the optic disc or elsewhere were excluded from the study. We also excluded smokers, patients with vitreous hemorrhages (because of difficult visualization and LF) and hypertension (which is a risk factor the development of DR)[34] and patients who were using vitamins with antioxidative effects. In addition, 30 patients with type 1 or 2 DM who were 40–60 years old without any clinical signs of DR and $HbA_{1c}<8\%$ were included in addition to 30 individuals without DM (Table 1).

Full scatter retinal laser photocoagulation (LF) was performed in all patients with DR with the following parameters: 600–700 laser spots each measuring 200–500 nm in diameter, 200–300 mW of power, and 0.20 sec exposure were applied using a Zeiss diode laser[37]. We also used a Mainster's wide-field panfundoscope. Visual acuity was tested with international charts at a 5-meter distance before the treatment as well as 1 day, 7 days and 30 days after LF treatment. FA was conducted as an adjunctive method especially in cases with unexplained loss of visual acuity. FA and fundus photographs were obtained using a Zeiss fundus camera in the Outpatient Department for Retinal Diseases of the Ophthalmology Department, Split University Hospital Center.

Biochemical parameters were measured in the Clinical Laboratory at Split University Hospital Center. Venous blood specimens were obtained before and 2 hours after the retinal LF from the diabetic patients with DR, whereas venous blood specimens were collected only once from the diabetic patients without DR and the individuals without DM. It is well known that diabetic patients are permanently exposed to oxidative stress, which can be a possible reason for the changes in the antioxidant levels in those who were not treated with LF; however we hypothesized that LF will stimulate more significant changes to the antioxidant levels[11].

We measured the concentrations of superoxide dismutase (SOD), glutathione peroxidase (GPOD), catalase, and total antioxidative status (TAS).

TABLE 1
PATIENT'S GENDER, AGE, DURATION AND TYPE OF DIABETES

	Gender (F/M)	Age (years)	Diabetes duration (years)	Type (1/2)
Patients with DR (n=30)	16/14	54.8±4.80	12.7±2.0	15/15
Patients without DR (n=30)	15/15	51.4±7.76	4.8±2.5	13/17
Individuals without DM (n=30)	7/23	50.3±6.46		

DR = diabetic retinopathy
DM = diabetes mellitus

The catalytic concentrations of SOD and GPOD were determined from the erythrocyte lysate using specific assays (Randox Laboratories LTD, Andmore, GB). The reference values were provided by Randox Laboratories: SOD, 1102–1601 U/gHb; and GPOD, 27,5–73,6 U/gHb.

The catalytic concentration of catalase was also determined from the erythrocyte lysate using a specific assay produced by Oxis International, Portland, USA. Because this was a non-commercial assay, the firm does not provide reference values.

TAS was measured in the plasma with an assay produced by Randox Laboratories LTD, Andmore, GB, using the Olympus AU2700 machine[38]. The reference values were provided by Randox Laboratories: 1.30–1.77 mmol/L.

Statistical analysis of the data was conducted using Excel 5.0, Microsoft, USA. The significance of the difference between the mean values of the independent variables was evaluated by the Student's t-test for independent samples. The significance of the difference between the dependent variables was evaluated with the Students's t-test for dependent samples. The analysis of variance (ANOVA) with one changeable factor was used to test the significance of the difference between more than two samples. The chi-square test was used to determine the difference between nominal variables. Significance levels were set at 0.05.

Results

Of the 30 patients with DR, 13 (43.3%) showed clinical signs of macular edema, and 17 (56.7%) did not. In these 13 patients, macular edema appeared or worsened after LF, whereas in the other 17 patients, there was no change. Clinically significant macular edema (CSME) was evaluated during the first or second week after LF. Of the 13 macular edema patients, 12 showed new or worsening CSME, whereas 1 patient showed worsening of manifest macular edema. The patients with manifest macular edema were previously treated with green LF"? Please clarify. All patients with new or worsening macular edema had significantly lower antioxidant levels before LF ($p<0.001$) (Table 2).

The mean visual acuity in group with diabetic retinopathy before LF was 0.47. One day after LF, it was significantly lower at 0.40 ($p<0.001$), as expected, and 7 days after LF, it was also significantly lower at 0.41 ($p=0.002$). Thirty days after LF, improvement in visual acuity was observed (0.48), but this improvement was not statistically significant ($p=0.448$) (Table 3).

TABLE 2
INCIDENCE OF MACULAR EDEMA IN PATIENTS WITH DIABETIC RETINOPATHY

	Before LF (n/%)	After LF (n/%)
Without macular edema	17/56.7	
Macular edema	13/43.3	
Development		12/40
Worsening of macular edema		1/3.3
Unchanged		17/56.7

LF=laser photocoagulation
$\chi^2=21.855$ df=2 $p<0.001$

The mean concentrations of various antioxidants in the plasma or erythrocyte lysate (SOD, GPOD, catalase, and TAS) were significantly lower in the patients without DR compared to the individuals without DM.

The mean concentrations of various antioxidants in the plasma or erythrocyte lysate (SOD, GPOD, catalase, and TAS) were significantly lower in patients with DR compared to the individuals without DM.

The mean concentrations of various antioxidants in the plasma or erythrocyte lysate (SOD, GPOD, catalase, and TAS) were lower in the patients with DR compared to the patients without DR; however, this difference was not statistically significant (Table 4).

The mean concentrations of various antioxidants in the plasma or erythrocyte lysate (SOD, GPOD, catalase, and TAS) after LF were significantly lower compared to before LF (Table 5).

Discussion

LF is undoubtedly an efficacious way of treating DR, and it lowers the risk of significant loss of visual acuity. However, the procedure can also be destructive and unsuccessful because of its side effects, and it may induce or worsen macular edema with consequent temporary or permanent visual loss.

TABLE 3
VISUAL ACUITY IN PATIENTS WITH DIABETIC RETINOPATHY BEFORE AND AFTER RETINAL LF

	VA before LF	VA 1 day after	VA 7 days after	VA 30 days after
Patients with DR	30	30	30	30
Mean value±SD	0.47±0.2	0.40±0.2	0.41±0.2	0.48±0.2
*p	p<0.001	p<0.002	p=0.448	

LF = laser fotokoagulation
DR = diabetic retinopathy
VA = visual acuity
*ANOVA test

TABLE 4
SERUM COCENTRATIONS OF SUPEROXIDE DISMUTASE (SOD), GLUTATION PEROKSIDASE (GPOD), CATALASE AND TAS IN ALL GROUPS

	SOD (U/gHb)	GPOD (U/gHb)	Catalase (Ku/L)	TAS (mmol/L)
Patients with retinopathy (n=30)	869.9±175.4[a,b]	40.7±9.7[d,e]	60.8±23.5[g,h]	1.05±0.09[j,k]
Patients without retinopathy (n=30)	900.7±179.1[c]	42.8±9.1[f]	64.2±19.4[i]	1.05±0.09[l]
Individuals without diabetes (n=30)	1327±175.6	49.6±13.9	80.0±13.6	1.43±0.12

Values are expressed as mean values±SD
[a] p=0.451 when comparing group 1 and 2, [b] p<0.001 when comparing group 1 and 3, [c] p<0.001 when comparing group 2 and 3, [d] p=0.375 when comparing group 1 and 2, [e] p<0.01 when comparing group 1 and 3, [f] p<0.05 when comparing group 2 and 3, [g] p=0.546 when comparing group 1 and 2, [h] p<0.001 when comparing group 1 and 3, [i] p<0.001 when comparing group 2 and 3, [j] p=0.848 when comparing group 1 and 2, [k] p<0.001 when comparing group 1 and 3, [l] p<0.001 when comparing group 2 and 3.

TABLE 5
SERUM COCENTRATIONS OF SUPEROXIDE DISMUTASE (SOD), GLUTATION PEROKSIDASE (GPOD), CATALASE AND TAS IN PATIENTS WITH DIABETIC RETINOPATHY BEFORE AND AFTER LF

	SOD (U/gHb)	GPOD (U/gHb)	Katalaza (Ku/L)	TAS (mmol/L)
Before LF (n=30)	865.9±175.4	40.7±9.7	60.8±23.5	1.05±0.09
After LF (n=30)	801.5±146.5	34.4±8.9	50.1±21.6	0.98±0.08
	p<0.001	p<0.001	p<0.001	p<0.001

LF = laser fotocoagulation
Student's t-test for dependent samples

The thermal effect of scatter LF is one of the factors that contributes to the development or worsening of macular edema. The breakdown of the hematoocular barrier is a possible consequence of the oxidative stress that is caused by the thermal effect of LF on diabetic retinas and their compromised antioxidative defenses[11,39].

Weakened antioxidative defenses in diabetic retinas (in humans and animals) have been found in many studies[40–45].

In our study, diabetic patients without DR also showed significantly lower concentrations of antioxidative agents when compared to individuals without DM. Although similar results have been reported by some authors[46–48], others have reported opposite results[49–52]. The concentrations of antioxidative agents in diabetic patients with DR were lower than in diabetic patients without DR, although this difference was not statistically significant. According to other similar reports, our study suggests that lower concentrations of antioxidative substances in diabetic patients favor the development of oxidative stress, which is an important factor in the development of DR and is also a prerequisite for the worsening of macular edema after LF.

Numerous authors have described the development and worsening of macular edema as a complication of scatter LF[13,14,40,53–55], but there are very few publications that address the causes of macular edema, especially the role of oxidative stress in those causes.

The aim of our study was to compare the antioxidant concentrations in diabetic patients with severe NPDR before and 2 hours after full scatter retinal LF. Our results show a significant decrease in the SOD, GPOD, catalase, and TAS concentrations after LF. We propose that the decrease in the concentrations of these antioxidants in the plasma and erythrocyte lysate after retinal LF is the result of the activation of antioxidative mechanisms, which neutralize the ROS and FR that are produced by the thermal effect of LF on the retina (Figure 1).

As previously reported by other authors, retinal LF significantly induces the development or worsening of macular edema[13,14].

We also observed deterioration in the mean visual acuity during the first few weeks after LF with a subsequent improvement one month after the therapy; however, this improvement was not statistically significant. Although the results from other studies vary widely, our results are similar to some of those studies[15,56,57].

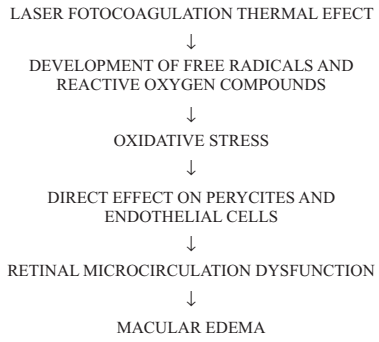

Fig. 1. Patophysiology of macular edema development after retinal laser fotocoagulation.

The results of our study may suggest that oxidative stress is a significant factor in the development of macular edema after scatter LF of the retina in severe NPDR.

Conclusions

The concentrations of antioxidative compounds in diabetic patients are significantly lower than in healthy patients. This result supports the theory of weakened antioxidant defenses in diabetic patients.

The concentrations of antioxidative compounds in diabetic patients with DR are lower than in diabetic patients without DR; however, the difference is not significant.

Macular edema is an important complication of scatter LF.

The concentrations of SOD, GPOD, catalase, and TAS are significantly lower in diabetic patients with DR after retinal scatter LF. This could be a consequence of the retinal oxidative stress caused by the thermal effect of LF, and ME may be a significant complication of this effect.

It may be possible to predict the increased risk of retinal edema that complicates the procedure by assessing the antioxidant reserve of patients.

In addition, dividing the LF treatment into multiple sessions with fewer, smaller, and lower intensity LF spots could also be useful.

The findings in our study suggest that the administration of stable and simple antioxidants, such as vitamin C and E, may help to overcome the oxidative stress caused by retinal LF in susceptible patients[58–60].

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ULOGA OKSIDATIVNOG STRESA NAKON FOTOKOAGULACIJE MREŽNICE KOD NEPROLIFERACIJSKE DIJABETIČKE RETINOPATIJE

S A Ž E T A K

Diabetična retinopatija predstavlja najčešću kroničnu komplikaciju dijabetesa. Ona je najčešći uzrok novih slučajeva sljepoće kod bolesnika između 20–74 godina starosti u razvijenim zemljama. Laserska fotokoagulacija predstavlja jedini efikasan način liječenja dijabetične retinopatije. Oksidativni faktori kao slobodni radikali se neprestano stvaraju u organizmu kao rezultat različitih metaboličkih procesa. Cilj ovog istraživanja je istražiti ulogu oksidativnog stresa na nastanak i razvoj dijabetične retinopatije. U isto vrijeme smo istražili utjecaj toplinskog efekta na stvaranje slobodnih radikala za vrijeme »scatter« laserske fotokoagulacije mrežnice. U ovu studiju je bilo uključeno 90 bolesnika. Bili su podijeljeni u 3 različite grupe; grupa 1 se sastojala od 30 bolesnika s dijabetičnom retinopatijom; grupa 2 se sastojala od 30 bolesnika bez dijabetične retinopatije; grupa 3 se satojala od 30 zdravih ljudi. Kod svih bolesnika iz grupe 1 učinjena je »full scatter« laserska fotokoagulacija mrežnice. Mjerili smo koncentracije superoxid dismutaze (SOD), glutation peroxidaze (GPOD), katalaze i ukupni antioksidativni status (TAS). Kod 13 bolesnika se pojavio ili pogoršao makularni edem nakon fotokoagulacije mrežnice, dok se kod 17 bolesnika nije zabilježila nikakva promjena. Zabilježeno je poboljšanje vidne oštrine trideset dana nakon fotokoagulacije, no razlika se nije pokazala statistički značajnom. Srednja vrijednost koncentracije raznih traženih antioksidansa u plazmi ili u lizatu eritrocita je bio značajno manji u grupi 2 nego u grupi 3, zatim u grupi 1 nego u grupi 3, no ta se razlika nije pokazala značajnom između grupe 2 i grupe 1. Rezultati su pokazali da su koncentracije superoxid dismutaze, glutation peroxidaze, katalaze te ukupni antioksidativni status bili značajno niži u grupi dijabetičara s dijabetičnom retinopatijom nakon laserske fotokoagulacije retine, što bi moglo biti posljedica oksidativnog stressa mrežnice uzrokovanog teramalnim učinkom retinalne fotokoagulacije. To znači da bismo davanjem jednostavnih i sigurnih antioksidansa kao što su to vitamin C i E mogli olakšati organizmu da brže prebrodi stanje oksidativnog stresa.