LASER PHOTOCOAGULATION IN DIABETIC RETINOPATHY

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Keywords: *diabetic retinopathy, diabetic maculopathy, macular edema, laser photocoagulation*

Abstract: *Diabetes mellitus is the most common endocrine disease. Diabetic retinopathy is a complication of diabetes and is the leading cause of blindness in the age segment of 20-50 years. We present theoretical and practical aspects related to diabetic retinopathy and treatment by laser photocoagulation.*

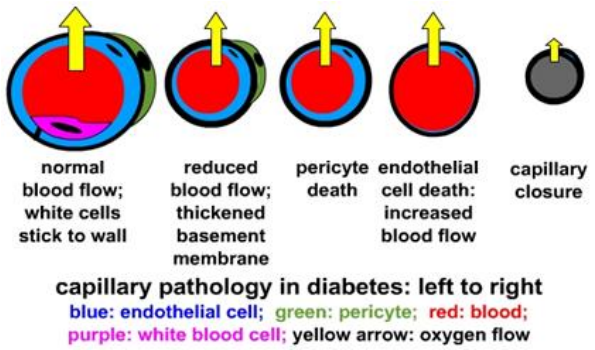
Cuvinte cheie: *retinopatie diabetică, maculopatie diabetică, edem macular, fotocoagulare laser*

Rezumat: *Diabetul zaharat este cea mai frecventă boală endocrină. Retinopatia diabetică reprezintă o complicație a diabetului, fiind principala cauză de orbire în segmentul de vârstă 20-50 ani. Se prezintă aspecte teoretice și practice legate de retinopatia diabetică și tratamentul prin fotocoagulare laser.*

INTRODUCTION

Diabetic retinopathy (DR) is one of the many complications of diabetes and is a microangiopathy affecting the arterioles, capillaries and venules of the retina. On average, it occurs about 10 years after the development of diabetes. Injuries found in RD take place in all the structural components of capillaries: Endothelial cells – they show an increase in volume with intercellular junctions destruction; basement membrane - it thickens with vacuolation and fat and cell debris infiltration; pericytes - there is a progressive decline in their number (figure no. 1).

Figure no. 1. Capillaries changes in DR (http://www.diabeticretinopathy.org.uk/diabetic_retinopathy_mech.html)



Lesions in retinal capillaries cause two types of changes: anatomical and functional. Microaneurysms are saccular dilatation of the vessel wall, which occur mostly on the venous side of the capillary network, with spherical or fusiform shape (figure no. 2). Alteration of the blood-retinal barrier causes extravasation of plasma and blood elements leading to bleeding and retinal edema. Haemorrhages are clumps of red blood cells in areas of nerve cells that stimulate retinal scarring consisting in collagen and pigment ferric (figure no. 3). Retinal edema is a transudate accumulating in the outer plexiform layer (figure no. 4).

Figure no. 2. Microaneurysm – angiofluorographic image

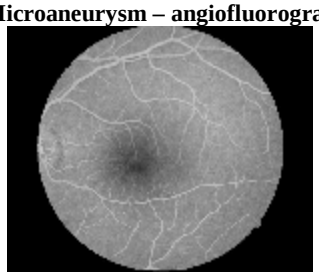
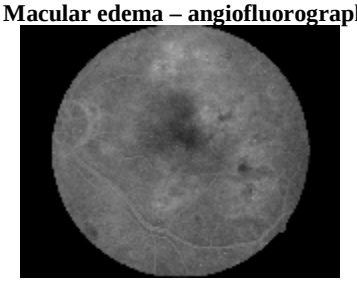


Figure no. 3. Retinal microhaemorrhages

Figure no. 4. Macular edema – angiofluorographic image



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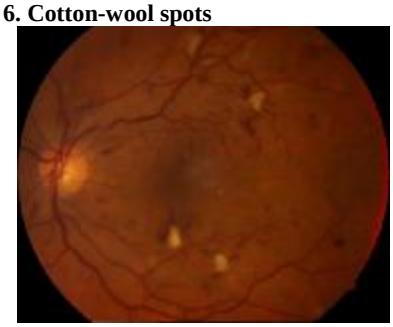
Exudates are another consequence of structural alterations of capillaries and are of two kinds:

- hard exudates are located deeply in the external plexiform layer; clinically, they appear as bright, yellowish-white spots, of vascular origin and are plasmatic suffusions crossing the wall microaneurysms (figure no. 5).
- cotton-wool spots are not specific to RD; they can be found in other diseases: hypertensive retinopathy, vein occlusion, diabetic nephropathy; clinically, they appear as white nodules with hazy margins are located in the superficial fibre layer; they are considered cytoplasmic residues, a result of axoplasmatic flow interruption caused by ischemia (figure no. 6).

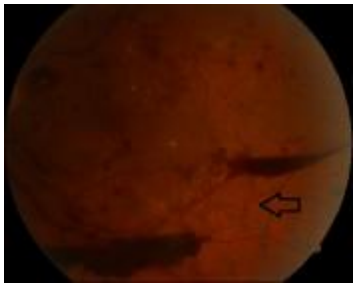
Figure no. 5. Hard exudates

Figure no. 6. Cotton-wool spots



In DR, together with the capillary wall lesions, hemorheologic changes also occur: there are red blood cells alterations with the decrease in oxygen transport and platelets alterations with the increase of platelet aggregation. These changes cause capillary occlusion, which in turn causes retinal ischemia that ultimately leads to hypoxia. The consequence of retinal hypoxia is retinal neovascularisation. It is believed to be caused by vascular growth factors produced by the hypoxic retinal tissue in order to reperfuse those areas (figure no.7).

Figure no. 7. Neovascularisation at disc



Diabetic retinopathy classification

Diabetic retinopathy is divided into two categories: non-proliferative diabetic retinopathy (NPDR) and proliferative diabetic retinopathy (PDR). In turn, NPDR is subdivided into mild, moderate and severe:

- Mild NPDR is characterized by the presence of at least one microaneurysm or microhemorrhage
- Moderate NPDR is characterized by the presence of at least one of the following changes: microaneurysms / hemorrhages, exudates, venous dilatation or intraretinal microvascular abnormalities (IRMA)
- Severe NPDR submit the 4-2-1 rule: microaneurysms / hemorrhages in all four quadrants, retinal or dilated veins in at least two quadrants, or IRMA in at least one quadrant
- PDR is characterized by the appearance of neovascularisation at optic disc (NVD) or elsewhere (NVE), and in more advanced stages occur vitreous hemorrhage, proliferative membranes and finally tractional retinal detachment.

Laser photocoagulation

Laser photocoagulation addresses severe NPDR and PDR before vitous haemorrhage, proliferative membranes or retinal detachment occur, which receive surgical treatment.

Laser photocoagulation can be performed in the central retina for macular edema as focal or grid photocoagulation, and the periphery as panphotocoagulation. When laser treatment should be applied in both macular region and periphery, it starts with macular region because panphotocoagulation can cause a temporary increase of macular edema.

For laser treatment, the patient signs an informed consent. Mydriatic drops are instilled about 30 minutes before and anesthetics drops about 10 minutes before. It is recommended that physicians have an angiofluorographic image of the retina / macular region.

For macular edema photocoagulation, the laser is set for 50-100 micrometres sport size, 50-100 ms duration and an intensity of 80 mW, which can be increased in 10 mW increments as needed. A contact lens (Yannuzzi, Mainster etc.) is placed on eye and the macular region is identified by comparing to angiogram image. The patient is asked to look at the fixation point with the fellow eye and not to stare into laser beam. Treatment involves focal photocoagulation of the microaneurysms ranging from 500-3000 μm from fovea (figure no. 8). Lesions closest to fovea are treated first, then the farthest, aiming to obtain grey-white burns in the retina. Grid photocoagulation in performed in case of diffuse retinal edema located over 500 micrometers from fovea or temporal edge of the optic nerve head (figure no. 9). If the patient requires both focal and grid photocoagulation, focal photocoagulation is preferred first, then the grid one. Care is taken when moving from more edematous areas to areas less so, because the latter require less power for effective burns. In addition, for better results of the laser treatment, it is preferred that macular edema is under 400μ. Areas of intraretinal hemorrhage are not treated directly but can be surrounded. After treatment, the contact lens is removed and antibiotic drops are instilled. The patient is asked to return in about three months.

Figure no. 8. Focal laser photocoagulation

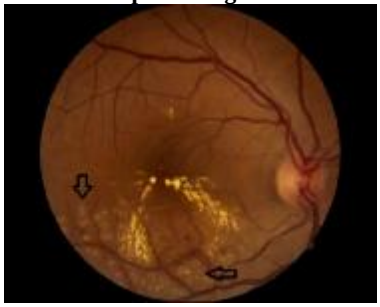
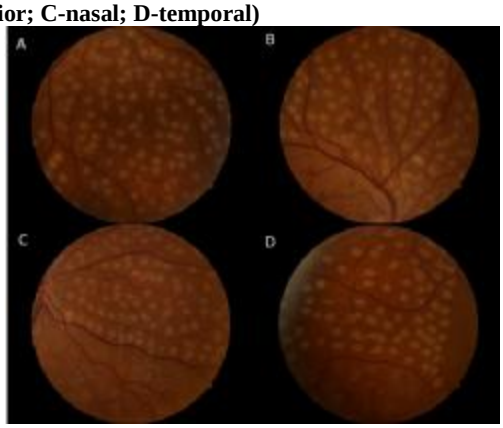


Figure no. 9. Grid laser photocoagulation

For panphotocoagulation, the laser is set at 500 micrometers spot size for Goldmann lens, or 200-300 μm for panfundoscope lens (Mainster wide-field, Volk QuadrAspheric, etc.), 100-200 ms duration and an intensity of 100 - 200 mW, which can be increased in 10 mW increments. The contact lens is placed on eye and the initial burns are placed in a double arc approximately 3 disc diameters temporal to the centre of the macula as a visual barrier to prevent accidental burn to the fovea. A total of 1500-2000 spots are placed divided into several sessions depending on patient compliance (figure no. 10). The area nasally to the optic disc or inferior retina is treated first and then the other areas with burns spaced one burn apart. It starts near the vascular arcades and continue to the periphery. Photocoagulation goes up to, but not over, areas of vitreo-retinal traction. NVD is not treated directly. After treatment, the contact lens is removed and antibiotic and cycloplegic drops are instilled for a few days. The patient is asked to return in about one to three months.

Figure no. 10. Laser panphotocoagulation (A-inferior; B-superior; C-nasal; D-temporal)



CASE REPORT

C.D., a-53-yearsold female presents for check-up.

Past medical history:

- Type 2 diabetes mellitus treated with insulin since 1999 (glycaemia between 70-220 mg/dl, last HBA1c=8.5%)
- Hypertension

Ophthalmologic exam:

- Best corrected visual acuity (BCVA) right eye (RE) =0.6; LE=0.8
- Intraocular pressure (IOP) RE = 24mmHg; Left eye (LE) =24mmHg
- Anterior segment: normal
- Fundus examination: optic disc normal, macula normal, many mycroaneurisms and hemorrhages disseminated throughout, few hard exudates RE>LE (figures no. 11,12).

Additional exams:

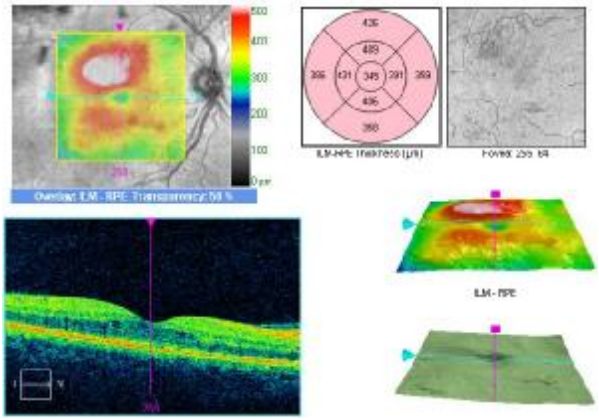
- Visual field: unorganised relative scotomas
- Optical coherence tomography (OCT): macular edema, optic disc and retinal nervous fibre layer normal (figures no. 13,14).

Diagnosis: Severe NPDR, Diabetic maculopathy with macular edema, Miopya, Astigmatism, Ocular Hypertension (IOHT).

Figure no. 11. Retinal image RE

Figure no. Retinal image LE

Figure no. 13. OCT RE

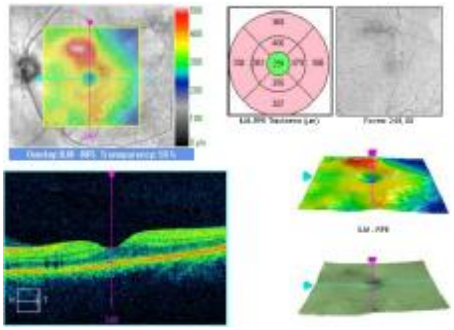


Differential diagnosis:

- DR: central retinal vein occlusion, anterior ischaemic optic neuropathy, carotid stenosis, macroaneurysm, hemoglobinopathies, orbital venous hypertension syndrome.
- Diabetic maculopathy with maculer edema: cystoid macular edema, branch retinal vein occlusion, macular pucker, Irvine-Gass syndrome, wet age-related macular degeneration (AMD).
- IOHT: primary-open-angle glaucoma (POAG), primary angle-closure glaucoma (PCAG), neovascular glaucoma (NVG).

Without treatment, NPDR gets worse and progresses toward PDR by the appearance of neovascularisation as a response to prolonged hypoxia. These abnormal blood vessels may bleed, causing vitreous hemorrhage. The development of the neovascularisation in iridocorneal angle is responsible for secondary neovascular glaucoma. In more advanced stages, vitreo-retinal membranes develop causing retinal traction and finally tractional retinal detachment.

Figure no. 14. OCT LE



DR treatment may be medical, physical (laser photocoagulation) or surgical. In our case, severe NPDR imposed treatment by laser photocoagulation. Focal laser photocoagulation was initially performed in macular region (figures no. 15,16), then panphotocoagulation in three sessions. The patient benefited from intravitreal anti-VEGF agent injection to OD, too.

Three months after completing the laser treatment, visual acuity (VA) slightly improved (RE=0.8, LE=0.8), IOP remained above the threshold, so ocular hypotensive therapy was initiated, and the fundus examination revealed regression of retinal hemorrhage (figures no. 17,18), and macular edema (figures no. 19,20).

Figure no. 15. Focal laser photocoagulation RE

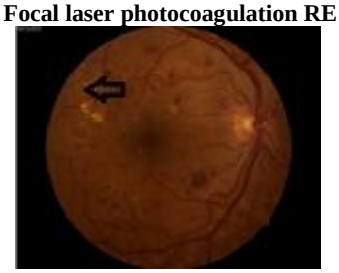


Figure no. 16. Focal laser photocoagulation LE

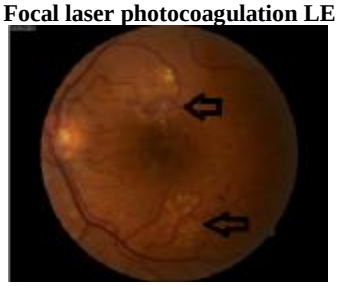
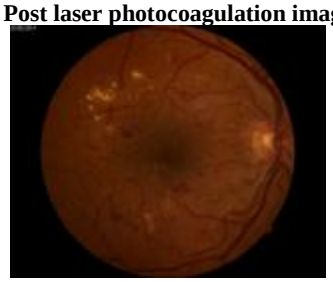


Figure no. 17. Post laser photocoagulation image RE



An important point to remember is that laser photocoagulation is a final therapy solution that practically destroys retinal tissue and is performed when medical treatment fails and the purpose is primarily to preserve and not to improve the visual function.

Figure no. 18. Post laser photocoagulation image LE

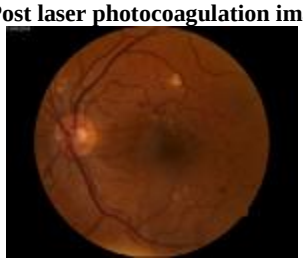


Figure no. 19. Post laser treatment OCT image RE

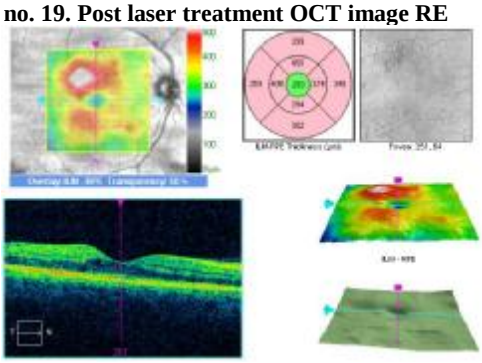
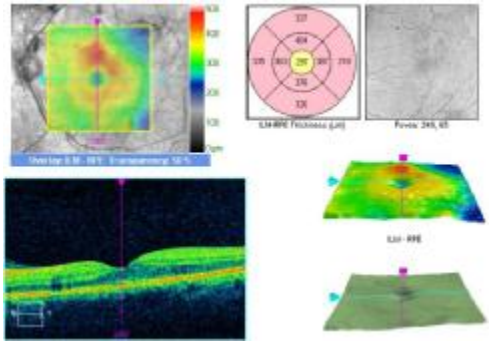


Figure no. 20. Post laser treatment OCT image LE



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