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Failure of Laser Therapy in the Course of Proliferative Diabetic Retinopathy with Coexisting Scleroderma - Case Report

Bow. Aneta Harasimiuk, MD*

Corresponding Author: Bow. Aneta Harasimiuk, MD, Bydgoszcz Center for Diabetology and Endocrinology, Ul. K. K. Baczyński 1785-822 Bydgoszcz.

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Abstract

I describe the case of a 69-year-old man treated in Bydgoszcz Diabetes Center since 1995 due to type 2 diabetes. Diabetic proliferative retinopathy in both eyes developed in 2005 (after 10 years of diagnosing diabetes). Panphotocoagulation of the retina of both eyes was performed. In 2006, at the Department of Dermatology of the University of Dr. A. Jurasza in Bydgoszcz, scleroderma was diagnosed.

Scleroderma edema / scleredema diabeticorum / is a disease entity that occurs in the course of diabetes / 2.5-3.0% cases /. This is usually the case with poorly balanced diabetes. The skin is hardened and swollen on the face, shoulders, upper torso and limbs. Joint movements are impaired in the affected area. The histopathological picture corresponds to the excessive growth of fibrous connective tissue and the accumulation of mucopolysaccharides in the skin.

In our patient, the skin lesions were located on the back, nape and back of the neck.

Despite the laser therapy performed, after 4 years there was a vitreous hemorrhage of the right eye and neovascularization appeared on the retina in the left eye. Retinal panphotocoagulation of both eyes was performed again. Then, in 2010, he was diagnosed with nuclear cataract and secondary glaucoma in both eyes. Cosopt 2x p / l was turned on. In 2011, cataract surgery was performed in both eyes with an artificial lens implanted into the posterior chamber. In February 2012, in the right eye with visual acuity Vod = 0.1 Tod = 20mmHg, iris rubeosis was found, the disk-to-cavity ratio c / d = 0.9, visual acuity in the left eye Vos = 0.8 tos = 19mmHg, c / d = 0.5 without neovascularization volleyball. Complementary panphotocoagulation and anti-glaucoma treatment did not bring the expected effect. My visual acuity has dropped to a sense of light. The aim of the study is to show that despite the use of laser therapy and constant ophthalmic control, decompensated diabetes leads to blindness.

Keywords

Proliferative diabetic retinopathy, scleroderma, retinal panphotocoagulation, neovascular glaucoma, type 2 diabetes mellitus.

Introduction

The aim of the study is to present the case of a 69-year-old patient treated for 17 years due to type 2 diabetes mellitus with diagnosed scleroderma.

Scleroderma edema / Latin cleredema diabeticorum / was first described in 1900 by Buschke. It is an uncommon disease of connective tissue affecting the upper back, shoulders and neck, and rarely affects the face, arms and chest. Internal organs may occasionally be involved, but never the hands and feet. Scleroderma is characterized by thickening, induration, and an absence of pain in the affected skin. Acute cases of scleroderma may be accompanied by respiratory disturbances.

Buschke distinguishes 3 types of scleroderma:

1/1 type appears after acute inflammation caused most often by streptoccocus - symptoms of scleroderma disappear after a few months.

2/2 type - its appearance is not related to diabetes or infection

3/3 type associated with long-lasting type 1 or 2 diabetes mellitus, poorly controlled, patients are treated with insulin, have complications in the form of retinopathy and nephropathy. This type is called scleredema diabeticorum / scleroderma / or scleredema adultorum of Buschke.

The pathogenesis of this disease is unknown. It is presumed that the non-enzymatic glycosylation of collagen fibers leads to its degradation. Another hypothesis suggests that hyperglycemia stimulates fibroblast proliferation and an increase in mucopolysaccharides in the matrix.

In scleroderma edema, the following treatment is used: antibiotics / penicillin /, corticosteroids, chemotherapy, radiation, tamoxifen, strict glucose control, PUVA and UV-A1 therapy / photochemotherapy- skin irradiation with UVA radiation after prior administration of photosensitizing drugs /. The best results are obtained with the last 3 therapies.

Description of the patient

Patient, 69 years old, treated in Bydgoszcz Diabetes Center since 1995 due to type 2 diabetes.

The results of an ophthalmological examination in 1995

Vod = cc + 2.0 = 1.0 Tod = 17.3 mmHg

Vos = cc + 2.5 = 1.0 Tos = 17.3mmHg

Anterior section p / l - correct

Bottom: Discs n. II pink, with distinct borders, flat. Arterial vessels narrowed, venous dilated.

Diabetes test results:

-weight: 103kg, RR 140/85, fasting glucose 149 mg / dl, urine test normal

-Treatment:

Glibenase 3x1 tabl

Metformax3x1 tabl

Insulin Mixtard 30 26j-16j-16j

Nootropil 1-1-0

In 2005, after 10 years of diagnosing diabetes, hypertension and significant obesity / 110 kg / were diagnosed. Treatment used: Humalog 50 24j-0-26j, Humalog 100 0-24j-0, Metformax 850mg 1-0-1, Diaprel MR 1-0-0, Amlopin 5mg 1-0-0, Acard 75mg 1-0-1 , Furosemide 1-0-0, Enarenal 10mg 1-0-1.

There were also symptoms of proliferative diabetic retinopathy in both eyes with visual acuity of Vou = 1.0ccwl, tou = 17.3mmHg. Retinal panphotocoagulation / PPK / was performed in both eyes with a 532 nm green laser.

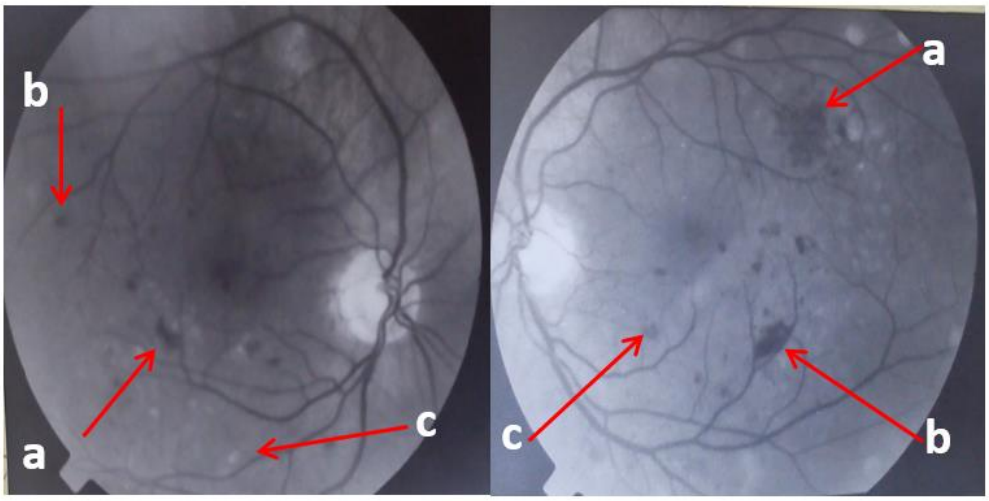

Fig. 1 Image of fluorescein angiography of the fundus of the right and left, analogue image.

OP- a / IRMA visible on the lower vascular arch, b / macular hemorrhages, c / cotton wool foci

OL- a / NVE below the upper vascular arch b / IRMA, c / macular hemorrhages

In 2006, at the Department of Dermatology of the University of Dr. A. Jurasza in Bydgoszcz, a patient was diagnosed with scleroderma. The diagnosis was made on the basis of the clinical picture and the result of the histopathological examination of the skin section. The patient was treated with procaine penicillin at a dose of 2,400,000 J a day for 30 days and Balneo-PUVA, neck massage, 10% salicylic ointment, solux, lyotone 1000, which resulted in a significant improvement of the local condition.

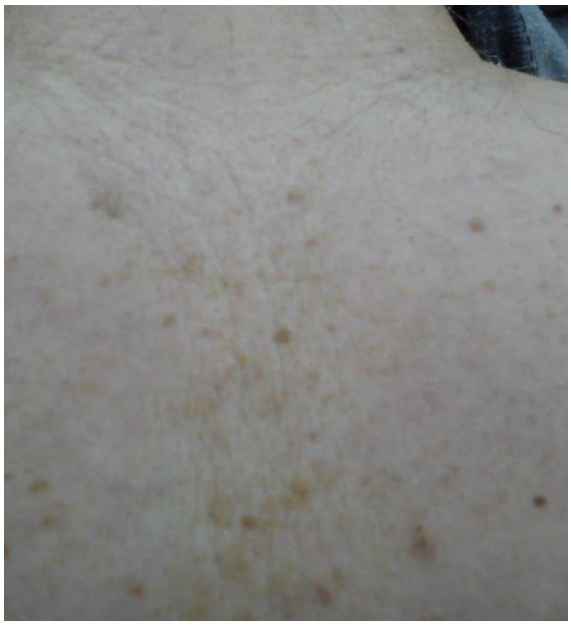

Fig. 2 Macroscopic image of the skin on the patient's back - scleroderma

In 2009, there was a sudden deterioration of vision in the right eye, Vod = rrpo, tod = 17mmHg, caused by massive vitreous hemorrhage. NVE / neovascularization around the periphery of the retina / with Vos = 0.63 visual acuity, tos = 18mmHg was noted in the left eye. Complementary retinal panphotocoagulation / PPK / in 628 foci, 0.1s, 300 focal diameter, 280mW power, in OL-347 foci, 0.1s, 300 micrometers focal diameter, 250mW power using a 532nm green laser.

In 2010, he was diagnosed with nuclear cataract and secondary glaucoma in both eyes. Cosopt 2x p / l was turned on. In 2011, cataract surgery was performed in both eyes with an artificial lens implanted into the posterior chamber. The risk of secondary neovascular glaucoma increases after cataract removal.

In February 2012, in the right eye with visual acuity Vod = 0.1, iris rubeosis was found Tod = 20 mmHg, c / d = 0.9. In the left eye, Vos = 0.8 Tos = 19mmHg, the fundus image after PPK was stable, without neovascularization. Complementary panphotocoagulation / 680 foci, 0.1s, 200 micrometers, 280 mW were again performed in the right eye. During laser therapy, the intraocular pressure increased to 30mmHg. In addition to Cosoptu 2x p / l, Diuramid 250 mg 2x1 was administered for 2 weeks. 1% Atropine, Maxitrol 4x was also administered to the right eye.

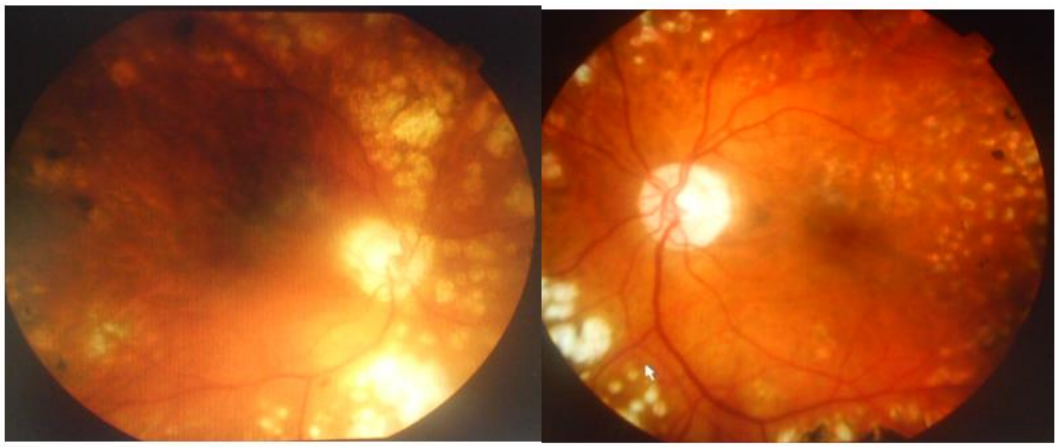

Fig. 3 and 4. Color fundus pictures. In both eyes visible scars after retinal panphotocoagulation, without neovascularization and macular edema.

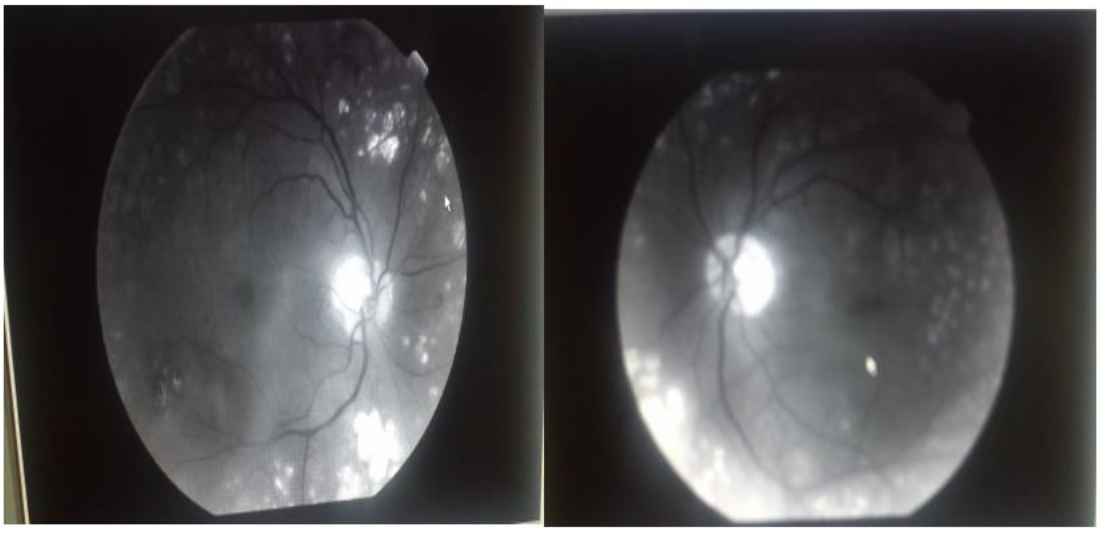

Fig 5 and 6 Fluorescein angiography images showing laser scarring in both eyes

The diabetic study found: weight 112 kg, RR 139/84, fasting glucose 270 mg / dl, HbA1c 9.0%. Treatment as above.

The diabetologist who has been treating the patient since 1995 was consulted. It was found that the patient was undisciplined, had no glycemic control, and missed visits to a diabetologist.

Conclusions

The aim of the presentation below is to show the destructive influence of "decompensated diabetes" on the organ of vision. Persistent, unbalanced blood glucose levels may result in irreversible loss of vision despite the use of comprehensive laser therapy and regular ophthalmic control. We can assume that with properly managed diabetes there is a greater probability that the treatment effect would be better. The disruptive effect of diabetes resulted in a worse prognosis and the treatment effect ineffective.

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