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*Bilateral Purtscher's retinopathy in a patient
with acute pancreatitis*

Purtscher's retinopathy is characterized by acute, usually bilateral ischemia of the central part of the retina, accompanied by considerable decrease of visual acuity and visual field changes. This retinopathy was first described in 1910 by Otmar Purtscher in a patient after severe head injury (4).

It occurs after sustained extensive body injuries, usually compressive injuries of the chest and head (4, 5). The condition can also accompany other non-injurious pathologies, e.g. acute pancreatitis, pancreatic cancer, lymphoma and renal failure, disease of connective tissue and can also occur in patients after medullar grafting (4, 5). The pathogenesis of this disease is not fully known yet. In patients with Purtscher's retinopathy an abrupt closure takes place of blood flow in pre-capillary arterioles supplying superficial network of capillaries around the disc of the optic nerve.

Embolic material in patients with acute pancreatitis can be of different kinds. Proteolytic enzymes: trypsin and amylase liberating themselves to systemic circulation lead to intravascular clotting and formation of fibrinous clots which can constitute embolic material (1). The latter can also be made up of leukocyte agglomerates which are formed as a result of complement activation (3). Fat embolisms seem to be hardly probable owing to too small diameter of fat particles (2).

Quite frequently, after 3–4 months of the disease spontaneous subsidence of changes on the eye fundus takes place as well as improvement of visual acuity. Final prognosis is uncertain because permanent decrease of vision may occur as a result of macular or optic nerve damage. Treatment is regarded as not fully efficient. General use of steroids is recommended as well as of drugs improving retinal blood flow, antiinflammatory and antiedematous ones.

CASE DESCRIPTION

The aim of the study is to present the case of a 20-year old male patient with bilateral Purtscher's retinopathy, which occurred during acute post-alcoholic pancreatitis. Decrease of visual acuity was observed by the patient on the third day of general illness. Two weeks from the onset of ocular ailments the patient reported at the 2nd Ophthalmology Department of the Medical University of Lublin.

On ophthalmologic examination the patient revealed: the visual acuity of the right eye V.o.d.= counts fingers close in front of the eye, sense and localization of light were normal; the visual acuity of the left eye V.o.s.= counts fingers from 0.5 m, sense and localization of light were normal; on the eye fundi visible slight blurring of disc borders of II nerve from the top and nose with delicate pallor from the temple, numerous white areas of ischemic retina called cotton-wool spots and localized symmetrically on the temporal side of the discs, hemorrhages and macular edema (Fig. 1, 2). Angiographic examination showed lack of capillary perfusion in the region of described fundal changes, hemorrhages and macular oedema (Fig. 3, 4). Our accessory lab investigations precluded as etiology of ocular changes infection with toxoplasmosis, boreliosis and HIV virus. Neurological examination did not reveal any anomalies. In ophthalmic treatment Solu-Medrol 1 g i.v. for 5 days was used with subsequent Encorton therapy for 10 days in the dose of

Medrol 1 g i.v. for 5 days was used with subsequent Encorton therapy for 10 days in the dose of 1 mg/kg body mass and then in decreasing doses for 1 month as well as Polfilin, Diuramid and Vessel Due F. After 10 days considerable improvement of visual acuity was observed and regression of fundal changes. V.o.d.=0.1 V.o.s.=0.2, visual field of both eyes possible then showed central and paracentral scotomas. After a month further improvement of vision was found. Right eye visual acuity was: V.o.d.=0.5 Sn.o.d=1.25 and acuity of the left eye: V.o.s.=0.8 Sn.o.s.=0.75. Further subsidence of fundal changes was going on during 4-month observation (Fig. 5, 6).

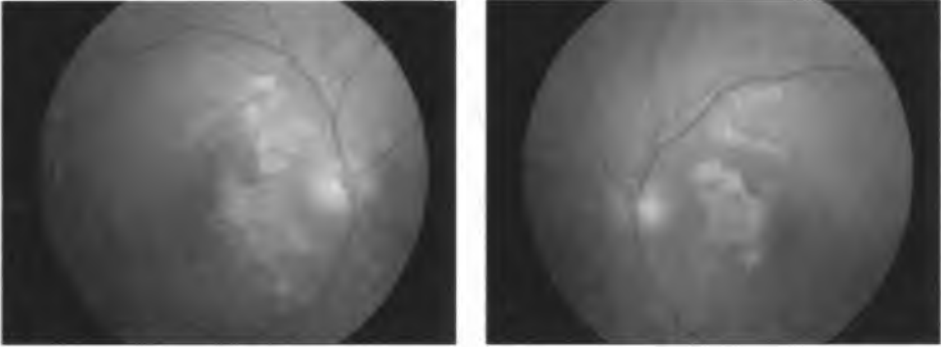


Fig. 1, 2. The right and the left eye fundus in the course of Purtscher's retinopathy in the described patient at the moment of starting the treatment

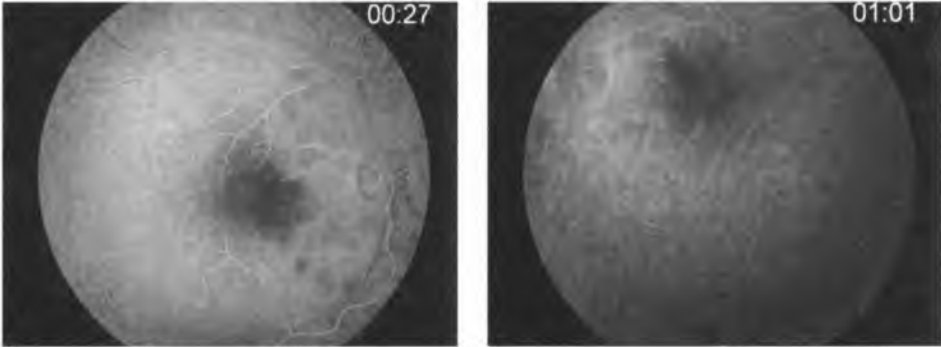
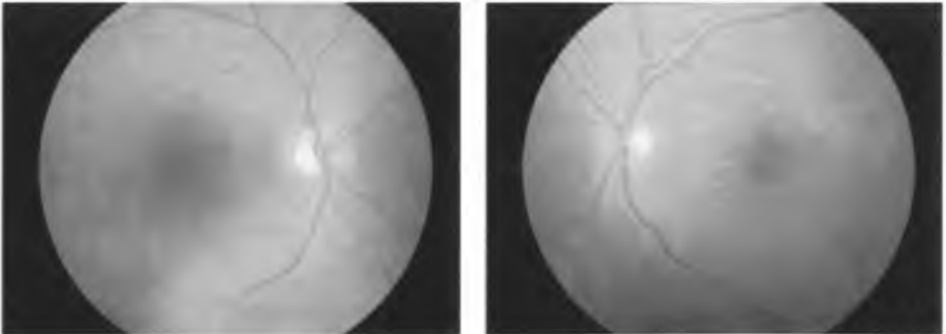


Fig. 3, 4. Fluorescein angiography of the right and the left eye in acute phase of Purtscher's retinopathy



DISCUSSION

Diagnosis of Purtscher's retinopathy in the described case was made basing on a typical case history and clinical picture and course of the disease. Purtscher's retinopathy should be differentiated with other posttraumatic retinal changes such as retinal concussion (Berlin's edema) and syndrome of abrupt increase of hydrostatic pressure (4). In patients with alcohol poisoning toxic damage of the optic nerve should also be considered as the cause of worsening of vision. Some infectious diseases, e.g. toxoplasmosis, boreliosis or HIV infection can also give ocular symptoms resembling Purtscher's retinopathy. In the above described patient ophthalmic treatment was considerably delayed, which could affect severity of ocular complications. It seems that the applied therapy remarkably influenced the speed and degree of vision improvement.

CONCLUSIONS

1. Purtscher's retinopathy should be considered one of diseases causing worsening of vision in people after extensive traumas or with some severe general diseases, especially with pancreatitis.
2. This retinopathy is a disease usually recognized late since in the initial stage patients, due to severe general condition, often do not report worsening of vision. Delay of treatment connected with this may affect the degree of retinal damage.
3. Patients after extensive traumas, with alcohol poisoning and suffering from the above mentioned general conditions, who report worsening of vision, should be absolutely examined by an ophthalmologist.

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SUMMARY

The aim of the study is to present the case of a 20-year-old male patient with bilateral Purtscher's retinopathy, which occurred during acute post-alcoholic pancreatitis. The patient observed decrease of visual acuity on the third day of general illness. On the fundi of the eyes were found: slight blurring of disc borders of nerve II from the top and nose with delicate pallor from the temple, numerous foci of ischemic retina called cotton-wool spots situated symmetrically on the temporal side of the discs, areas, hemorrhages and macular edema. Conclusions: Purtscher's retinopathy is usually diagnosed too late since at an early stage patients, due to severe general condition, do not report worsening of vision. Delayed treatment connected with this may affect the degree of retinal damage.

Obustronna retinopatia Purtschera u chorego z ostrym zapaleniem trzustki

Celem pracy jest przedstawienie przypadku 20-letniego chorego z obustronną retinopatią Purtschera, która wystąpiła w czasie ostrego poalkoholowego zapalenia trzustki. Obniżenie ostrości wzroku chory zaobserwował w trzecim dniu schorzenia ogólnego. Na dnie oczu stwierdzono: nieznaczne zatarcie granic tarcz n. II od góry i nosa z delikatnym zblednięciem od skroni, liczne białe ogniska niedokrwionej siatkówki typu „kłębków waty” położone symetrycznie po skroniowej stronie tarcz, krwotoki oraz obrzęk płamki. Retinopatia Purtschera jest schorzeniem najczęściej zbyt późno rozpoznawanym, gdyż w okresie początkowym choroby ze względu na ciężki stan ogólny nie zgłaszają pogorszenia widzenia. Związane z tym opóźnienie leczenia może mieć wpływ na stopień uszkodzenia siatkówki.