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CENTRAL RETINAL ARTERY OCCLUSION ASSOCIATED WITH COCAINE

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✧ This article contains a case of central retinal artery occlusion in a young man associated with cocaine abuse. Survey data, dynamic monitoring of the patient are presented in the article. Possible mechanisms of vascular pathology associated with stimulant drugs are described.

✧ ***Keywords:*** central retinal artery occlusion; cocaine; vasospasm.

НАРУШЕНИЕ КРОВООБРАЩЕНИЯ В СИСТЕМЕ ЦЕНТРАЛЬНОЙ АРТЕРИИ СЕТЧАТКИ НА ФОНЕ ПРИЁМА КОКАИНА

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✧ В статье описан случай нарушения кровообращения в системе центральной артерии сетчатки у молодого мужчины на фоне приёма кокаина. Представлены данные обследования и динамического наблюдения за пациентом. Описаны вероятные механизмы возникновения сосудистой патологии, обусловленной приёмом стимулирующих средств.

✧ ***Ключевые слова:*** окклюзия центральной артерии сетчатки; кокаин; вазоспазм.

INTRODUCTION

Retinal vascular occlusion is uncommon in young patients. It is usually caused by systemic diseases or in people with a genetic predisposition [1, 8, 9]. Vascular catastrophes may be caused by the use of stimulating drug preparations, primarily cocaine, which ranks second after cannabis among drugs forbidden from use and distribution [18].

Cocaine addiction causes irreparable harm to the physical and mental health of society. Cocaine has a powerful stimulating effect on the central nervous system, causing a feeling of euphoria, a surge of mental and physical energy, increased libido, etc., for which people are getting addicted to it. A payment for the pharmacological stimulation of the body is the numerous destructive effects of this substance on the body, including the organ of vision in both acute and chronic exposure.

The article presents description of a serious ocular complication associated with cocaine use.

DESCRIPTION OF A CLINICAL CASE

Patient S., 32 years old, was admitted as an emergency case to the St. Petersburg City Multi-Field Hospital No. 2 in March 2018 with complaints of a rapid decrease in visual acuity of his right eye for the last 5 days, which arose in the situation of psychoemotional stress. The medical case history revealed that the patient had noted an episode of a pronounced 6-hour visual acuity impairment in the right eye 2 months before the hospitalization, which resolved unaided. Ophthalmological examination accomplished then (although after the regression of symptoms) revealed no ophthalmic pathological condition.

In the course of history-taking, the patient admitted that over the past 2 years, he had been using a stimulant drug, cocaine (intranasal insufflations, 2–3 times a week).

The visual acuity of the right eye was 0.02 sph (–) 10.0 D = 0.2 and that of the left eye was 0.06 sph (–) 10.0 = 1.0 at the time of admission to the hospital. Intraocular pressure was 20 mm Hg (by Maklakov). The visual field limits of both eyes were not narrowed.

Right eye. The vitreous body and the anterior segment of the eyeball did not present any changes. Ophthalmobiomicroscopy revealed moderate ischemic edema of the retina in the macular area and along the vascular arcades, significantly narrowed arteries (suggesting the ischemic nature of edema of the paravasal and macular regions of the retina), single hemorrhages on the optic disc, and petechial intraretinal hemorrhages along the vascular arcades, as well as insignificant tortuosity and plethora of retinal veins.

Changes in the venous system were considered as the manifestations of noncompensated arterial hypertension.

Left eye did not present any pathological changes.

Automated perimetry showed changes in the central visual field of the right eye, correlating with the topography of the retinal edema areas.

Doppler ultrasound (US) of the neck and head vessels revealed normal blood flow through the internal carotid arteries. There were signs of local stenosis in the territory of both (especially the right) ophthalmic arteries, and in the central retinal artery of the right eye there was a threefold decrease in blood flow.

An electrophysiological examination of the optic nerve of the right eye in the variant of the analysis of visual evoked cortical potentials revealed a disorder of signaling transduction at the level of the third neuron (axons of retinal ganglion cells). Left eye indices had no abnormalities.

Optical coherence tomography (OCT) of the right eye showed retinal thickening and decreased transparency in the foveolar zone, as well as disorder of differentiation of its layers. The left eye had no pathological changes.

OCT angiography confirmed the ischemic nature of edema of the paravasal and macular parts of the retina (Fig. 1). The left eye revealed no pathological changes.

The patient's examination included a clinical blood test, lipidogram, coagulogram, indicators of intravascular platelet activation, blood sugar level, blood plasma homocysteine, antiphospholipid antibodies, protein C and S levels, electrocardiogram, an internist's consultation, and magnetic resonance imaging of the brain in vascular mode.

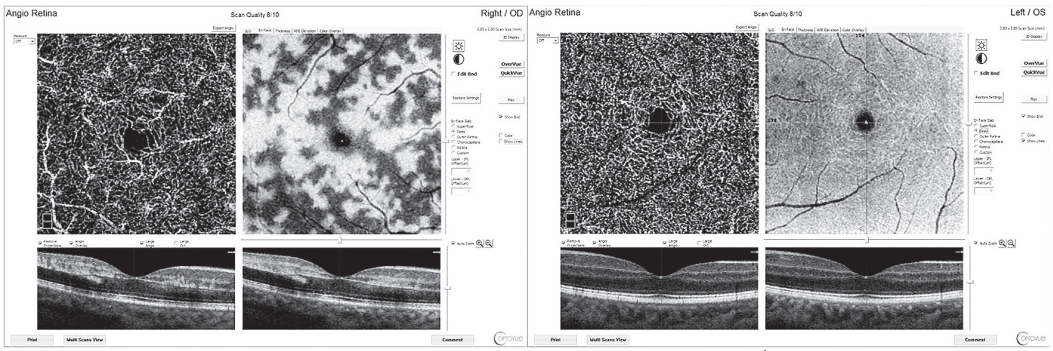
The clinical, biochemical, and immunological blood parameters evaluated together with ophthalmological examination ruled out hyperhomocysteinemia, coagulopathy and thrombophilia, antiphospholipid syndrome, retinovasculitis, diabetes mellitus, as well as heart and kidney diseases in the patient [1, 8].

Magnetic resonance imaging of the brain did not reveal any pathological changes.

A concomitant diagnosis of hypertension of the degree I and the risk of cardiovascular complications of the degree II was made as per the results of the examination.

In addition to quitting drug consumption, treatment involved improvement of microcirculation (vinpocetine infusions), lowering intraocular pressure (Lasix, Trusopt), local vasodilator and antiedematous therapy (pentoxifylline, dexamethasone) [1].

The corrected visual acuity of the right eye reached 0.7 after 2 weeks of treatment. Ophthalmoscopy revealed obvious positive changes in the form of normalization of the caliber of retinal vessels, reduction



a *b*

Fig. 1. Optical coherence tomography angiography of both eyes showing ischemic retinal edema in the right eye; *a* – right eye, *b* – left eye

Рис. 1. Данные оптической когерентной томографической ангиографии сетчатки обоих глаз, демонстрирующие ишемический отёк сетчатки на правом глазу; *a* — правый глаз, *b* — левый глаз

of ischemic retinal edema, and resorption of some hemorrhages.

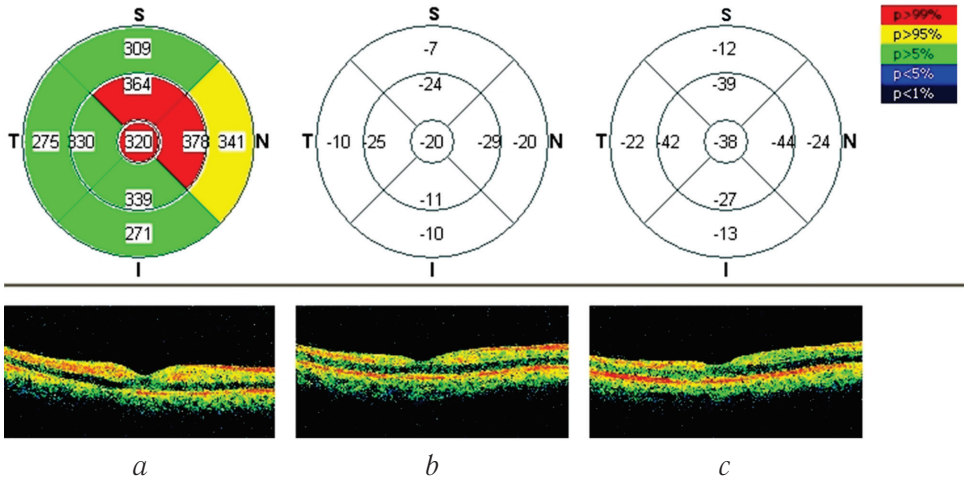
The corrected visual acuity of the right eye increased to 0.85 after 1 month, accompanied by an almost complete regression of the retinal edema with the restoration of the macular reflex, the disappearance of hemorrhages, and minor blanching of the optic disk. The OCT results confirmed a decrease in the retinal thickness, an increase in its transparency, and differentiation of the layers (Fig. 2). The vascular bed was completely passable, and no extravasation from the vessel walls was detected as per the data of fluorescein angiography.

Retinal ischemia was reversible in this case, since it was caused by arterial spasm [17]. The prognosis is usually more favorable with angiospasm than with embolism. Vision can be restored completely with a short-term spasm; however, a prolonged spasm of the central retinal artery can lead to occlusion of its branches with persistent visual deficit [15, 17].

DISCUSSION

Cocaine is one of 14 alkaloids that are extracted from the leaves of the coca plant of the genus *Erythroxylum* (*Erythroxylum novogranatense, Erythroxylum coca*), which grows in South and North America, India, and on the island of Java. Local residents have chewed the leaves of the plant for a long time to fight fatigue and to cheer up. Cocaine is a semi synthetic drug obtained from ecgonine (a product of saponification of coca alkaloids) by precipitating it from an aqueous solution. Additive-free cocaine and cocaine hydrochloride are represented by white solid crystals. There are various routes of cocaine administration: intranasal insufflation, smoking, intravenous administration, etc. [11, 14]. Cocaine is found in the blood after about 30 minutes when taken orally, reaching a maximum concentration within 50–90 minutes. Clinical effects appear 3 minutes after inhalation and last for 30–60 minutes when administered intranasally; the maximum plasma concentration is found in about 15 minutes. Intranasal absorption occurs through the highly vascularized mucous membrane of the upper respiratory tract [2]. Cocaine metabolism occurs predominantly in the liver within 2 hours after administration. The drug penetrates through the blood–brain barrier.

Cocaine has a complex pharmacological action, providing powerful sympathomimetic and vasoactive effects. The drug enhances the effect of catecholamines (norepinephrine, dopamine, and serotonin) and inhibits their reuptake, increasing sensitivity of adrenergic nerve endings to norepinephrine. This effect results in direct vasoconstriction of vascular smooth muscles, increase in platelet aggregation, and oxidative stress, leading to mitochondrial dysfunction [5, 15]. The local anesthetic effect occurs due to the blockade of sodium channels. Activation of catecholamines causes a rapid increase in the heart rate, respiration rate, metabolism, vascular tone,



a *b* *c*

Fig. 2. Optical coherent tomography of the right eye in dynamics: *a* – on admission to the hospital: an increase of the retinal thickness and decrease in its transparency, a violation of the differentiation of layers; *b*, *c* – 2 weeks and a month after treatment: showing a decrease in the thickness of the retinal macula and an improvement in the differentiation and transparency of the inner layers of the retina

Рис. 2. Данные оптической когерентной томографии правого глаза в динамике: *a* — при поступлении: утолщение сетчатки и снижение её прозрачности, нарушение дифференциации слоёв за счёт отёка; *b*, *c* — через 2 недели и месяц после лечения: снижение толщины сетчатки по всей площади макулярной зоны, улучшение дифференциации и прозрачности внутренних слоёв сетчатки

blood pressure, and body temperature [9]. Cocaine increases the endothelin-1 production; the effect is noted 12 hours after administration and reaches its maximum after 48 hours. J. Fandino [6] pointed out that endothelin-1 causes cocaine-induced cerebral vasospasm. This can cause an increase in the blood pressure, circulatory disorders in the central retinal artery, and the central retinal vein. The half-life of cocaine is 50–90 minutes, therefore it is sometimes difficult to establish a causal relationship between the drug intake and vascular lesions [11].

A large number of complications such as cardiovascular lesions, strokes, and arrhythmias are associated with cocaine abuse [15]. Numerous complications occur mainly with intravenous administration. Mydriasis, corneal lesions, circulatory disorders in the central retinal artery and central retinal vein (more commonly with intravenous use), "talc retinopathy," retinal hemorrhages, ischemic optic neuropathy, endophthalmitis (with intravenous use), and orbital lesions are the ophthalmic manifestations of cocaine abuse [7, 11, 12, 14, 16, 17, 20].

Corneal lesions (epithelial defects, keratitis, and ulcers with superinfection) result from the toxic effect of cocaine on the trigeminal nerve and a local anesthetic effect, which leads to impairment of the neurotrophy and epithelial barrier of the cornea. Microcrystals of cocaine themselves can cause keratopathy due to reflux through the nasolacrimal duct. The foreign literature presents cases of keratitis of various etiology and severity associated with cocaine intake [14, 16]. Cocaine addiction can cause keratitis in patients in the absence of risk factors for corneal infectious lesions,.

Orbital lesions with intranasal use of cocaine are associated with local ischemic damage to the mucous membrane, up to bone destruction with the development of pneumatocele. Sinusitis with orbital involvement can lead to optic neuropathy and orbital apex syndrome [4, 13, 19, 20].

Cocaine-induced vasoconstriction and vascular embolization lead to ischemic damage to the retina and optic nerve. There are cases of talc retinopathy caused by intravenous administration of cocaine [3, 12]. The English literature provides examples of vascular complications for the purpose of the necessary differential diagnosis of such conditions in young, healthy people [2, 5, 9–11, 15, 17, 18, 21].

Ischemic vascular accidents in young patients may be associated with the use of narcotic drugs that cause angiospasm and secondary changes in tissues. Special attention should be paid to these issues at the time of history-taking.

CONCLUSION

Drug abuse can cause serious diseases and even death. Currently, in Russia, serious measures are being taken to prevent the use and distribution of narcotic drugs. However, specialists should be cautious and be able to recognize the effect of drugs on the visual system and the possible complications they can evoke. Timely therapy can preserve visual function.

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