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Central retinal artery occlusion (CRAO) - case report

Jędrzej Tkaczyk[1], Klaudia Brożyna[1], Katarzyna Baltaziak[2], Krystian Ciechański[1], Dominika Nowakowska[3], Robert Rejdak[3]

[1] Student's research group at the Clinical Department of Vascular Surgery and Angiology, Medical University of Lublin
[2] Student's research group at the Clinical Department of Ophthalmology, Medical University of Lublin
[3] Clinical Department of General Ophthalmology, Medical University of Lublin

Corresponding author: Jędrzej Tkaczyk, e-mail: jedrzej.tkaczyk@gmail.com

Abstract:

Central retinal artery occlusion (CRAO) is an ophthalmic emergency, most commonly caused by an embolism, usually from the carotid arteries. It can be considered as an acute stroke of the eye, and is an ocular analogue of cerebral stroke. The incidence of CRAO is estimated to be 1 in 100 000 people. Risk factors include hypertension, carotid atherosclerosis, structural cardiac pathology, coronary heart disease, cerebral vascular accident, and diabetes mellitus. CRAO usually presents with a sudden, painless loss of vision.

52 - year old male was admitted to the Ophthalmology ER, reporting a painless monocular vision loss in a right eye for two days. After such a long time from the occurrence of symptoms, there weren't any possible treatment methods for this patient, besides preventing future embolic events. Ophthalmological examination showed only a light perception in a right eye and vision in the left eye was 1.0. Patient was diagnosed to identify risk factors of CRAO: hypertension, hypercholesterolemia, atherosclerosis or thrombophilia. He was

qualified for a careful observation for the atherosclerotic risk factors and educated, that in case of occurrence of the similar symptoms, he needs to report to a Ophthalmologist immediately.

Currently, there are two main types of treating an acute non-arteritic CRAO: the first: 'standard', non-invasive method, and the second: using the thrombolytics deployed intravenously or intra-arterially. Unfortunately, they couldn't be used to treat described patient, since he has already been in a late phase of CRAO.

Key words: CRAO, occlusion, ophthalmology

Introduction:

For the first time in the history, Central retinal artery occlusion (CRAO) was described by Von Graefe in 1859[1]. CRAO is an ophthalmic emergency, most commonly caused by embolism, usually from the carotid arteries, while the heart is probably a source of ca. <10% of emboli [2]. 74% of retinal emboli are made of cholesterol, 10.5 % of calcified material and 15.5 % of platelet fibrin [3]. It can be considered as an acute stroke of the eye, and is an ocular analogue of cerebral stroke [4]. The incidence of CRAO is estimated to be 1 in 100 000 people. Moreover, 1 in 10 000 ophthalmological outpatients suffers from CRAO [5]. Risk factors of CRAO include hypertension, carotid atherosclerosis, structural cardiac pathology, coronary heart disease, cerebral vascular accident, and diabetes mellitus [6]. CRAO is a sign of an end-organ ischaemia and often the underlying atherosclerotic disease. Moreover the CRAO episode is a risk factor of a future cerebral stroke and ischemic heart disease. CRAO usually presents with a sudden, painless, monocular vision loss [4]. However, when the cilioretinal artery is present, which occurs in ca. 49.5 % of patients, the blood supply for papillomacular bundle is enough to preserve the central vision [7]. Patients with such symptoms, should be carefully evaluated for the artherosclerotic risk factors and a family history [8]. In a study, that included 240 patients with CRAO, within 7 days, early findings in ocular evaluation were: retinal opacity in the posterior pole (58%), cherry-red spot (90%), cattle trucking (19%), retinal arterial attenuation (32%), and optic disk oedema (22%) and pallor (39%) [9].

Case report:

Figure 1. OCT of retina with CRAO, edema of macula

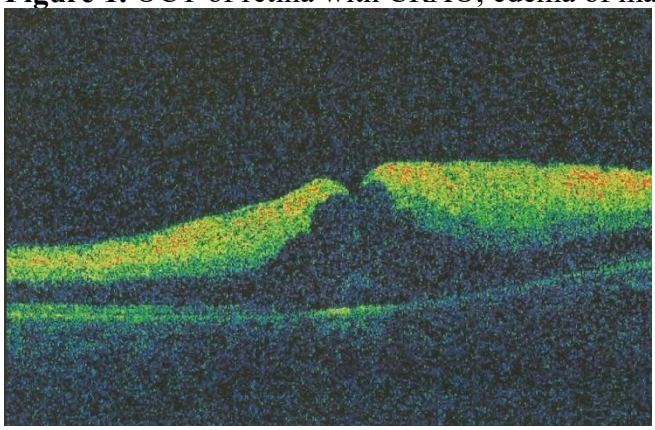
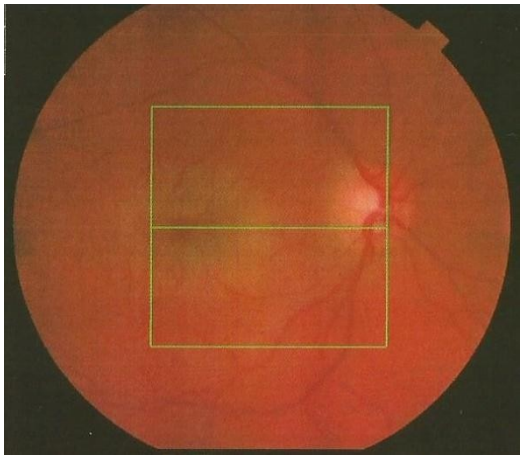


Figure 2. Color photo of fundus with CRAO



52 - year old patient was admitted to the Ophthalmology ER, reporting a painless monocular vision loss in a right eye for two days. He had no history of earlier ophthalmological treatment, and didn't visit any other doctors. After the ophthalmological examination, which consisted of fundus examination and OCT, physician confirmed Central Retinal Artery Occlusion. Unfortunately, after such a long time from the occurrence of symptoms, there weren't any possible treatment methods for this patient, besides preventing future embolic events. Patient was also directed to a Intern Medicine and Cardiology specialist, to diagnose if he has a CRAO risk factors: hypertension, hypercholesterolemia, atherosclerosis or thrombophilia. Lab tests showed high level of Total Cholesterol, LDL and TG (Table 1).

Figure 3. OCT after 4 month of CRAO

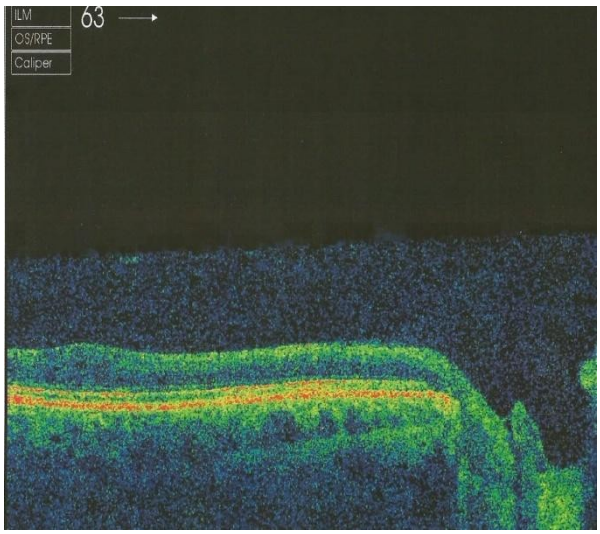


Figure 4. color photo of fundus after 4 month of CRAO

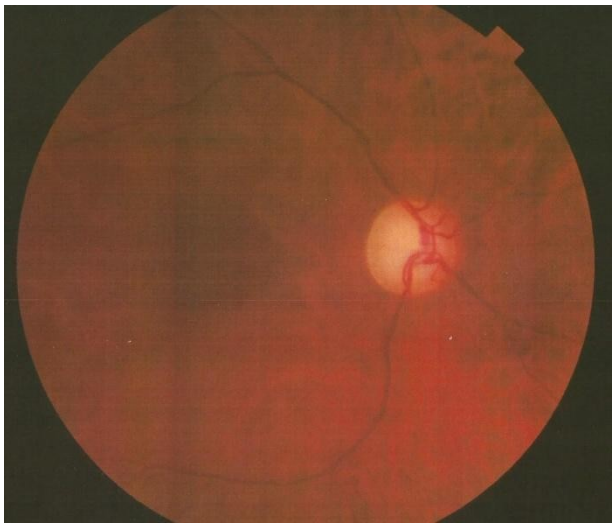


Table 1. Lab test results of the patient.

	Total Cholesterol (mg/dl)	LDL (mg/dl)	TG (mg/dl)
Result	318 ↑	208 ↑	307 ↑

Doctors prescribed Atorvastatin in a dose of 40 mg, to lower these parameters. During a follow up appointment, fundoscopic examination showed signs of a deep ischemia - optic nerve head was mealy. Ophthalmological examination showed almost no vision in a right eye - the patient only had light perception from the nasal side and from the bottom. Vision in the left eye was 1.0. The patient was qualified for a careful observation for the atherosclerotic risk factors and educated, that in case of occurance of the similar symptoms, he needs to report to a Ophthalmologist immediately.

Discussion:

Without a doubt, CRAO must be considered as a ophthalmological emergency [4], and for this reason, an effective treatment of this condition must be applied in an acute phase. Potentially a time window for a efficient therapy is 6 hours [10]. The tolerance time of retina for an acute ischemia has been evaluated in experimental studies on an elderly, atherosclerotic and hypertensive rhesus monkeys [11]. The retina showed no detectable damage to CRAO for 97 min. After that, partial recovery was possible, if the ischemia is reversed up to 240 min [11]. Analysing the natural history of the disease, we can observe that in some cases spontaneous visual improvement can occur in CRAO, but the extent of improvement depends a lot on the type and duration of the CRAO [12]. Significant improvement occurs in only ca. 10% of people with spontaneous reperfusion [13].

In the case of presented patient, there was no chance for an effective treatment, as he ignored acute symptoms of CRAO, and reported ca. month after incident. In this case, the target should be a long-term treatment that allows to prevent other vascular ischaemic events on the eye, or the other organ. Unfortunately, a few patients with CRAO are seen acutely, which is a major barrier to effective treatment [14].

Currently, there are two main types of treating an acute non-arteritic CRAO: the first: 'standard', non-invasive measures, and the second: using the thrombolytics deployed intravenously or intra-arterially [4]. Possible options of treatment are presented in the table 2 [10].

There have been two randomised controlled trials, investigating 'conservative' CRAO treatment methods [15], that showed, that oral pentoxifylline and enhanced external counterpulsation may have a role in CRAO treatment. The efficacy of 'conservative' therapy varies between 6 and 49%, and mean visual improvement rate 15-21% [16]. However, these therapies do not alter the outcome more than the natural history of the disease [13, 17].

Local intra-arterial (IA) thrombolysis has been used in CRAO since 1984. It was described to be effective in some retrospective studies [18, 19]. However, in a prospective, multicentre, randomized controlled trial EAGLE (The European Assessment Group for Lysis in the Eye), which contained 85 patients with CRAO, there was no statistically significant difference in clinical improvement between the lysis and standard therapy groups, and the rate of adverse events was far higher in the local IA fibrinolysis compared with the standard therapy group [16].

Thrombolysis, can be also through a vein, as in a standard ischaemic stroke thrombolysis protocol. This method, has a theoretical advantage of no need for specialized interventional radiology set-up and lower risk of hemorrhagic complications [20]. Disadvantages include risk of direct vascular injury, stroke, and longer procedural time [21]. Satisfying effect can be achieved, if the thrombolysis is administered within 6 hours [11].

Future studies of treating CRAO, must focus on the potential, life-threatening adverse effects, and balance it with the eyesight-preserving benefits of thrombolysis [4].

Table 2. Treatment options for an acute CRAO [10].

	Mechanism of action	Group of drugs
1.	Increasing the blood oxygen content	Vasodilators Pentoxifylline Inhalation of carbogen Hyperbaric oxygen Sublingual Isosorbide Dinitrate
2.	Reducing intraocular pressure and hence increase the retinal artery perfusion or help dislodge the embolus.	Ocular massage Anterior chamber paracentesis Intravenous acetazolamide Intravenous mannitol Topical antiglaucoma medications
3.	Reduction of retinal oedema	Intravenous methylprednisolone
4.	Lysing or dislodging the clot	Nd YAG Laser Embolectomy
5.	Help in thrombolysis of the embolus	Intra-arterial or intravenous thrombolysis

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