Polskie Towarzystwo Okulistyczne
www.pto.com.pl
KLINIKA OCZNA 2021, 123, 1: 35–38
Received: 18.07.2020 Accepted: 18.08.2020

CASE REPORT

Access this article online
Website: www.klinikaoczna.pl
DOI: https://doi.org/10.5114/ko.2021.104750

Branch retinal artery occlusion as an acute complication of coronary angiography

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ABSTRACT

Introduction: Retinal artery occlusion (RAO) is one of the rare complications of intravascular procedures. It is most often caused by atheromatous plaque material moved from the aortic arch or carotid artery wall as a result of mechanical manipulation.

Case report: We report a case of a 70-year-old man, who presented with vision deterioration in his right eye (RE) during coronary angiography (CAG). Computed tomography (CT) scan of the patient's brain was unremarkable. The patient was subsequently referred to the Department of Ophthalmology. On admission, the best-corrected visual acuity (BCVA) was hand motions in the RE. He had a relative afferent pupillary defect and posterior segment examination revealed pale macula with cherry red spot as well as embolic material in the lower vascular arch in the RE. Ocular massage and an anterior chamber paracentesis were performed. Fundus fluorescein angiography (FFA) confirmed the lack of perfusion through the lower temporal branch of the right central retinal artery, and partial return contrasting of this vessel in late phases was described. This clinical picture suggested lower temporal branch right retinal artery occlusion (BRAO).

Conclusions: Intravascular procedures, including CAG, may result in RAO. Awareness of the possibility of ocular symptoms during and after the surgery, early diagnosis and initiating appropriate treatment are crucial in preventing permanent visual impairment.

KEY WORDS: branch retinal artery occlusion, BRAO, coronarography, coronary angiography.

INTRODUCTION

Coronary angiography (CAG) plays a key role in detecting and assessing the severity of atherosclerotic coronary artery disease (CAD). Although there are no absolute contraindications for CAG, one must bear in mind that, like any invasive procedure, it carries some risk. Older age, renal insufficiency, uncontrolled diabetes mellitus, morbid obesity, the extent of CAD, congestive heart failure (CHF) with low ejection fraction (EF), recent stroke, myocardial infarction (MI) and coagulation disorders increase the risk of complications [1]. Moreover, operator expertise and caution are closely associated with new retinal emboli [2].

Complications after the procedure include allergic and adverse reactions, infections, nephropathy, cholesterol emboli, local vascular injury, conduction disturbances, MI, cerebrovascular complications, dissection and perforation of vessels, hypotension, hypoglycemia and respiratory insufficiency [1, 3]. Visual symptoms after CAG are extremely rare, and their differential diagnosis should include contrast induced transient cortical blindness, retinal artery occlusion (RAO), amaurosis fugax and hysterical blindness [4, 5]. Both branch (BRAO) and central (CRAO) RAO can lead to a wide spectrum of visual changes from asymptomatic through partial vision loss up to complete blindness.

CASE REPORT

A 70-year-old man complaining of chest pain was admitted to the Department of Cardiology. His medical history included hypertension, second-degree atrioventricular block, and past MI. He had no history of diabetes mellitus, hyperlipidemia or smoking. The patient underwent coronarography because of recurrent chest pain, during which he reported a sudden and painless visual loss in his right eye (RE). Immediately after the onset of symptoms, the procedure was discontinued. Head computed tomography (CT) as well as CT angiography of the cerebral arteries were performed to exclude an ischemic stroke, but the examinations showed no significant changes and normal contrast flow through the ophthalmic arteries.

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The patient was subsequently referred to the Department of Ophthalmology. On admission, the best-corrected visual acuity (BCVA) was hand motions in RE and 1.0 in the left eye (LE). Intraocular pressure (IOP) was 16 mmHg in the RE and 15 mmHg in the LE on applanation tonometry. Slit lamp examination showed a relative afferent pupillary defect in the RE, keratopathy and an incipient cataract in both eyes. There were no signs of inflammation. Fundus examination of the RE revealed a pale macula with a cherry red spot as well as embolic material in the lower vascular arch. No abnormalities were detected in the picture of the OL fundus. Ocular massage was performed, and the patient immediately underwent an anterior chamber paracentesis.

Doppler ultrasound (DUS) examination of the retinal artery showed in the RE maximal velocity (V_{max}) = 8.9 cm/s; minimal velocity (V_{min}) = 0.8 cm/s; pulsatility index (PI) = 2.08; resistance index (RI) = 0.91. Results in the LE were within normal limits.

In fundus fluorescein angiography (FFA), the lack of perfusion through the lower temporal branch of the central retinal artery in the RE (Figure 1A) and partial retrograde filling of this vessel in late phases were described (Figure 1B). This clinical picture suggested lower temporal BRAO RE; however, partially recanalized CRAO cannot be excluded. The probable cause of BRAO, in this case, was atheromatous plaque material dislodged by catheter manipulation inside the vascular lumen. During hospitalization the patient received 40 mg of low molecular weight heparin (LMWH) subcutaneously, topical fluoroquinolone as well as brimonidine and dorzolamide twice daily.

After 3 days of hospitalization at the Department of Ophthalmology, the patient did not agree to be transferred to the Department of Cardiology where he had stayed previously. He was discharged with the recommendation of urgent cardiological and neurological control. At discharge, BCVA of the RE was hand motions. 2 days later, the patient was taken to the Department of Surgery due to gastrointestinal bleeding. In addition, after 2 weeks, another coronarography with subsequent percutaneous coronary intervention (PCI) of the right coronary artery and the implantation of two drug-eluting stents (DESs) was performed in the Department of Cardiology. At the 1-year clinical follow-up BCVA of the right eye was still hand motions.

DISCUSSION

RAO (BRAO more frequently than CRAO) is considered to be a rare complication of intravascular procedures used for diagnostic and therapeutic purposes including transfemoral cerebral angiography (TFCA), PCI, cardiac catheterization and vascular stenting [6]. There have been some case reports of RAO after CAG [6-10]. However, some researchers suggest that the incidence of this complication may be higher (from 2% to even 6%) due to the small size of the emboli and possibly asymptomatic course [2, 11].

Mechanical manipulation over ulcerated, disrupted atherosclerotic plaques causes the release of cholesterol crystals in the arterial bloodstream, which can result in many organs failing. The symptoms may include skin lesions, lower extremity ischemia, renal insufficiency, gastrointestinal bleeding, arterial hypertension, and retinal embolism [12, 13]. Moreover, patients undergoing coil embolization for aneurysms or balloon aortic valvuloplasty can develop RAO as a result of embolism caused by the displacement of a newly

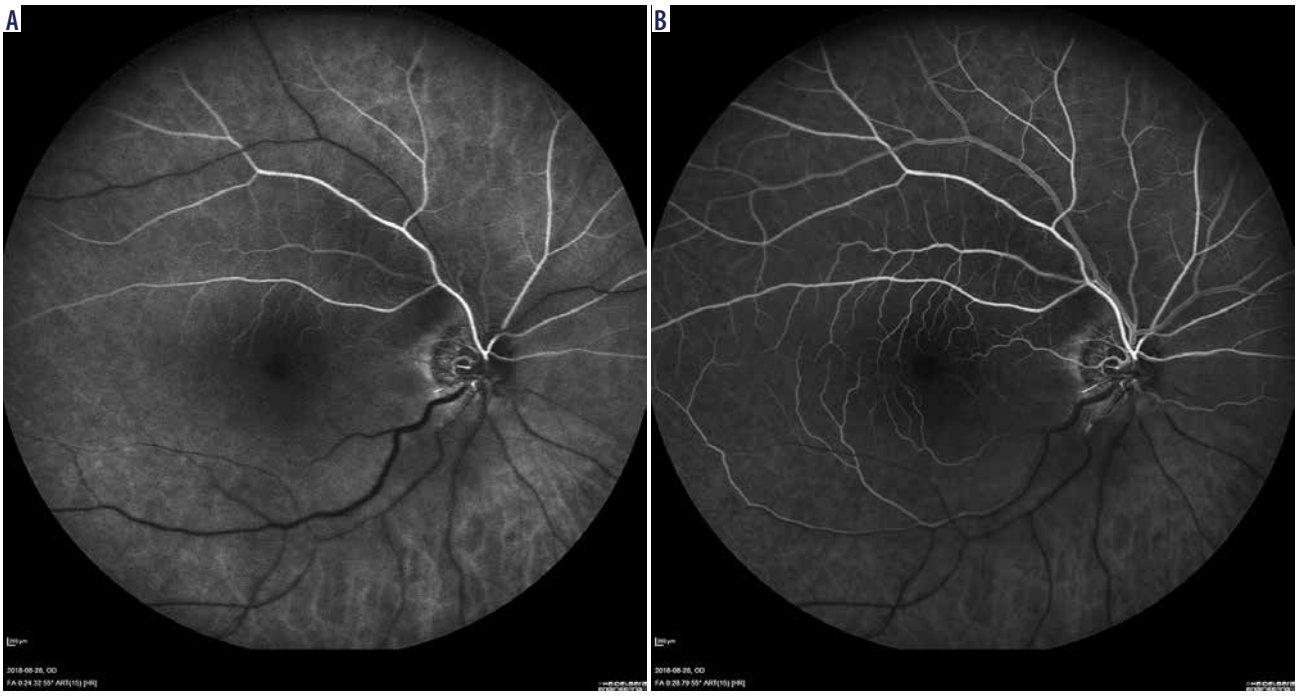

Figure 1. Fundus fluorescein angiography of the right fundus (28.08.2018, 11 a.m.): **A**) 0:24.32 – the lack of perfusion through the lower temporal branch of the central retinal artery; **B**) 0:27.94 – partial retrograde filling of the lower temporal branch of the central retinal artery in late phases

formed thrombus that was formed at the site of the coil or repaired valve. Furthermore, sclerosing agents, used for sclerotherapy for arteriovenous malformation or hemangioma, may induce thrombus formation by denaturation of red blood cell proteins [6, 14].

In 2/3 of patients RAO occurs immediately after or even during intravascular procedures, whereas in 1/3 delayed onset of symptoms is recorded (1-3 days after surgery). This may be due to delayed release of embolic material after the procedure or partial artery obstruction, which becomes symptomatic after complete artery occlusion as a result of a delayed inflammatory response associated with endothelial proliferation, foreign body reaction and eosinophilia [6, 15]. In addition, RAO can be diagnosed with delay due to late detection, especially in critically ill or impaired patients who do not report symptoms [6, 9].

Ocular manifestations after CAG are rare and are usually associated with contrast induced transient cortical blindness, which is the result of a neurotoxic action of the contrast agent on the blood-brain barrier in the occipital lobe [4]. It should be borne in mind that cortical stroke is also one of the complications after arterial surgery and can also lead to decreased vision, although without changes in the fundus. CT scan of the brain plays a key role in ruling out a cerebral vascular event.

There is currently no universal, effective treatment for vision loss caused by RAO. However, ocular massage, reduction of IOP (by topical and systemic medications, anterior chamber paracentesis), and vasodilatation of the retinal arteries (by 95% oxygen with 5% carbon dioxide inhalation, sublingual isosorbide dinitrate) can be tried in the acute phase. These actions are aimed at reperfusion of the blocked retinal vessels and reversing or reducing the damage caused by tissue infarction [10, 16, 17]. An experimental study [18] showed that if circulation is restored within 97 minutes, no permanent ischemic retinal damage is reported. However, after 240 min CRAO causes massive and irreversible damage to the retina. Therefore, the above-mentioned actions do not bring results when retinal edema and a cherry-red spot are already visible in the fundus examination, because these symptoms develop a few hours after retinal artery occlusion. Additionally, chronic treatments (antiplatelet and heparin therapy) are aimed at reducing platelet adhesiveness and preventing dislocation of potential future emboli and thrombus formation [17].

Acute retinal arterial ischemia is a stroke equivalent and constitutes an ophthalmologic and neurologic emergency. Assessment by a neurologist, with immediate brain and vascular imaging, should be performed as soon as possible, because cerebrovascular accidents are associated with a greater risk of disability and mortality. Ideally, an ophthalmological examination needs to be conducted in the emergency department. However, the ocular evaluation should not delay diagnostic imaging tests. Therefore, the present patient underwent head CT and CT angiography of the cerebral arteries first and was then referred to the Department of Ophthalmology. Even if an ophthalmologist as the first specialist confirmed the diagnosis of CRAO or BRAO, the patient should be immediately referred to the closest emergency department to initiate an urgent and thorough stroke evaluation as well as to identify and treat disorders connected with a higher risk for cerebrovascular and cardiovascular events. It should be noted that acute cerebral infarctions were found in 27% to 76.4% of CRAO patients [19].

Among patients with vascular risk factors, including atherosclerosis, the risk of RAO or cerebral infarction after intravascular procedures should be considered. In addition, the patients should be informed about possible thromboembolic complications and the need to immediately report disturbing symptoms. If visual symptoms appear, an ophthalmic examination should be performed urgently.

CONCLUSIONS

The development of retinal vascular occlusion during or directly after intravascular procedures cannot be completely prevented. However, the cooperation of cardiologists, interventional cardiologists and ophthalmologists, as well as being vigilant for mild visual disturbances during and following the procedure, should aid in making the right diagnosis and initiating the appropriate treatment as soon as possible.

DISCLOSURE

The authors declare no conflict of interest.

References

1. Tavakol M, Ashraf S, Brener SJ. Risks and complications of coronary angiography: a comprehensive review. Glob J Health Sci 2012; 4: 65-93.
2. Kojuri J, Mehdizadeh M, Rostami H, et al. Clinical significance of retinal emboli during diagnostic and therapeutic cardiac catheterization in patients with coronary artery disease. BMC Cardiovasc Disord 2011; 11: 5.
3. Endemann DH, Philipp A, Hengstenberg C, et al. A simple method of vascular access to perform emergency coronary angiography in patients with veno-arterial extracorporeal membrane oxygenation. Intensive Care Med 2011; 37: 2046-2049.
4. Tatli E, Buyuklu M, Altun A. An unusual but dramatic complication of coronary angiography: transient cortical blindness. Int J Cardiol 2007; 121: 4-6.
5. Akhtar N, Khatri IA, Naseer A, et al. Transient cortical blindness after coronary angiography: a case report and literature review. J Pak Med Assoc 2011; 61: 295-297.
6. Choo SC, Jung C, Lee JY, et al. Retinal artery occlusion after intravascular procedures. Case series and literature review. Retina 2019; 39: 766-778.
7. Kopsachils N, Brar M, Marinescu AI, et al. Isolated retinal cotton wool spot after coronary angiography. Oman J Ophthalmol 2013; 6: 51-52.

8. Selton J, Maalouf T, Olivier A, et al. Occlusion de l'artère centrale de la rétine après une coronarographie : à propos d'une observation. J Fr Ophtalmol 2011; 34: 181-185.
9. Kymionis GD, Tsilimbaris MK, Christodoulakis EB, et al. Late onset branch retinal artery occlusion following coronary angiography. Acta Ophthalmol Scand 2005; 83: 122-123.
10. O'Neill MD, Akerele T, Dancy M. Branch retinal artery occlusion during coronary angiography. Postgrad Med J 2005; 81: 710.
11. Kreis AJ, Nguyen T, Rogers S, et al. Acute retinal arteriolar emboli after cardiac catheterization. Stroke 2008; 39: 3086-3087.
12. Blanco VR, Moris C, Barriales V, et al. Retinal cholesterol emboli during diagnostic cardiac catheterization. Catheter Cardiovasc Interv 2000; 51: 323-325.
13. Saric M, Kronzon I. Cholesterol embolization syndrome. Curr Opin Cardiol 2011; 26: 472-479.
14. Lee JJ, Do YS, Kim JA. Serum ethanol levels after alcohol sclerotherapy of arteriovenous malformations. J Korean Med Sci 2004; 19: 51-54.
15. Bittl JA. Cholesterol embolization syndrome: unifying principles. Catheter Cardiovasc Interv 2000; 51: 326-327.
16. Hsien YM, Mustapha M, Hamzah JC, et al. Why can't I see after my heart is fixed: a case series of ocular complications after cardiac intervention. BMC Ophthalmol 2016; 16: 32.
17. Sun R, Peng XY, You QS, et al. Branch retinal artery occlusion following carotid stenting: A case report. Exp Ther Med 2016; 12: 2183-2186.
18. Hayreh SS, Zimmerman MB, Kimura A, et al. Central retinal artery occlusion. Retinal survival time. Exp Eye Res 2004; 78: 723-736.
19. Biousse V, Nahab F, Newman NJ. Management of Acute Retinal Ischemia: Follow the Guidelines! Ophthalmology 2018; 125: 1597-1607.