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CENTRAL RETINAL ARTERY OCCLUSION AFTER CARDIOVERSION OF ATRIAL FIBRILLATION

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Purpose: To describe a rare case of unilateral central retinal artery occlusion in the postprocedural period of electrical cardioversion (CV) for atrial fibrillation.

Methods: History and ophthalmic examination.

Results: A 79-year-old female underwent electrical cardioversion. This resulted in the restoration of sinus rhythm. Ten days after the cardioversion, the patient experienced sudden vision loss in her right eye. She was diagnosed with a right occipital infarct. and a right central retinal artery occlusion.

Conclusion: Central retinal artery occlusion is a rare complication of cardioversion that can lead to severe visual loss in the affected eye.

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We report a rare case of unilateral central retinal artery occlusion in the postprocedural period of electrical cardioversion (CV) for atrial fibrillation. In this case, sudden visual loss and a stroke occurred a few days after the procedure.

Cardioversion represents an essential component of the rhythm control strategy for atrial fibrillation (AF). The thromboembolic risk of CV is well established, and the mechanisms behind the phenomenon have been comprehensively described.[1]

Embolism is the main cause of central retinal artery occlusion (CRAO), and the main sources of embolism are the carotid artery and the heart. Atrial fibrillation is known as a risk factor of retinal vessel occlusion; however, no case reports are available on CRAO related to AF ablation by electrical CV.

We report a case of CRAO in the postprocedural period of CV for AF.

Case Report

Our patient is a 79-year-old female patient with a medical history of hypercholesterolemia and atrial fibrillation. Her treatment included a direct oral anticoagulant, rivaroxaban and antiarrhythmics. However, despite the administration of these medications, the patient began to experience symptoms of shortness of breath on inclines and tiredness, both of which reduced her exercise capacity.

Based on these findings, a decision was made to proceed with an elective DC cardioversion.

A transthoracic echocardiogram completed 3 months before the CV reported a severely dilated left atria with a volume of 107 mL and a diameter of 4.8 cm, associated with a mild-to-moderate mitral regurgitation. No cardiac thrombus was detected. The ultrasound doppler of the carotid arteries excluded other sources of emboli as it showed no evidence of any significant extracranial carotid artery stenosis.

Before the procedure, the patient was recommended to use edoxaban instead of rivaroxaban for 1 month. After this, the

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electrical CV was successfully performed, resulting in the restoration of a sinus rhythm. The medical team advised against discontinuing the anticoagulation for at least four weeks following the procedure.

However, 10 days after the CV, the patient experienced sudden vision loss in her right eye. She was promptly admitted to the stroke unit, where she was diagnosed with a right occipital infarct. As a result of the complete loss of vision in her right eye persisting for over 4 hours, she was subsequently referred to our eye casualty clinic for further evaluation and management.

On ophthalmological assessment, it was observed that the right eye had no perception of light, whereas the left eyesight was 0.22 logMar.

The patient did not report any additional ophthalmic symptoms.

On fundoscopy (Figure 1), the retina in the right eye appeared pale with a distinct cherry red spot at its center, leading to the diagnosis of central retinal artery occlusion.

Discussion

Central retinal artery occlusion is caused by any process that creates a transient or permanent interruption in the blood supply to the central retinal artery, and its causes are similar to those of cerebral infarctions in the anterior circulation. A CRAO may be broadly classified as arteritic or nonarteritic, depending on its etiology.

In almost all patients, CRAO is said to be nonarteritic. The most common cause of CRAO is a thromboembolic disease originating in the ipsilateral carotid artery, aortic arch, or heart, although the source of the causative embolus remains unknown in 60% of patients with CRAO. Retinal emboli may be composed of cholesterol (75% of cases), platelet-fibrin (15%), or calcium (10%).[2]

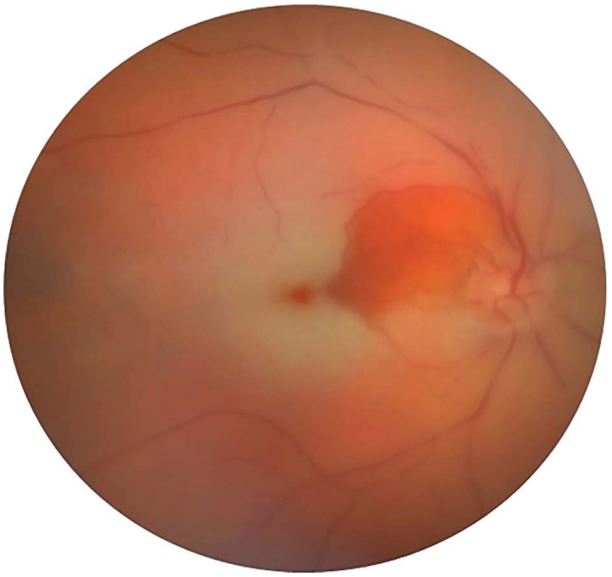

Fig. 1. Right central retinal artery occlusion with retinal edema and cherry red spot at fovea. There is cilio-retinal artery perfusion in the superior macula.

Nonarteritic CRAO represents the ocular analog to ischemic events of the cardiovascular and cerebrovascular systems. As is the case with these entities, CRAO is strongly related to the presence of cardiovascular risk factors, including hypertension, atherosclerosis, and diabetes mellitus. The European Assessment Group for Lysis in the Eye trial identified greater than 70% stenosis of the carotid artery in 40% of patients with CRAO; 73% had arterial hypertension, 22% had coronary artery disease, 20% had a cardiac arrhythmia, and 17% had valvular heart disease.[2]

Atrial fibrillation is a risk factor of CRAO. A previous study using implantable loop recorders reported that 15% of patients with CRAO had subclinical AF.[3] Another study aiming to determine the prevalence of AF in patients with ischemic monocular blindness revealed that a total of 9% of patients with ischemic monocular blindness had AF.[4]

The essential and effective component in the rhythm management of AF is CV. However, the procedure includes a well-established risk of thromboembolic complications.[1]

There are several mechanisms facilitating the well-established risk of thromboembolism during AF. These pathophysiological mechanisms include abnormalities in the blood constituents, the activation of inflammatory responses, and anatomical and structural abnormalities of the atrial tissue. In addition to the risk of a preexisting left atrial thrombus dislodgement once the atrial contractility recovers, the rhythm conversion may also initiate a process, leading to a thrombus formation. This phenomenon is explained in part by the generally accepted concept of atrial stunning, where the rhythm conversion (with CV or spontaneously) from AF to sinus rhythm results in mechanical stunning of the atria. The resulting deceleration of blood flow in the atria promotes thrombus formation, especially in the left atrial appendage where the majority of AF-related cardiogenic thromboembolisms originate.[1]

Early studies on the thromboembolic risk of CV showed a high (3.4–7%) periprocedural risk for patients not using anticoagulation, but the risk has lowered to as low as 0.8% with vitamin K antagonists.[1]

The contemporary risk of thromboembolic complications with the periprocedural use of non–vitamin K oral anticoagulants seems low at first glance, but randomized non–vitamin K oral anticoagulant trials (RE-LY, ARISTOTLE, ENGAGE-AF, and ROCKET-AF) have established a lower monthly ischemic stroke rate, varying between 0.08% and 0.12% per month in anticoagulated patients. Taking all these figures into consideration, each elective CV seems to predispose the patient to an additional fourfold (0.4% versus

0.1%) risk of stroke during the post-CV month—even during effective periprocedural anticoagulation with non–vitamin K oral anticoagulants.[1]

Despite the well-established risk of thromboembolic complications following CV, the occurrence of CRAO after CV has not been previously reported. The reason might be that atrial thrombi are less likely to reach the ophthalmic artery. A hypothesis suggested to explain this observation pertains to the emboli's size. Retinal symptoms may occur with embolism of smaller particles, characteristic of artery-to-artery embolism, in contrast to brain symptoms, which require larger particles, perhaps more typical of clots from a cardiac source.[5] In other words, emboli arising from the carotid artery tend to be smaller than thrombi arising in the heart. Therefore, biomechanically, the smaller emboli from carotid plaques are more likely to drift near the edges of the bloodstream and be swept into the nearby ophthalmic artery, but the large cardiac thrombi tend to travel centrally in the bloodstream and reach the most distal vascular territory in the brain.[6]

Central retinal artery occlusion following electrical CV of AF is rare. It is a cause of severe visual loss with no consensus about its management. All proposed treatments are of questionable efficacy, and many have uncertain risk profiles. In certain circumstances, thrombolysis may be attempted as a treatment option; however, the evidence to support the broad use of thrombolytics in the treatment of acute CRAO remains elusive.[2]

Conclusions

We highlight in this case a potentially unreported complication of CV: CRAO. It is important that cardiologists are aware of this potential complication, so they can provide suitable informed consent to their patients before this procedure.

Key words: central retinal artery occlusion, cardioversion, atrial fibrillation, cardiogenic embolism, retinal arteries, anticoagulation.

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