

# Central retinal artery occlusion after catheter ablation of atrial fibrillation

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## Abstract

We report the first case with central retinal artery occlusion (CRAO), which is a rare but ophthalmic emergency complication, in periprocedural periods of atrial fibrillation ablation. In this case, sudden visual loss occurred after the procedure and visual loss was persisted. Operators should know the incidence and management of CRAO.

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**Short title:** Central retinal artery occlusion after ablation

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**Key words:** Atrial fibrillation, Catheter ablation, Complication, Cardiogenic embolism, Central retinal artery occlusion.

## Abstract

We report the first case with central retinal artery occlusion (CRAO), which is a rare but ophthalmic emergency complication, in periprocedural periods of atrial fibrillation ablation. In this case, sudden visual loss occurred after the procedure and visual loss was persisted. Operators should know the incidence and management of CRAO.

## Key Clinical Message

Central retinal artery occlusion (CRAO) is a rare but severe and urgent complication of atrial fibrillation ablation. Prompt treatment is needed because ischemic damage to the retina is irreversible from 4 hours after the onset of CRAO.

## Introduction

Catheter ablation is an important therapy for atrial fibrillation (AF).<sup>1</sup> Although there is abundant evidence that catheter ablation is superior to antiarrhythmic drugs, there is a small but definite probability of complications in the periprocedural period.<sup>1</sup> Two of the major complications of AF ablation are stroke and other thromboembolic events. Previous studies have reported incidences of thromboembolism associated with AF ablation of 0%–7%.<sup>2</sup>

Central retinal artery occlusion (CRAO) is an ophthalmic emergency because it causes sudden and severe visual loss.<sup>3</sup> Prompt treatment is needed because ischemic damage to the retina is irreversible from 4 h of onset.<sup>3</sup>

Embolism is the main cause of CRAO, and the main sources of embolism are the carotid artery and the heart.<sup>4</sup> AF is known as a risk factor for retinal vessel occlusion,<sup>5</sup> and some previous studies have reported on catheterization procedures as the cause of CRAO.<sup>6,7</sup> However, no case reports are available on CRAO related to AF ablation.

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

## Case report

A 79-year-old male was referred to our hospital because of dyspnea on exertion in May 2019. His CHA<sub>2</sub>DS<sub>2</sub>-VASc score was 3 points and he had moderate aortic valve stenosis. AF was found on 12-lead electrocardiography. We performed a radiofrequency catheter ablation with pulmonary vein isolation and cavo-tricuspid isthmus ablation in May 2019. AF recurred at 4 months post ablation.

After confirming there was no thrombus in the left atrial appendage by transesophageal echocardiography, we performed a second AF ablation in November 2019.

The patient had been taking apixaban 5 mg twice daily before the procedure. On the day of the procedure, the morning dose was withheld and the evening dose was administered.

The procedure was performed under intravenous conscious sedation with dexmedetomidine and fentanyl. Electrophysiological studies and catheter ablation were performed using an electro-anatomical mapping catheter (Rhythmia, Boston Scientific, Boston MA, USA) and an open-irrigated linear ablation catheter with a 3.5-mm tip (INTELLATIP MiFi, Boston Scientific).

The cardiac rhythm at the beginning of the procedure was atrial tachycardia, and electrophysiological study revealed that the atrial tachycardia was perimitral flutter. Ablation creating left atrial anterior line changed perimitral flutter into biatrial tachycardia. After that, lateral mitral isthmus linear ablation including both endocardial and epicardial radiofrequency application terminated the biatrial tachycardia.

After linear ablation of the left atrium, no atrial tachyarrhythmia was induced by atrial burst stimuli. Reconnections of the pulmonary vein and cavo-tricuspid isthmus were not found. In this case, the body mass index was high and glossoptosis with conscious sedation tended to occur irrespective of using noninvasive positive pressure ventilation. The procedural time was 209 minutes. We used heparin and set target activated clotting time at 300 s during the procedure (Figure 1). Baseline and procedural characteristics of the patient are shown in Table 1.

The patient was asymptomatic until visual loss in the left eye occurred 3.5 h after the procedure. Eyesight in the left lower quadrant was limited to counting fingers, and eyesight in the rest of the left eye was light perception only. There was no identifiable ischemic stroke or hemorrhage which caused visual loss on brain computed tomography or magnetic resonance imaging. We consulted with ophthalmology. A fundus examination revealed a cherry red spot in the left eye and he was diagnosed with CRAO (Figure 2).

Ocular massage was performed, and nitroglycerine and 5000 units of heparin sodium were infused. However, eyesight did not improve. An infusion of 10 µg/h of alprostadil was also initiated 4 h after the symptoms onset, and continued for 3 d. Carotid artery ultrasound performed on the day after the procedure showed

no mobile plaque. Although peripheral vision was improved, central visual field loss of the left eye persisted. Because the patient needed physical therapy, he was discharged 8 d after the procedure.

## Discussion

We report a case of CRAO occurring after AF ablation. In this case, sudden visual loss in the left eye occurred and did not improve well with treatment.

CRAO can be caused by retinal artery embolism or hemodynamically-induced retinal ischemia. The risk factors for CRAO are shown in Table 2.<sup>3-5</sup> AF was a cause of CRAO. A previous study using implantable loop recorders reported that 15% of CRAO patients had subclinical AF.<sup>8</sup>

There have been few reports of ophthalmic complications of AF ablation. Although a previous study showed one patient developed a unilateral quadrantanopia consistent with a retinal artery embolus after catheter ablation,<sup>9</sup> there have been no reports about CRAO with complete unilateral visual loss. To our knowledge, this is the first case report to describe CRAO following AF ablation.

Ischemic damage to the retina is irreversible within 4 hours of the onset of CRAO,<sup>3</sup> therefore prompt management is important. Common treatments for CRAO are shown in Table 3.<sup>3,10-18</sup> Ocular massage was performed because the embolus in the central retinal artery was expected to be dislodged.<sup>3</sup> As medical therapies, vasodilators such as nitroglycerine and anticoagulant such as heparin have been used; however, there is no evidence that these agents are effective. A recent study showed that digital subtraction angiography-guided superselective ophthalmic artery or selective carotid thrombolysis with urokinase and papaverine was an effective treatment method for CRAO.<sup>12</sup>

Another study showed that alprostadil infusion resulted in significant visual improvement in patients with CRAO.<sup>11</sup> In this study, the logarithm of the minimum angle of resolution best corrected visual acuity significantly improved from 2.73 to 1.48 at 1 month after the onset of CRAO, and all 6 patients experienced vision improvement.<sup>11</sup> Our use of alprostadil for 3 d somewhat improved peripheral vision.

We needed a long procedural time in this case. Therefore an embolic event was more likely to occur because long left atrium dwell time exposed this patient to thrombus formation on catheters.<sup>19</sup> The differential diagnosis in this case is air embolism vs. thromboembolism. In this case, the body mass index was high and glossotropia with conscious sedation tended to occur irrespective of using noninvasive positive pressure ventilation. This condition induced a long apnea and might be a cause of an air embolism. Deep sedation with a laryngeal mask airway might be a preventive method. If possible, avoiding a long procedural time, which is a risk factor for complication,<sup>20</sup> might also prevent complications.

To minimize the risk of permanent visual loss from CRAO, operators suspecting it should closely cooperate with an ophthalmologist to diagnose this disease as soon as possible.

## Conclusions

We present a case of CRAO, a rare, severe, and emergent complication that occurred after an AF ablative procedure. The cause may have been thromboembolism or an air embolism.

## Author Contributions

Yasuhiro Matsuda, MD: Acquisition of data, drafting the manuscript, and revising the manuscript

Masaharu Masuda, MD, PhD: Drafting and revising the manuscript

Mitsutoshi Asai, MD, PhD: Revising the manuscript

Osamu Iida, MD: Revising the manuscript

Takashi Kanda, MD: Revising the manuscript

Toshiaki Mano, MD, PhD: Revising the manuscript

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## Figure legends

### Figure 1. Activated clotting time and heparin dose during the procedure.

We used heparin and set a target activated clotting time of 300 seconds during the procedure. Heparin was reversed with protamine at the end of the procedure.

### Figure 2. The schema of fundus examination.

A cherry red spot, which is typically observed in CRAO, was found in fundus examination.

CRAO: Central retinal artery occlusion.

**Table 1. Baseline and procedural characteristics of the patient**

Age	Gender	Type of arrhythmia	Past history	BMI (kg/m <sup>2</sup> )	LVEF (%)	LAD (mm)	Valvular heart disease	NT-Pro BNP (pg/mL)	eGFR (mL/min/1.73m <sup>2</sup> )	Procedural time (min)
79	Male	AT	HT	31	68	53	Aortic valve stenosis	1167	49	209

AT: Atrial tachycardia, HT: Hypertension, BMI: body mass index, LVEF: Left ventricular ejection fraction, LAD: Left atrial diameter, NT-proBNP: N-terminal pro-Brain natriuretic peptide, eGFR: Estimated glomerular filtration rate.

**Table 2. Risk factors for central retinal artery occlusion**

Cardiovascular disease
Atrial fibrillation
Ischemic heart disease
Hypertension
Cerebrovascular disease
Carotid artery stenosis
Stroke / Transient ischemic attack

Cardiovascular disease
Metabolic disease
Dyslipidemia
Diabetes mellitus
Renal disease
Nephrotic syndrome
Hemodialysis
Hematologic disease
Leukemia
Lymphoma
Sickle cell anemia
Others
Fabry disease
Marfan syndrome
Incontinentia pigmenti
Autoimmune disease
Smoking
Drug abuse
Oral contraceptives
Migraine

**Table 3. Common treatments for central retinal artery occlusion**

Treatment	Goal of treatment	Effectiveness
Ocular massage	Dislodgement of the embolus	One case report reported vision improvement
Paracentesis	Reduction of intraocular pressure	No evidence of benefit
Acetazolamide	Reduction of intraocular pressure	No evidence of benefit
Alprostadiol	Improvement of blood flow	Vision improvement in all 6 patients <sup>11</sup>
Nitrates	Improvement of blood flow	No evidence of benefit
Hyperbaric oxygen	Improvement of blood oxygen	No evidence of benefit
Inhalation of oxygen and carbon dioxide	Improvement of blood oxygen	No evidence of benefit
Rebreathing of expired carbon dioxide	Improvement of blood oxygen	No evidence of benefit
Heparin	Thrombolysis of the embolus	No evidence of benefit
Thrombolysis	Thrombolysis of the embolus	Effective with papaverine in 21/50 (42%)
Retrograde injection of papaverine	Prevention of vasospasm	Effective with urokinase in 21/50 (42%)
Pentoxifylline	Reduction of red blood cell rigidity	No evidence of benefit
Steroids	Reduction of retinal edema	No evidence of benefit
Laser arteriotomy and embolectomy	Lysis or dislodgement of the embolus	Two case reports reported vision improvement

