

# Branch Retinal Artery Occlusion in a Patient with Patent Foramen Ovale

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**Purpose:** To report branch retinal artery occlusion (BRAO) in a patient with patent foramen ovale (PFO).

**Case Report:** A 29-year-old female patient was referred for sudden onset superior visual field defect in her left eye. Ocular examination revealed visual acuity of 20/32 in the affected eye along with a positive relative afferent pupillary defect. A calcified white embolus was noted at the first bifurcation of the inferior temporal artery in her left eye together with mild retinal edema. With a diagnosis of BRAO, the patient received oral acetazolamide, topical timolol, ocular massage and anterior chamber paracentesis. The visual field defect partially recovered and the embolus moved to the third bifurcation level as revealed by fundus examination. An extensive workup, including neurology, rheumatology, cardiology and hematology consultation, carotid ultrasonography, transthoracic/transesophageal echocardiography and laboratory testing was performed. All results were within normal limits except for a small-sized PFO detected by transesophageal echocardiography. Low-dose aspirin therapy was initiated and over the subsequent two years, no other embolic event occurred.

**Conclusion:** The association between PFO and BRAO has not yet been reported. Intracardiac right-to-left shunting through a PFO, accentuated by Valsalva maneuver, may predispose to embolic events while the source of initial thrombosis remains unknown.

**Keywords:** Patent Foramen Ovale (PFO); Branch Retinal Artery Occlusion (BRAO); Visual Field Loss; Transesophageal Echocardiography

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## INTRODUCTION

Patent foramen ovale (PFO) is a condition in which the normal fetal communication between the right and left atria persists after birth. PFO occurs in approximately 27% of individuals undergoing routine autopsy, and is present in 25% of the general population as detected by transesophageal echocardiography (TEE).

PFO is generally a benign condition and rarely accompanied by cerebrovascular accidents such as stroke.<sup>1</sup>

Branch retinal artery occlusion (BRAO) is a vascular occlusive disorder of the eye in which the pathogenesis, clinical characteristics, and hence management differ from central retinal artery occlusion (CRAO).<sup>2</sup> A number of reports have established the association between CRAO

and PFO in some patients.<sup>4-8</sup> Herein, we report a young woman with BRAO and concomitant PFO.

## CASE REPORT

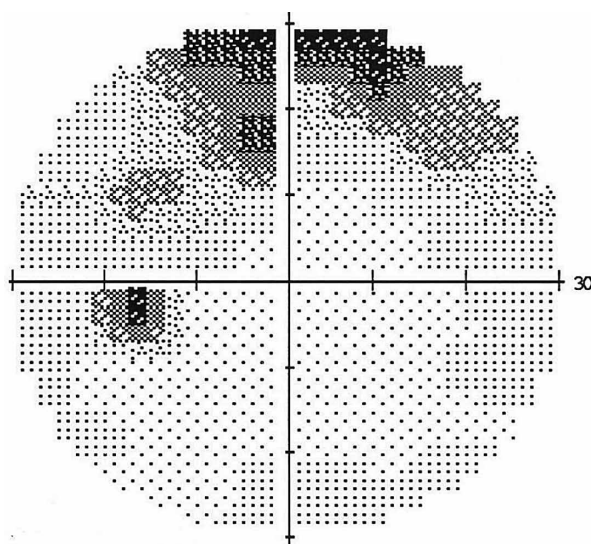
A 29-year-old woman presented with superior visual field defect of 30 minutes' duration in her left eye which had suddenly appeared after swimming.

On ocular examination, visual acuity was 20/20 and 20/32 in her right and left eyes, respectively. A weak relative afferent pupillary defect was identified on the affected side. No remarkable finding was observed in the anterior segment of either eye. Funduscopic examination revealed a calcified white embolus at the first bifurcation of the inferior temporal artery of the left retina. Except for mild retinal edema in the left eye, no other ocular abnormality was noted.

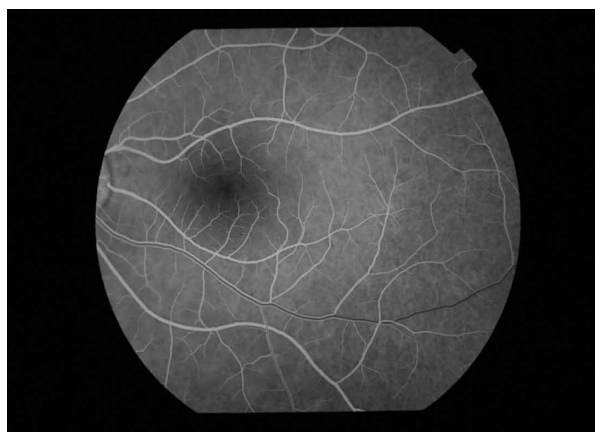
With a diagnosis of BRAO, the patient was treated with oral acetazolamide, topical timolol, ocular massage, followed by anterior chamber paracentesis. Visual field loss partially recovered within 15 minutes after initiation of treatment, meanwhile the embolus moved to the third bifurcation level.

Visual field testing (Humphrey Field Analyzer; Carl Zeiss, Jena, Germany) was performed two days after the onset of symptoms and disclosed a superior visual field defect in the left eye (Fig. 1). At the same time, fluorescein angiography depicted no embolus and delayed filling of the affected artery (Fig. 2).

An initial review of the patient's medical history revealed no systemic disease. The patient underwent an extensive diagnostic work-up consisting of neurology, rheumatology, cardiology, and hematology consultation, carotid color Doppler ultrasound evaluation, and complete laboratory testing to determine the source of embolization. These studies however, could not identify the cause of the condition. Accordingly, further echocardiographic studies were performed in search of a probable cardiac source which identified a small PFO which was 1 to 2 mm in diameter (Fig. 3A). TEE illustrated abnormal passage of agitated



**Figure 1.** 30-2 perimetry of the left eye demonstrates an arcuate defect within the superior visual field.



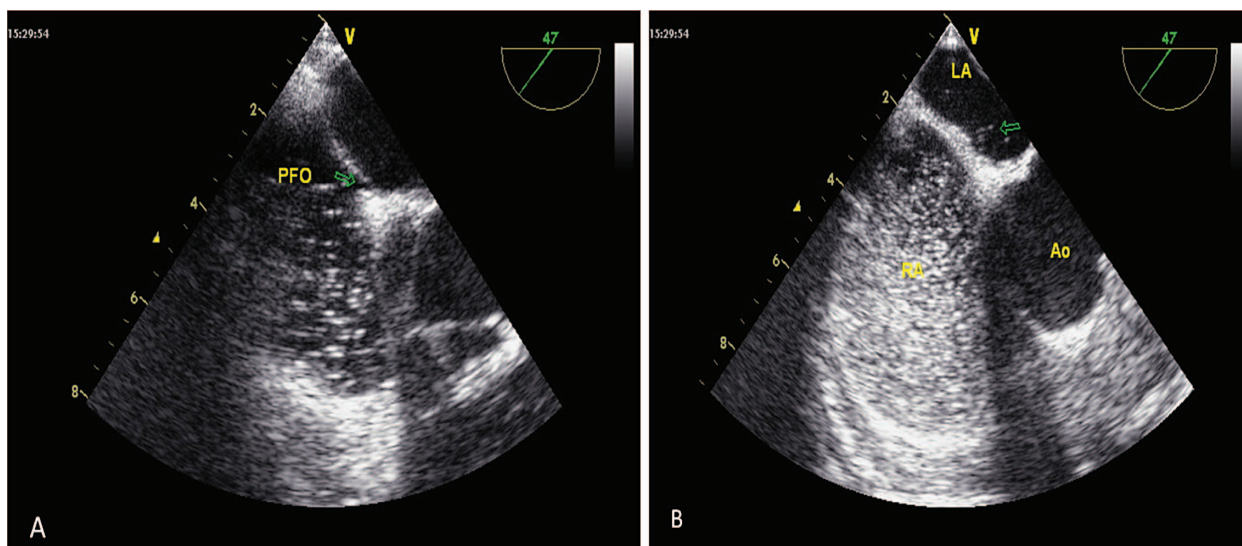
**Figure 2.** Early phase fluorescein angiography two days after the attack reveals delayed filling of the affected branch but no embolus.

saline microbubbles through the PFO; following peripheral administration of agitated saline, about 10 micro-bubbles were detected to pass from the right to the left atrium via the PFO with Valsalva maneuver (Fig. 3B). The interatrial septum was not aneurysmal and all other tests were normal.

Subsequently, the patient received long-term oral aspirin and developed no other embolic event over a two-year follow-up course.

## DISCUSSION

PFO is the persistence of a normal fetal communication between the right and left



**Figure 3.** Echocardiography of the patient shows a small patent foramen ovale (PFO, green arrow) by a transesophageal approach (A), note the passage of bubbles (green arrow) from the right atrium (RA) to the left atrium (LA) via the PFO (B).

atria which may occur in 20-34% of the population. PFOs have been associated with cryptogenic stroke, decompression illness (symptoms arising from decompression of the body, for example, in scuba divers on poorly managed ascent from depth or in aviators flying in inadequately pressurized aircrafts), systemic arterial embolism, and migraine with aura.<sup>1</sup>

PFO-related emboli have already been reported in young adults.<sup>3</sup> The source of the emboli is often not found, but they are usually attributed to occult thrombosis. Nakagawa et al presented a case of bilateral CRAO in a patient with PFO and deep venous thrombosis.<sup>4</sup> Clifford et al described CRAO and ischemic optic neuropathy in a young patient with PFO.<sup>5</sup> Likewise, Gabrielian et al reported CRAO in a healthy 17-year-old male patient with sudden, painless vision loss. Although routine diagnostic work-up including transthoracic echocardiography (TTE) were negative, TEE could establish a PFO.<sup>6</sup> In another report, Sheth et al described a man aged 18 years with a hemiretinal artery occlusion presenting with sudden onset visual loss on the left side. Subsequently TEE could demonstrate a PFO which was later closed surgically.<sup>7</sup>

Mohamed et al reported a 24-year-old man with history of migraine and visual auras

from the age of 16 years who presented with sudden-onset painless scotoma in his right eye.<sup>8</sup> He was diagnosed based on localized retinal artery obstruction and cotton-wool spots. Comprehensive physical examination, medical history and initial laboratory tests were normal. A contrast echocardiogram showed spontaneous right to left shunting at the atrial level and a PFO.<sup>8</sup>

Our patient's history was remarkable for engagement in swimming prior to the onset of symptoms. This exertion may have resulted in a paradoxical embolus and BRAO.

Chen et al reported a four-fold increased incidence of PFO in patients with ischemic events compared with controls, and greatly increased sensitivity for detecting PFO with TEE as compared to TTE.<sup>9</sup> Kramer et al found TEE to have higher yield than TTE in the evaluation of patients with CRAO.<sup>10</sup>

PFO is usually asymptomatic, however it should be considered among the possible causes of BRAO/CRAO. In younger patients, in particular, with no other risk factors, normal TTE and negative corresponding work-up, TEE can be a more sensitive means of diagnosis.

#### Conflicts of Interest

None.

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