

Exacerbation of Amaurosis Fugax Syndrome Following a Photographic Trip Led to the Diagnosis of Patent Foramen Ovale: An Adventure from Equatorial Forest to the Cardiac Lab

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Abstract:

An amateur photographer went to the equatorial rain forest to take photographs of birds of paradise. On his return he developed a severe exacerbation of his previous mild transient vision loss. It took a transesophageal echocardiogram to reveal a patent foramen ovale which probably led to paradoxical embolism that caused his symptoms. Percutaneous, transcatheter closure of the defect was carried out and he was relieved of his symptoms with no relapse up to three years.

Keywords: amaurosis fugax; transient vision loss; patent foramen ovale; pfo-closure

Introduction

One of the commonest hobbies today is photography and one of the ultimate frontiers for the photographer is the equatorial rain forest. It boasts of a huge reserve of flora and fauna rich in quantity and diversity. As an example, the birds of paradise are unique to the forest of Papua, New Guinea. These birds present with such stark beauty that the name paradise was aptly applied to them at first sight, and subsequently recycled to name a flower of similar, but less unique, features. It was in the quest to explore these birds that a patient went on a photographic trip to Papua. As expected, he was rewarded with a rich collection of pictures of these exotic birds. Unexpectedly, the trip also led to the discovery and solution of a long hidden medical problem which had been troubling him for many years.

Case report

The patient is a middle age male, an interior architect by profession and an ardent armature photographer of birds in wild life. He has a strong family history of cancer. Both parents had lung cancer, and in addition, breast cancer also affected his mother, a maternal aunt and a sister. Remarkably, with zealous and timely anticancer treatment they all had the cancer under control except his aunt. Consequently, he went for medical checkup from time to time but usually end up with negative findings. In 2021, he presented with transient loss of vision for about 10-20 seconds after taking a swim in the pool and sipping half a glass of beer. The vision was blurred suddenly without any warning sign. On further questioning, he remembered he had similar attacks dating back to the 1990s, i.e. when he was in his 30s. It was

always brief and bilateral, no flashing of light or colour, no scotoma, tunnel vision, doughnut vision (central anopia), spontaneously resolving within 10 to 20 seconds and never amounting to complete blindness. There were no concurrent or residual epilepsy or loss of motor or sensory function and no hint of platypnea-orthodeoxia (having to lie flat to catch one's breath). The visual loss remained always just blurring, never amounting to total blindness. The duration of remission was variable. Relapse usually came within a matter of weeks or months, but never delayed over two years. An ophthalmologist, after a detailed examination, including ophthalmoscopy, found no abnormalities in his eyes. Based on the negative finding he suggested the diagnosis of visual aura of migraine, although the loss of vision was never associated with headache or pain in the eye.

A neurologist was consulted but no abnormal neurological signs were found which suggested the differential diagnosis of TIA (transient ischemic attack), with the differential diagnosis of aura of atypical migraine, followed by occipital seizure. To further investigate the likelihood of TIA, the patient had an MRI stroke package which showed completely normal common carotid arteries, vertebral arteries, internal carotid arteries, basilar artery, circle of Willis, and all the major arteries supplying the brain. It did show one tiny focus of old haemorrhage of the right occipital lobe and another small infarct in the left cerebellar hemisphere. A large left vertebral artery was found to arise directly from the aorta rather than from the left subclavian artery. The radiologist considered this a normal variant, and the two small brain lesions

likely due to small vessel disease. In any case, the carotid origin of emboli to the brain was ruled out, but the cardiac origin of emboli remained unresolved. A cardiologist examined the patient, studied the 12 Lead ECG, followed by a thoracic echocardiogram, and found no atrial fibrillation, intracardiac clots or cardiac wall or septal abnormalities to account for emboli generation. At this stage the consensus of both the patient and the management team was that the patient was suffering from some form of TVL (transient vision loss) or, in Greek, amaurosis fugax. The most likely cause was thought to be related to computer fatigue, as it seemed to be associated with the intense usage of computer graphics and designs. As his symptoms were minimal and short-lived with spontaneous complete recovery, and as he never drove a car or use formidable machineries, the chance of injury to

himself or others were considered negligible. It might not be in his best interest to pursue further intensive and invasive investigations. He was put on low dose aspirin, warned against working on machineries that demanded uninterrupted intense visual concentration such as driving, warned against swimming alone and put on close observation. Over the next two years the patient’s medical condition remained stable. Then he resumed his hobby of wild bird photography. He had taken similar trips before, traveling to the heights of the Himalayas and the freezing arctic zone of Scandinavia and had never encountered any problem. This time, his destination was the equatorial rain forest of Papua, New Guinea – his target, the famous birds of paradise. And he was not disappointed. The sight of such birds was literally “breath-taking”. (Figure 1)



Figure 1: A bird of paradise photographed by the patient in the equatorial forest of Papua, New Guinea.

However, following this adventure, the patient suffered another episode of transient visual loss, this time of longer duration and more severe than before, lasting over 30 seconds and exhibiting a greater disability. The patient made a spontaneous recovery and consulted another ophthalmologist, who made the diagnosis of amaurosis fugax and suggested the additional investigation of ultrasonography of the carotid arteries. This was duly carried out without significant findings of wall-thickening, mural thrombi,

atherosclerotic ulceration or aneurysmal dilatation. (Figure 2) Repeat of the MRI stroke package (Figure 3) and echocardiogram showed no change from the findings two years ago. Our consensus now is to go one step further and do a trans-esophageal echocardiogram (TEE) with air microbubbles as contrast. This was carried out under general anaesthesia the next day, with the finding of a patent foramen ovale which was successfully closed with a percutaneous transcatheter-deployed occluding device. (Figure 5)



Carotid arteries left anterior oblique view

Figure 2: MR angiogram showing patient’s almost unblemished arterial supply to the brain.

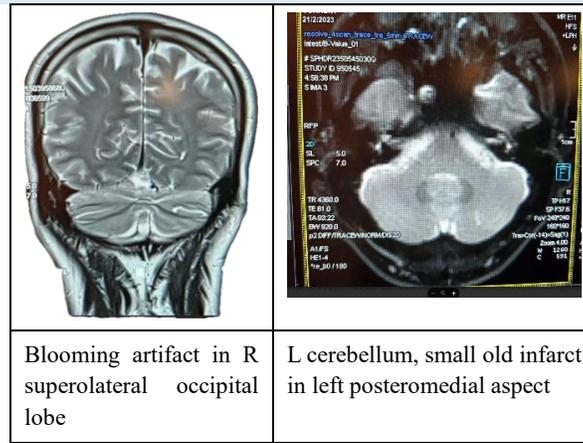


Figure 3: MRI Brain

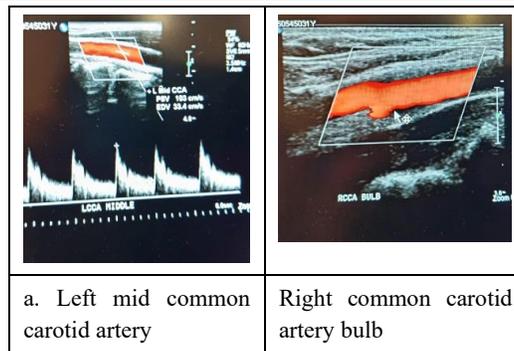


Figure 4: Ultrasonographic study of carotid arterial system showing no significant disease

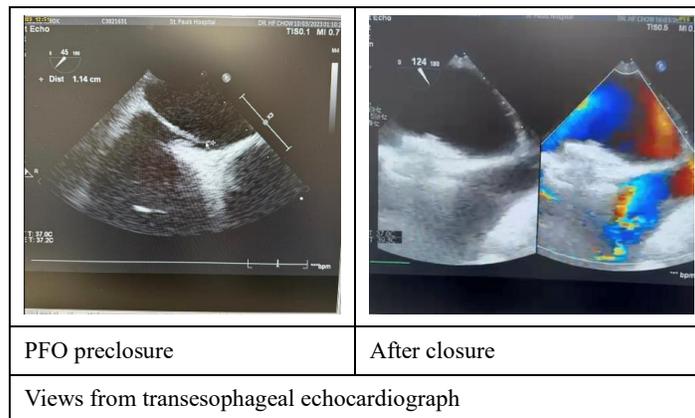


Figure 5: Closure of Patent Foramen Ovale (PFO)

The patient tolerated the procedure well and was discharged home on low dose aspirin the next day. He was regularly followed up, partly by physical clinic attendance and partly via tele-medicine. Eighteen months later, he had a minor episode of nasal bleeding and the aspirin was stopped altogether. It is now almost three years after the closure of his foramen ovale, and so far, the transient visual blurring never recurred

Discussion

This case raised a number of problems involving multiple specialties including ophthalmology, neurology, cardiovascular radiological and ultrasonic imaging, and, for the ultimate definitive correction, interventional cardiology. The following is a brief review of the issues involved and with clarification where necessary.

Transient vision loss (TVL) and Amaurosis fugax (AF)

Theoretically, the Greek term amaurosis fugax (AF) should mean the same as loss of vision of a transient nature. However, there has been lack of uniformity on the concept and terminology. Some clinicians would confine the term AF to the unilateral transient visual loss, while others would advocate AF to include or even to be restricted to bilateral transient visual loss. The most reasonable approach seems to be that of Ferose et al. (2024)1. Their concept may be summarized as follows. Transient vision loss may be unilateral or bilateral, lasting seconds or minutes. Its cause ranges from migraine’s aura through optic nerve neuritis and neuropathy to a wide variety of transient ischemia from minor embolism. Unilateral vision loss is usually related to a pre-chiasmal pathology, but bilateral vision is usually related to a post-chiasmal condition. Our patient is a good example of the approach of

Feroze, his transient vision loss is bilateral and his only positive findings are in the post chiasma zone of the brain. For management, a multi-discipline team is required. To reconcile the different usage of the Greek term amaurosis fugax we suggest adding the term “syndrome” to render it more inclusive and applicable to patients with bilateral loss of vision like our case.

Meniere syndrome

Transient vision loss was reported as an aura of Meniere syndrome as early as the late 19th century², and it remains an important entity in the differential diagnosis. Its frequency has probably been exaggerated because with modern technology, such as MRI and antibody detection, many of the headaches and ocular pain have been traced to neuritis and autoimmune reactions involving the optic tract, e.g. MOGAD (Myelin Oligodendrocyte Glycoprotein Antibody-associated Disease)³, further reducing the number of transient losses of vision ascribed to migraine aura.

Transient ischemic attack (TIA) – emboli of arterial origin

TIA is regarded as a common and important form of cerebral vascular episodes. It comprises a variety of emboli derived from mural thrombi and ruptured atheroma plaques on the interior of the atheromatous aortic arch, the common and internal carotid arteries and the carotid sinuses. When the embolization is confined to a single retinal artery the condition conforms with the diagnosis of amaurosis fugax in the narrowest and strictest sense. When the embolization extends beyond the retinal artery, and involves more areas of the brain the condition develops into the picture of TIA from which the full picture of the ischemic stroke may ultimately arise, accompanied by its well-known morbidity and mortality. Therefore, all TIAs should be regarded as medical emergencies. It is crucial for the clinician to make the correct diagnosis with the relevant MRI and ultrasonic studies and instigate antiplatelet therapy or even thrombo-embolectomy or intravascular stenting [4].

Cardiac cause – emboli of cardiac origin related to atrial fibrillation (AFib)

Atrial fibrillation (AFib) is a well-established cause of cerebral embolism [5], but its relation to transient loss of vision is less studied. One report finds that 9% of patients with monocular ischemic loss of vision had AFib [6], but concedes this could be an under estimation because not all cases of visual loss had gone through detailed AFib screening. The fact remains that the majority of AFib-related cerebral vascular events present as generalized ischemic stroke rather than the more specific TIA or amaurosis fugax [6].

Patent foramen ovale and transient vision loss

Patent foramen ovale (PFO) is one of the commonest cardiac conditions affecting both sexes in about 25% of all races. During intra-uterine life, the structure and function of the foramen is to allow blood to pass from the high-pressure right atrium to the low-pressure left atrium. After birth the pressure gradients are reversed. With the cut off of the umbilical vessels and the expansion of the lungs the pressure of the left atrium is now higher than the right atrium. But, as the blood follows the new reversed pressure gradient and flows from left to right, it forces the flappy septum primum against the thicker and stabler septum secundum resulting in closure of the foramen. After a period, varying from days to months, but usually within the first year, the majority of foramen ovale would seal up by structural fusion, while the remaining approximately 25% would persist as patent foramen ovale [7].

Most PFOs will remain asymptomatic throughout life. Rarely, however, there might be occasions when blood would flow through the PFO from the right to the left atrium. The cause of such revival of embryonic circulatory

blood flow is complex and not completely understood. In mitral atresia, it might be the means to sustain life, and needs to be enlarged for improving the child’s chance of survival. Likewise, in newborns with transposition of great vessels, balloon enlargement of the foramen ovale might be a useful palliative measure pending a total anatomical correction. The fact remains that a right to left interatrial shunt is not without its drawbacks. Apart from causing some desaturation of the arterial blood, it permits the passage of thrombi formed in the peripheral circulation, most commonly in the veins of the lower limbs, to bypass the filtering function of the lungs to enter the systemic circulation and affect important and sensitive organs⁹, top of the list being the eyes and the brain, resulting in clinical pictures such as amaurosis fugax, as illustrated in the presented case.

In the past, management of PFOs was always conservative because the complications were considered rare and trivial to justify open surgical repair. The patient was usually put on antiplatelet therapy (which was not without its own risk). With the development of percutaneous transcatheter closure technology, the treatment scenario has radically altered and we are able to offer a more acceptable choice of definitive interventional treatment with the prospect of weaning off the anti-platelet medication and improving the patient’s quality of life. Indeed, as from ten years ago, the technique of transcatheter PFO closure has been firmly established [10].

Conclusion

We have reported a case of bilateral transient loss of vision associated with patent foramen ovale and reviewed the various factors involved in the pathogenesis. However, more questions have arisen from such a review. What are the criteria to guide the front-line clinician to go deeper into elaborate investigations? Is there any simple inexpensive test to screen the patients on a point-of-care basis to pick out the more likely PFO-positive ones? And having made the diagnosis what are the criteria for sending the patient to the cardiac lab for closure?

Acknowledgement

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Declarations

All authors took an active part in the management of this patient and each author declares no conflict of interest in preparing this article.

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