

Patent Foramen Ovale (PFO) and Association with Cryptogenic Stroke in Young Patients

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Abstract

Patent foramen ovale (PFO) is a flaplike opening between the atrial septa primum and secundum at the location of the fossa ovalis that persists after age 1 year. With increasing evidence being found that PFO is the culprit in paradoxical embolic events. By demonstrating our case report, we want to highlight the relevant aspects of the patent foramen ovale and the association of cryptogenic stroke, its clinical presentation, diagnosis and management.

Keywords: Ischemic Stroke; Cryptogenic Strokes; Interatrial Septal Abnormalities.

Introduction

Patent foramen ovale, a small communication between the left and right atria, is considered to be a risk factor for cerebral embolism. On an average 40% of ischemic strokes does not have clearly definable etiology hence they are termed cryptogenic strokes.

If the possible causes of an ischemic event is not discovered it is categorized as cryptogenic. Even though there are different possible causes of stroke, approximately 20% are of cardioembolic origin [1]. It is common between young adults to have strokes of unknown causes. This accounts for 10-40% depending on the population [2].

The aim of this report is to discuss the relevant aspects of the patent foramen ovale and the cryptogenic strokes, its clinical presentation, diagnosis and management.

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Case Report

A 39-year Caucasian old woman was hospitalized in the Emergency Department with 20-hours anamnesis of aphasia, sensory disturbances in the right arm, leg and right side of the tongue. No motor dysfunction. She was afebrile (37.2°) with oxygen saturation 99% with normal circulatory parameters. Laboratory test revealed normal haematology, mild increase in alanine transaminase 85 U/L (Reference 10-70 U/l), normal white blood cell count 4.7, normal C-reactive protein level of 2 mg/L (Reference < 6mg/l). She was examined by an immediate CT cerebrum which was normal without acute ischaemic or haemorrhagic changes. Carotid and vertebral Doppler scans were normal Transthoracic echocardiogram performed on the next day which was normal. According to the clinical presentation and with suspicion of stroke, she was started with dual antiplatelet treatment. The patient continued to show symptoms for several days and therefore supplied by a MR cerebrum which showed signs of 3 small sub-acute ischaemic lesions in the left cortical area supplied by arteria cerebri media (Figure 1).

During admission the patient was also checked for antithrombin III activity, protein C and S antigen, plasma homocysteine, antiphospholipid antibody panel, determination of antinuclear factor and anti-DNA antibodies, and HIV antibodies search which revealed normal. The contrast-enhanced transeso-

phageal echocardiography showed for the presence of the PFO. Further investigation excluded atria septum aneurysm.

The patient was successfully operated with Amplatzer PFO occluder under continuous monitoring through the transesophageal echocardiography. After percutaneous closure, the patient received clopidogrel (75 mg/day) for 6 months and was followed up within 6 months. Her symptoms had not worsened.

Discussion

Generally speaking there are two kinds of holes in the heart, one is called Atrial Septal Defect (ASD) and the other is Patent Foramen ovale (PFO). Although both are holes in the wall of tissue (septum) between the atrium, their causes are quite different [3,4]. PFOs can only occur after birth when the foramen ovale fails to close. The foramen ovale is a hole in the wall between the left and right atria of every human fetus. This hole allows blood to bypass the fetal lungs, which cannot work until they are exposed to air [5]. When a newborn enters the world and takes its first breath, the foramen ovale closes, and within a few months it has sealed completely in about 75 percent of us [6]. When it remains open, it is called a patent foramen ovale, patent meaning open. For the vast majority of the millions of people with a PFO, it is not a problem, even though blood is leaking from the right atrium to the left. Problems can arise when that blood contains a blood clot and give which gives rise to a stroke symptoms especially in their younger ages. Cardiologists estimate that 70,000 to 100,000 strokes per year in the United States are secondary to paradoxical embolism via a PFO [7,8]. Depending on the criteria used for diagnosis and the technology used in cardiac assessment, the prevalence of PFO in the healthy population is approximately 20-25% [9]. Thus, detection of a PFO during evaluation of a patient with a stroke is not surprising, and the frequency of PFO detection in these patients can be as high as 40-45% [9]. This frequency of detection is especially high among people without any other obvious explanation for the stroke. Overell et al. [10] concluded from a meta-analysis of several studies that the relative risk of stroke compared to non-stroke controls increased by a factor of 1.83 if a PFO was present.

Conclusion

Lifelong anticoagulation or antiplatelet therapy along with surgical or endovascular closure could be the best option. Surgical or endovascular closure could be the best option in very young patients if we

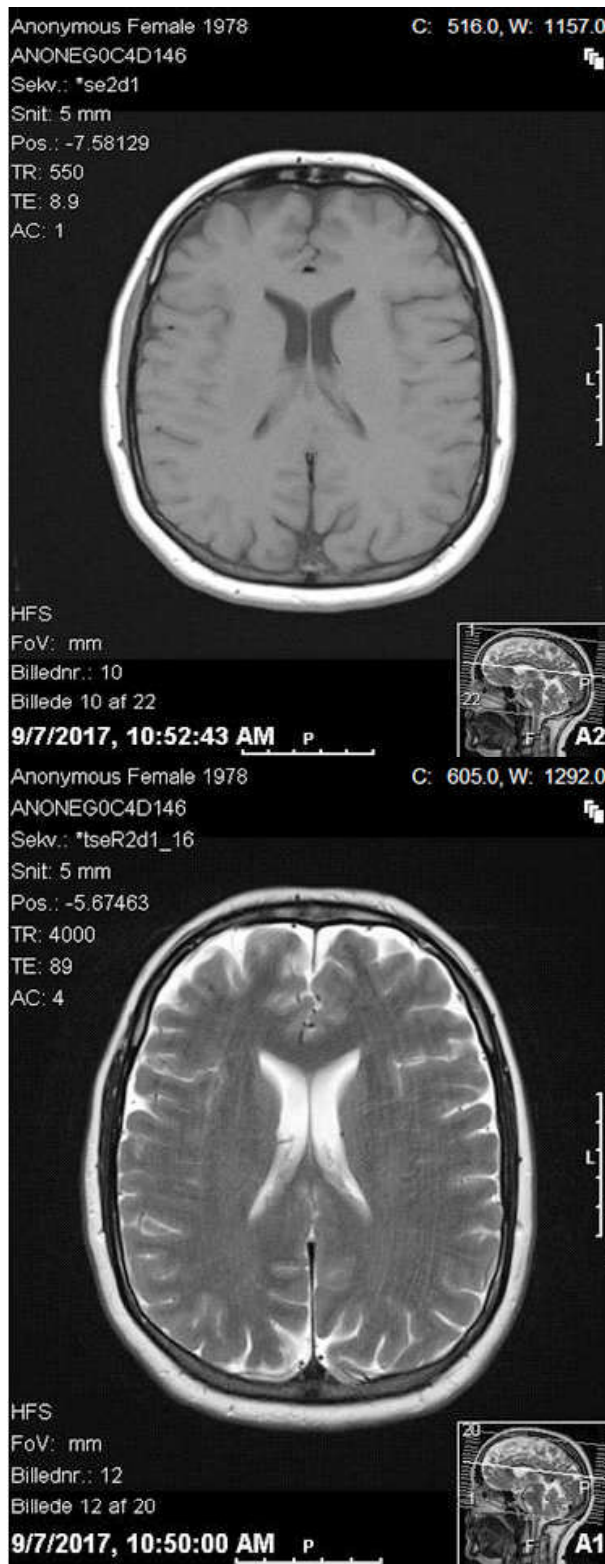


Fig. 1: MRI scan of the brain showing signs of ischaemic changes in the cortex and in basal ganglia

consider the risks of a lifelong anticoagulation or antiplatelet therapy [11]. Mechanical closure of the PFO has been proposed as the definite way to prevent recurrent paradoxical embolism. With the advances in transcatheter endovascular placement of closure devices, the role of major cardiac surgery seems to be diminishing [11]. The procedure has several advantages as it can be done on an outpatient basis using local anesthesia and usually takes less than 30 minutes. The PFO can be closed completely in approximately 95% of patients [7].

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Contribution by authors

AN: Involved in treatment, patient follow up and wrote the manuscript.

SJ: Involved in patient follow up and contributed in the manuscript.

Conflict of Interest

The authors declare that there is no conflict of interest regarding the publication of this article.

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Ethical aspects and consent

Written consent for case report publication obtained from the patient in December 2017.

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