

Patent Foramen Ovale Device Closure in Nepali Migrant Worker Performed at Shahid Gangalal National Heart Centre, Kathmandu, Nepal

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ABSTRACT

Patent Foramen Ovale is highly prevalence in the general population and in patients with cryptogenic stroke. Patent Foramen Ovale device closure emerged as a practical solution to the problem of Patent Foramen Ovale-mediated illness. In patient with previous history of stroke and absence of other embolic etiologies with shunt across the Patent Foramen Ovale warrants closure. We highlight a Patent Foramen Ovale device closure of a migrant worker in Shahid Gangalal National Heart Centre.

Keywords: patent foramen ovale (PFO); device closure; Nepali migrant worker; Shahid Gangalal National Heart Centre; Kathmandu; Nepal.

INTRODUCTION

Patent Foramen Ovale (PFO) is a congenital defect in the septum separating the two atrial chambers. PFO provides communication between the atrial chambers of the heart through fossa ovalis on the right side and the ostium secundum on the left side. Septum primum acts as a one-way valve permitting blood flow from the right to left atria, bypassing the lungs.¹ Normally, this septum remains open before birth and closes with the first breath due to increase left sided pressure. Anatomical closure usually occurs by the age of 2 years.² For most people, a PFO remains undetected or is found incidentally during cardiac examinations. However, some PFOs can cause transient or continuous right-to-left shunt, which may allow bloodborne materials, such as thrombi, vasoactive substances (e.g., serotonin, prostaglandins, or nitric oxide), or nitrogen bubbles to enter the systemic circulation.³ PFO is present in 15-35% of the adults.⁴ Autopsy studies indicate

that the prevalence of PFO is 27% in the general population.²

PFO has been identified as a potential cause of cryptogenic stroke, particularly in children and young adults without risk factors for athero-embolic stroke.⁵ Cryptogenic stroke accounts for 25-40% of all ischemic stroke and poses diagnostic and therapeutic challenges due to the lack of an identifiable cause.⁶ Among young patients with cryptogenic stroke, the prevalence of PFO can be as high as 50%.⁷ The role of PFO in cryptogenic stroke may involve promoting clot formation or acting as a pathway for paradoxical embolism, where a thrombus bypasses lung filtration and moves from venous to arterial circulation through the foramen ovale, potentially resulting in cerebral ischemia. However, establishing a direct causal link between PFO and stroke can be complex, and the evidence is often speculative and inadequately documented, requiring thorough diagnostic evaluation.⁸

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PFO device closure emerged as a practical solution to the problem of PFO-mediated illness. Recent randomized control trials have demonstrated that PFO device closure in patients with cryptogenic stroke is associated with reduced rates of recurrent stroke in carefully selected patients.⁹ This resulted in a dramatic increase in the number of PFO device closure procedures performed worldwide, primarily for the indication of cryptogenic stroke, with high procedural success and low complication rates.¹⁰ International labour migrant workers from Nepal have grown substantially in the recent decades. Nepali migrant workers, who appear healthy and show no signs of illness are suddenly dying during sleep. Most of these cases are reported as ‘cardiovascular deaths’ in a post-mortem report but comprehensive post-mortems are not performed so the reports do not specify the exact cause of death (for e.g., myocardial infarction, stroke for cardiovascular related deaths).¹¹

In this case report, we highlight a PFO device closure for a migrant worker at Shahid Gangalal National heart Centre. He developed stroke in a foreign country, was managed effectively there, and returned to his country for PFO closure.

CASE PRESENTATION

A 43-year-old male presented to a hospital in Qatar initially with left facial drop, dizziness, deviation of the mouth, slurred speech and left upper limb weakness and numbness. A CT head was done, which was suggestive of Right Middle Cerebral Artery territory infarct. CT brain perfusion revealed a mismatched segmental perfusion defect noted in the right parietal area with penumbra. CT intracranial angiogram revealed bilateral common carotid, internal carotid, anterior cerebral and middle cerebral arteries are normal in course and caliber with good contrast enhancement. Bilateral vertebral, basilar and posterior cerebral arteries are patent. MRI showed small, predominantly cortical recent infarctions at the right frontotemporal parietal region with early hemorrhagic transformation. ECG was a normal sinus rhythm. No sonological evidence

of DVT on either side. Echo revealed evidence of a small PFO. He was treated with Aspirin 100mg and atorvastatin 40mg once daily and was planned for PFO device closure. But the patient came to Shahid Gangalal National Heart Centre for PFO closure.

He denied a personal or family history of arrhythmias, stroke, blood clots, coronary artery disease, hypertension, or diabetes. His vitals at our center were stable. His Risk of Paradoxical Embolism (RoPE) score was 7. A 12-lead electrocardiogram recorded at our institution revealed sinus rhythm with Right bundle branch block as shown in Figure 1.

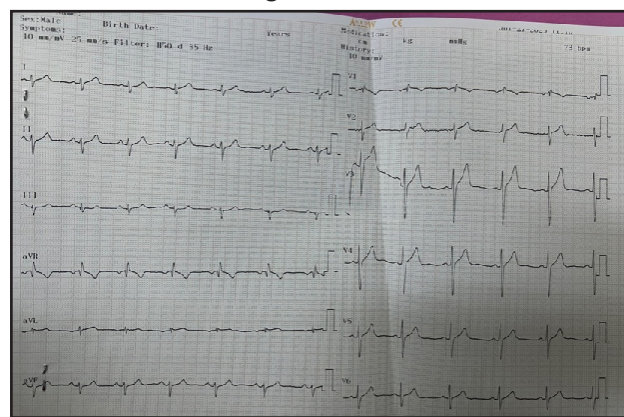


Figure 1. ECG.

Transthoracic echocardiography (TTE) showed Normal finding. A transesophageal echocardiogram (TEE) revealed 2.8 mm PFO as shown in Figure 2. There was no significant valvular abnormalities and no left atrial appendage thrombus.

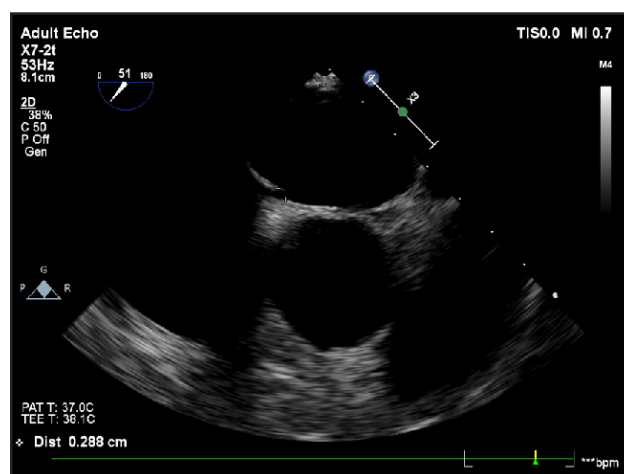


Figure 2. TEE.

He was informed about the risk and benefit of PFO closure. After proper consent from the patient and his relatives, we planned for PFO closure. The procedure was performed under local anesthesia and transthoracic echocardiogram guidance. Thrombus formation during the procedure was prevented using an intravenous bolus of unfractionated heparin (100 UI/kg). Subacute bacterial endocarditis prophylaxis was done using cefazolin (1 gram intravenously) at the start of the procedure and 2 hours later. Venous access was gained via the right femoral vein. PFO was crossed with a Terumo wire and a 6F multi-purpose catheter under fluoroscopic guidance. Terumo wire was removed, and a 0.035" × 260 cm exchange stiff wire was positioned in the left upper pulmonary vein. PFO closure was performed using an 8-F Amplatzer delivery sheath positioned in the left upper pulmonary vein. No balloon-sizing catheter was used. A 25 mm Amplatzer Talisman PFO Occluder was deployed. Residual shunt was evaluated using a transthoracic echocardiogram, shown in Fig. 4, and also with a contrast study

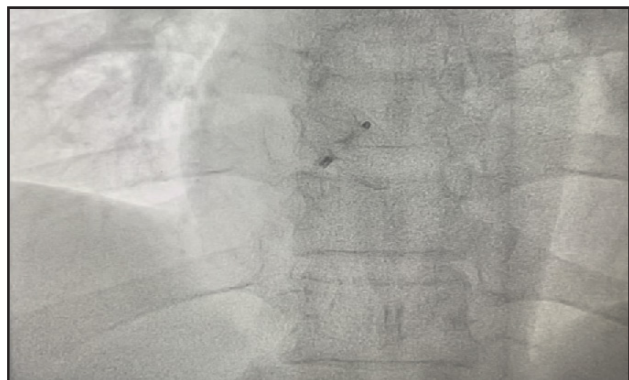


Figure 3. PFO device in fluoro.

before the release of the device.

DISCUSSION

Most patients with a PFO are asymptomatic, however, the association with cardiac and medical manifestations is well documented. Given the high prevalence of PFO in the general population and the low risk of stroke related to PFO, there has been a degree of uncertainty about the causal relationship between a PFO and an embolic-appearing ischemic

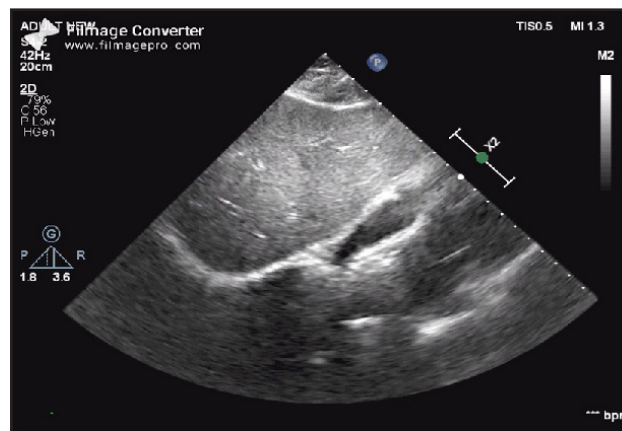


Figure 4. PFO device in echo.

stroke with no other evident stroke mechanism despite a comprehensive evaluation.¹² The Northern Manhattan Study (NOMAS) was a multiethnic prospective cohort of Northern Manhattan which revealed that approximately one-third of PFOs detected were incidental findings and not associated with an increased risk of stroke.¹³

A case-control trial revealed that PFO was prevalent in cryptogenic stroke, particularly in younger patients (age < 60).¹⁴ This called for further objective analysis, and different possibilities that stroke was causally related to PFO were ultimately suggested by RoPE score and modified PFO-Associated Stroke Causal Likelihood (PASCAL) classification.¹⁵ These tools can be used to estimate the probability of whether a PFO is associated with cryptogenic stroke as it analyzes parameters such as age, risk factors, shunt size, imaging results, etc. To generalize, younger patients (age < 60) with no history of hypertension, diabetes, stroke, or transient ischemic attacks would have a high RoPE score raising clinical suspicion for PFO-associated stroke. Further management should be individualized for these patients and medical therapy with or without potential percutaneous intervention should be considered.¹⁶

Clinical trials have shown that PFO device closure combined with long-term medical antiplatelet therapy significantly reduces the recurrence of ischemic stroke compared to long-term medical antithrombotic therapy alone (antiplatelet or anticoagulant). A meta-analysis demonstrated that device closure reduced the rate of recurrent ischemic

stroke with a hazard ratio (HR) of 0.30 (95% CI, 0.13-0.68; p-value=0.004) compared to medical therapy alone.¹⁷ The Society of Cardiovascular Angiography and Intervention (SCAI) has provided contemporary guidelines for PFO management as an evidence-based collection of recommendations supported by data pooled from retrospective studies mentioned above. The SCAI guidelines address patient selection and outline current recommendations to support PFO closure in PFO-associated stroke.¹⁰ In our patient, high RoPE score, large PFO, shunt across the PFO in

TEE, history of stroke and absence of other embolic etiologies warrants PFO closure.

CONCLUSION

Stroke/TIA in a patient without embolic etiologies needs evaluation of probable PFO. In patient with a previous history of stroke and absence of other embolic etiologies with a shunt across the PFO warrants closure; however, proper patient selection is important.

Conflict of interest: None

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