# Papillary fibroelastoma as a rare cause of recurrent strokes and myocardial infarction

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A 40-year-old male patient after two ischemic strokes was referred to our outpatient cardiac center for extended assessment. Previous evaluation showed no significant causes of stroke. It excluded proximal atherosclerosis, arteritis, arterial dissection, or atrial fibrillation (AF). A transthoracic echocardiogram demonstrated no intraventricular thrombus or valvular/regional wall motion dysfunction. Thrombophilia testing was negative; the patient was checked for lupus anticoagulant, anticardiolipin antibodies, anti-β2 glycoprotein I antibodies, antithrombin deficiency, protein C or S deficiency, factor V Leiden, prothrombin 20210 mutation, and hyperhomocysteinemia. Screening for connective tissue disease showed no inflammatory markers elevation, presence of ANA, cANCA, or proteinogram abnormalities. Cancer of other organs and infection were excluded. Apart from mildly elevated troponin concentrations in the acute peri-stroke period, the patient's blood tests were normal. ECG showed sinus bradycardia and non-specific ST-T changes in the inferior leads.

To search for minor-risk cardioembolic sources, we performed transesophageal echocardiography (TEE), which showed no cardiac thrombi or patent foramen ovale. It demonstrated a small, mobile, pedunculated mass arising from the anterior mitral leaflet (A1 segment), with a maximum size of 4 mm (Figure 1A), considered in the differential diagnosis of infective endocarditis, myxoma, or papillary fibroelastoma (PFE). The patient was assessed as eligible for surgery. Coronary computed tomography angiography (CCTA) showed normal coronary arteries with no lesions and confirmed the suspicious formation attached to the mitral valve. We repeated prolonged ECG monitoring for 72 hours, which again did not show any AF episodes, but showed episodes of second- and third-degree atrioventricular blocks with a spectacular 17-second pause during blood collection, resulting in syncope. The Heart Team planned cardioneuroablation after the patient's surgery. To check for potential causes of conduction disorders, we performed cardiac magnetic resonance imaging (MRI). Unexpectedly it visualized subendocardial areas of late gadolinium enhancement (LGE) in the inferior wall, indicating an old myocardial infarction [1]. Considering that coronary arteries were normal, the presumable cause of myocardial infarction must have been embolic.

Finally, our patient was referred for surgical resection of the mitral lesion. He underwent surgery in another center according to his wish. The surgical procedure was performed using the robotic da Vinci Surgical System. We present intraoperative images (Figure 1B–C). Acetylsalicylic acid 75 mg was used in secondary thromboembolic prevention. Histopathological analysis of the tumor concluded it was a PFE. Since the excision, our patient has had no further thromboembolic complications so far. Further decisions regarding treatment of conduction disorders are at the discretion of the center chosen by the patient.

The presented case illustrates how challenging the assessment of stroke causes may be. In approximately one-fourth of strokes, their causes cannot be determined, thus many strokes are cryptogenic [2]. We present a case of a PFE causing two embolic strokes and a myocardial infarction. Although primary cardiac tumors are rare (0.02% prevalence) [3],

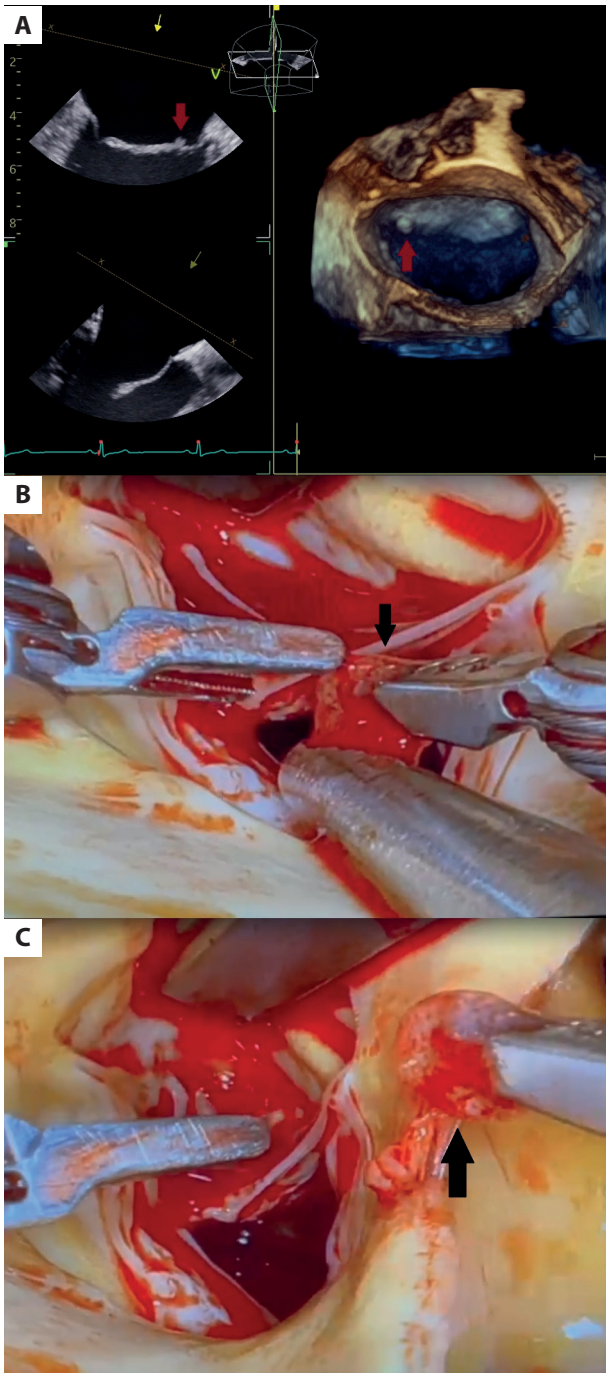


**Figure 1. A.** Transesophageal echocardiography showing pedunculated mass arising from the anterior mitral leaflet (A1 segment). **B, C.** Intraoperative images

with PFE as one of the most common, we should remember the importance of a wide diagnostic approach when determining the cause of thromboembolic events, especially in cases of negative workup [4]. Secondary prevention with acetylsalicylic acid, $P2Y_{12}$ inhibitors, or VKA/direct oral anticoagulants might not always be sufficient.

## *Article information*

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