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A Rare Case of Ischaemic Stroke in A Young Man with Mitral Valve Prolapse

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ABSTRACT

Mitral valve prolapse is a common valvular abnormality. It affects 2-3% of the general population. It often runs a benign course and the majority of patients are asymptomatic. Cardiac sources of thrombi account for about 20% of ischaemic strokes. There is lingering uncertainty regarding the causal link between mitral valve prolapse and ischaemic stroke. It has been suggested that changes on the surface of the valvular leaflets trigger platelet adherence and aggregation, leading to formation of platelet-fibrin thrombus which can embolize to the cerebral arteries, causing an ischaemic stroke. We herein report a case of a 25 year old young man who presented with sudden onset of left-sided weakness. Cardiac auscultation was notable for a late systolic murmur preceded by a midsystolic click at the apex. Transthoracic echocardiography showed an elongated and thickened anterior mitral valve leaflet which prolapsed into the left atrium during systole. Computed tomography scan of the brain revealed infarcts in the right frontal lobe and the right insular cortex. A diagnosis of myxomatous mitral valve prolapse complicated by ischaemic stroke was made and the patient was accordingly started on anticoagulant therapy with warfarin.

Keywords

Mitral, Valve, Myxomatous, Stroke.

Introduction

Mitral valve prolapse refers to the superior displacement of one or both mitral valve leaflets into the left atrium during ventricular systole [1]. It is also known as systolic click-murmur syndrome, Barlow's Syndrome or floppy valve syndrome [1]. The prevalence of mitral valve prolapse is estimated to be about 2-3% of the general population [2]. It is twice as common in females as in males and has been observed in a wide age range, particularly between the ages of 14 and 30 years [3]. Its occurrence may be sporadic or familial [4]. It is frequently associated with heritable connective tissue disorders such as Marfan's Syndrome, Ehlers-Danlos Syndrome and osteogenesis imperfecta [5].

Mitral valve prolapses results mainly from myxomatous degeneration of the mitral valve leaflets. This gives rise to anatomic abnormalities which result in the failure of the mitral valves to close completely during ventricular systole, causing mitral regurgitation [6]. There is lingering uncertainty about increased risk of ischaemic stroke in patients with mitral valve prolapse. Some observational studies have shown the risk for ischaemic stroke in these patients to be <1% annually [7]. It has been suggested that changes on the surface of the mitral valve leaflets might trigger platelet adherence and aggregation, leading to formation of a platelet-fibrin thrombus which could later embolize [8].

Most patients with mitral valve prolapse are asymptomatic. Symptoms such as fatigue, dyspnoea, palpitations, anxiety, and syncope often occur due to complications like mitral regurgitation, infective endocarditis, arrhythmias, cerebral ischemic events, autonomic dysfunction and sudden cardiac death. The classic auscultatory finding is a dynamic, midsystolic to late systolic click frequently associated with a high-pitched, late systolic murmur at the cardiac apex [9]. Two-dimensional echocardiography is the diagnostic gold standard [10,11]. It is confirmed by the presence of >2mm displacement of valvular leaflets above the mitral annulus into the left atrium during systole. It is classic if valvular leaflet thickness is > 5mm. It is non-classic if valvular leaflet thickness

is less than or equal to 5mm [1]. Other echocardiographic features are redundant valvular leaflets, annular dilatation and elongation of the chordae tendineae.

In this article, we report a case of a young man who was referred to our hospital on account of ischaemic stroke and was found to have myxomatous mitral valve prolapse.

Case Presentation

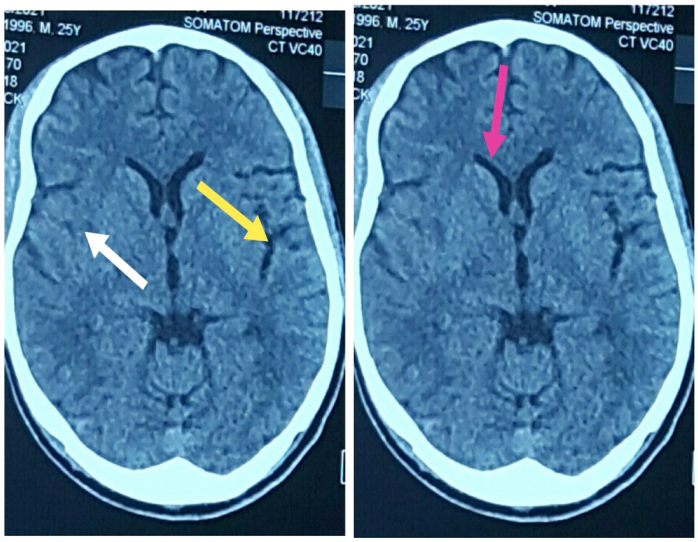
A 25 year old man with a one year history of recurrent palpitations was referred to our hospital with a complaint of sudden onset left-sided weakness which started three days prior to presentation. There was no associated loss of consciousness or seizures. There was also no antecedent history recurrent headaches. There was however a history of mild chest discomfort and easy fatigability. The recurrent palpitations were not associated with orthopnea, paroxysmal nocturnal dyspnea, syncope, panic attacks or fever. He had no family history of cardiac disease or sudden cardiac death. There was no history of valve replacement surgery.

Neurological assessment revealed dense weakness with power of 0/5 in the left upper and lower limbs. Initial Modified Rankin Scale score was 4. Pulse and blood pressure were 102bpm, regular and 110/62mmHg respectively. Jugular venous pressure was not raised. Other notable findings on cardiovascular examination were displaced, thrusting apex beat and a late systolic murmur preceded by a midsystolic click heard at the cardiac apex and accentuated by Valsalva manoeuvre. The chest was clear on auscultation. There were no peripheral signs of infective endocarditis. He had a slim body habitus with no Marfanoid features.

Non-enhanced computed tomography scan of the head showed hypodense lesions in the right frontal lobe measuring 1.0x1.8cm with mild compression of the frontal horn of the right lateral ventricle and in the right insular cortex measuring 1.3x1.2cm, consistent with acute right frontal and right insular cortex infarcts. Chest radiograph showed cardiomegaly, with cardiothoracic ratio of 0.56. A 12-lead ECG showed sinus rhythm with premature ventricular complexes and left ventricular hypertrophy according to Cornell voltage criteria. Transthoracic echocardiography showed a prolapsed, myxomatous and elongated anterior mitral valve leaflet with associated severe mitral regurgitation, annular dilatation and eccentric left ventricular hypertrophy. Ejection fraction was 52.48%. There were neither vegetations on the valves nor thrombi in the cardiac chambers. There were no features suggestive of cardiac tumour or rheumatic mitral valve disease. Complete blood count, erythrocyte sedimentation rate, sickling test, lipid profile, glycated hemoglobin as well as liver and renal biochemistries were normal. There was no bacterial growth on blood culture.

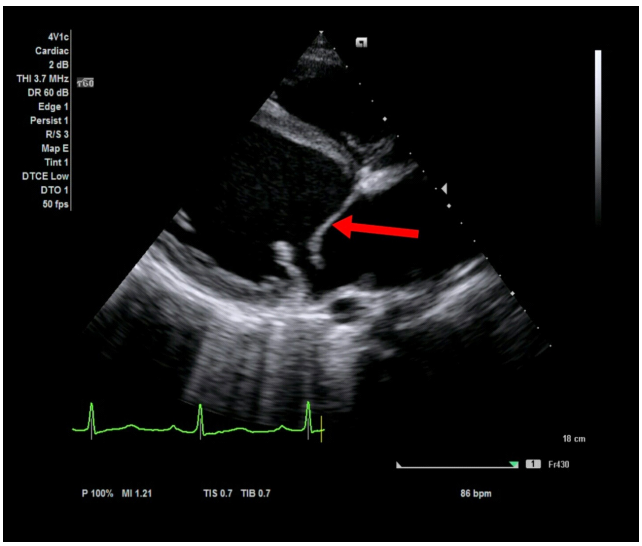
Based on the aforementioned findings, a diagnosis of myxomatous mitral valve prolapse complicated by ischaemic stroke was made. In consultation with the neurology team, the patient was put on warfarin. Physiotherapy was also started. At discharge after two weeks on admission, power in the left upper and lower limbs had improved to 2/5. The Modified Rankin Scale score, however, was still 4. He was scheduled to continue physiotherapy on OPD basis and to have regular follow-up reviews at the Cardiology and Neurology Clinics.

Figure 1: Plain head CT image: axial section at the level of the insular cortex.



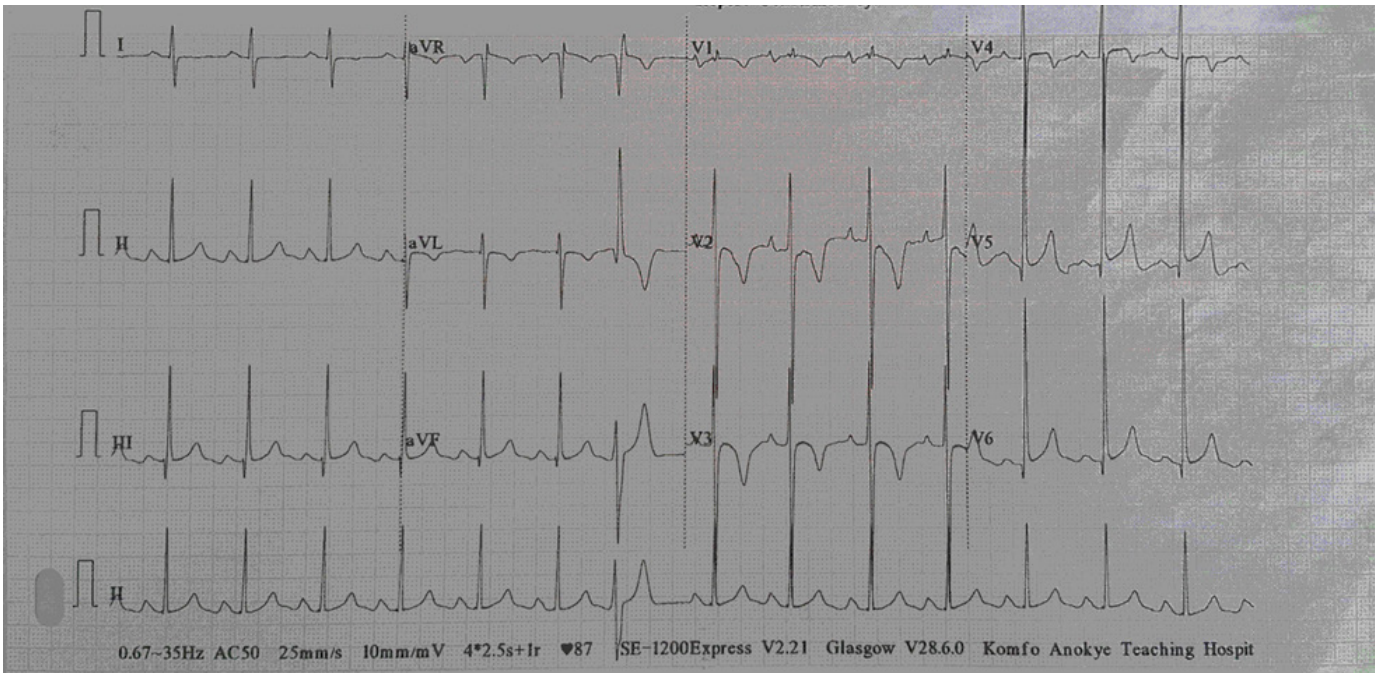
- Yellow arrow points to normal insular ribbon (bright white) in the lateral margin of the left insular cortex.
- White arrow points to loss of grey-white matter differentiation of the right insular cortex (insular ribbon sign).
- Red arrow shows mild compression of frontal horn of right lateral ventricle caused by hypodense lesion in the right frontal lobe.

Figure 2: Transthoracic echocardiogram: parasternal long axis view.



Red arrow pointing to elongated and myxomatous anterior mitral valve leaflet.

Figure 3: A 12-lead ECG showing sinus rhythm with premature ventricular complexes and left ventricular hypertrophy according to Cornell voltage criteria.



Discussion

The causal relationship between mitral valve prolapse and ischaemic stroke has been surrounded by controversies [1]. Barnett et al were the first to suggest a link between the two in 1976. They proposed that the ischaemic episodes were thromboembolic in origin [12].

Cardiovascular examination of our patient revealed a midsystolic click at the apex which was followed by a systolic murmur. This classic auscultatory finding is highly sensitive for making a diagnosis of mitral valve prolapse even though its specificity is limited [9]. The young man had an asthenic body habitus. Among the numerous reported correlates of this condition, only the association with leaner body mass has been reproducibly associated with mitral valve prolapse in the literature [13].

Pomerance in 1979 described pathological changes that occur at the margins of the prolapsed valves which have been found to result in the formation of platelet-fibrin thrombus over the redundant valvular leaflets. This phenomenon, in association with paroxysmal cardiac arrhythmias such as atrial fibrillation, may be responsible for embolization of the thrombus formed on the prolapsed valvular leaflets. Though there was no evidence of significant arrhythmia in our patient, it does not entirely exclude the possibility of a sudden arrhythmia which might have caused embolization to the cerebral arteries. It has been suggested that cerebral ischaemic events in these patients can arise from emboli emanating from the abnormal mitral valves with or without associated paroxysmal arrhythmia [13].

The non-contrast computed tomography scan of the brain showed right frontal lobe and right insular cortex infarcts. The arterial supply to the insular cortex is from branches of the middle cerebral artery. The results of a study conducted among 1,311 patients with middle cerebral artery territorial infarction suggested that cardioembolism was the primary stroke mechanism in patients with insular involvement (approximately 50% of cases) while large artery atherosclerosis constituted the most common cause of ischemic stroke in the middle cerebral artery [14]. A similar study carried out in the United States of America showed a greater association between insular involvement and cardioembolism (55.6% with insular lesions vs. 37.2% without insular lesions) [15]. These observations suggest cardioembolism as the likely mechanism underlying the ischaemic stroke suffered by our patient and mitral valve prolapse the most probable source of the thrombus.

With respect to secondary prevention, there have been no randomized trials to ascertain the efficacy of selected antithrombotic therapies in ischaemic stroke patients with mitral valve prolapse. In those with ischaemic stroke and mitral valve prolapse who have no evidence of atrial fibrillation, mitral regurgitation, left atrial thrombus or echocardiographic evidence of valvular thickening or redundancy, antiplatelet therapy is recommended. However, long-term anticoagulation therapy with warfarin is recommended for ischaemic stroke patients with mitral valve prolapse who have mitral regurgitation, atrial fibrillation, left atrial thrombus or echocardiographic evidence of valvular thickening or redundancy [16]. Our patient had mitral regurgitation and myxomatous, elongated valvular leaflet for which reason he was put on warfarin.

Conclusion

There is a tenuous relationship between mitral valve prolapse

and ischaemic stroke. This case report highlights the possibility, however remote, of myxomatous mitral valve prolapse as the source of thrombus formation in young patients presenting with cerebral ischaemic events, especially when they have clinical features consistent with this valvular abnormality and alternative causes of cerebral ischaemia cannot be identified.

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