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Case Report

A Rare Case of Spontaneous Calcific Cerebral Embolization Revealing a Calcified Rheumatic Mitral Stenosis: Case Report

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Abstract

Background: Cerebral cardiac embolism accounts for an increasing proportion of ischemic strokes and transient ischemic attacks. Calcific cerebral emboli are rare and mostly iatrogenic secondary to heart or aorta catheterization. However, spontaneous cerebral calcific embolism in the case of calcific aortic valve is very rare and there are less than 10 case reports in the literature. And more interesting fact, such an event in the context of calcific mitral valve disease has never been reported, at least to our knowledge. We are reporting a case of spontaneous calcific cerebral embolism revealing a calcified rheumatic mitral valve stenosis.

Case Presentation: We report a case of a 59 year-old-patient, with a history of rheumatic fever at the age of 14 and no history of recent cardiac intervention or aortic/carotid manipulation, admitted to the Emergency Department after a transient ischemic attack. Physical examination at admission found normal BP at 124/79 mmHg, HR at 90 bpm. 12-lead ECG showed an atrial fibrillation, no other anomalies. Unenhanced cerebral CT imaging was performed, revealing calcific material inside both middle cerebral arteries. Transthoracic echocardiography was performed, showing severe mitral leaflets calcification with a severe mitral stenosis, probably due to rheumatic heart disease. Cervical arteries Duplex was normal. A vitamin K antagonist (acenocoumarol) was prescribed, targeting an INR of 2 - 3 and mitral valve replacement surgery was performed using mechanical prosthesis. Short and long term with a 1-year follow-up were good and patient didn't experience any stroke.

Conclusion: Spontaneous calcified cerebral emboli secondary to mitral valve leaflets calcifications is a n extremely rare condition. Replacement of the valve is the only option to prevent recurrent emboli and outcomes are still to be determined.

Keywords: *Spontaneous Calcific Cerebral Emboli; Transient Ischemic Attack; Mitral Leaflet Calcification; Rheumatic Mitral Stenosis; Mitral Stenosis*

Abbreviations

AF: Atrial Fibrillation; LVEF: Left Ventricle Ejection Fraction; CT: Computed Tomography; TIA: Transient Ischemic Attack

Introduction

Cerebral cardiac embolism accounts for an increasing proportion of ischemic strokes and transient ischemic attacks [1]. Calcified cerebral emboli are a rarely reported but potentially cause of strokes and transient ischemic attacks and may be the first manifestation

of vascular or cardiac disease. Identification of the source of embolization is crucial to prevent future emboli, neurological damage and death. Non-contrast CT of the head is the most common imaging procedure used as the initial assessment of suspected stroke or transient ischemic attack [2].

Cerebral calcific embolus can occur after percutaneous and surgical intervention in the context of calcified aortic or mitral valve disease [3]. These emboli are presumed to occur because of valve trauma. However, spontaneous cerebral calcific embolism in the case of calcific aortic valve is very rare and there are less than 10 case reports in the literature. And more interesting fact, such an event in the context of calcific mitral valve disease has never been reported, at least to our knowledge.

We are reporting a case of spontaneous calcific cerebral embolism revealing a calcified rheumatic mitral valve stenosis.

Case Presentation

We are reporting a case of a 59-year-old man presenting to the emergency department after a transient ischemic attack (right hemiparesis and left central facial paralysis resolving briefly and spontaneously). There was a history of stage II NYHA dyspnea on moderate exertion for 2 years, no history of recent cardiac intervention or aortic/carotid manipulation, no other symptoms. Physical examination on admission found an irregular heart rhythm at 90 bpm, BP at 124/79 mmHg, mid diastolic rumble at apex, no signs of heart congestion and no signs of neurological impairment. 12-lead-electrocardiogram showed an atrial fibrillation without other anomalies. 2D transthoracic echocardiograms revealed important mitral valve leaflets calcifications, probably related to rheumatic heart disease, planimetry of the valve was not possible. Continuous wave Doppler interrogation of the mitral valve found a severe mitral stenosis with a mean gradient of 15 mmHg and a continuity equation surface of 1 cm^2. The aortic valve was thickened but not calcified, a mild aortic regurgitation was noticed. LV function was normal, LVEF at 55%. There was an RV longitudinal systolic dysfunction: TAPSE: 11 mm and S'VD: 6 cm/s. Tricuspid valve was thin with a mild tricuspid regurgitation. Continuous wave Doppler interrogation of the tricuspid valve allowed to estimate systolic pulmonary artery pressure at 69 mmHg. Mitral valve calcifications were best shown on a cardiac CT (Figure 1).

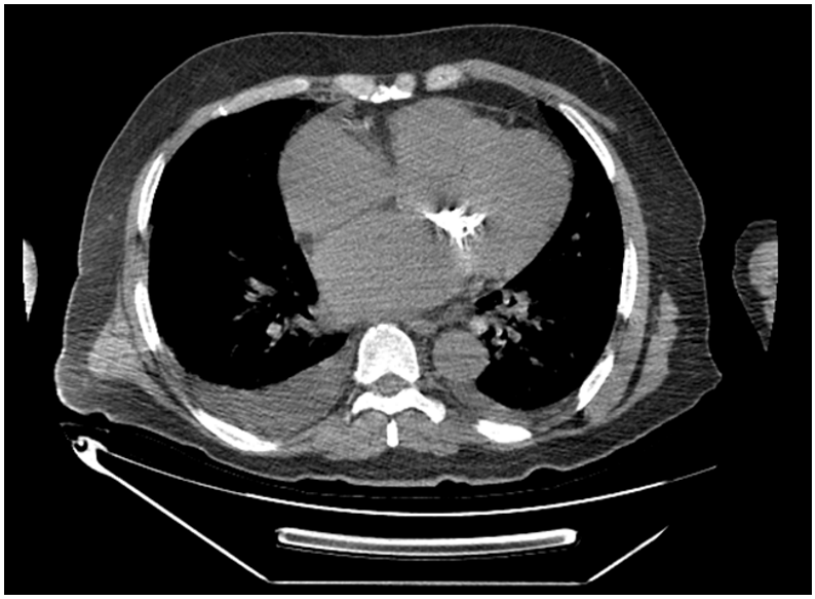

Figure 1: *Chest CT confirming mitral leaflets calcifications.*

Un-enhanced cerebral CT was performed revealing calcific emboli in both middle cerebral arteries (M3 and M4 segments) (Figure 2). Susceptibility weighted magnetic resonance cerebral sequences result is reported in figure 3.

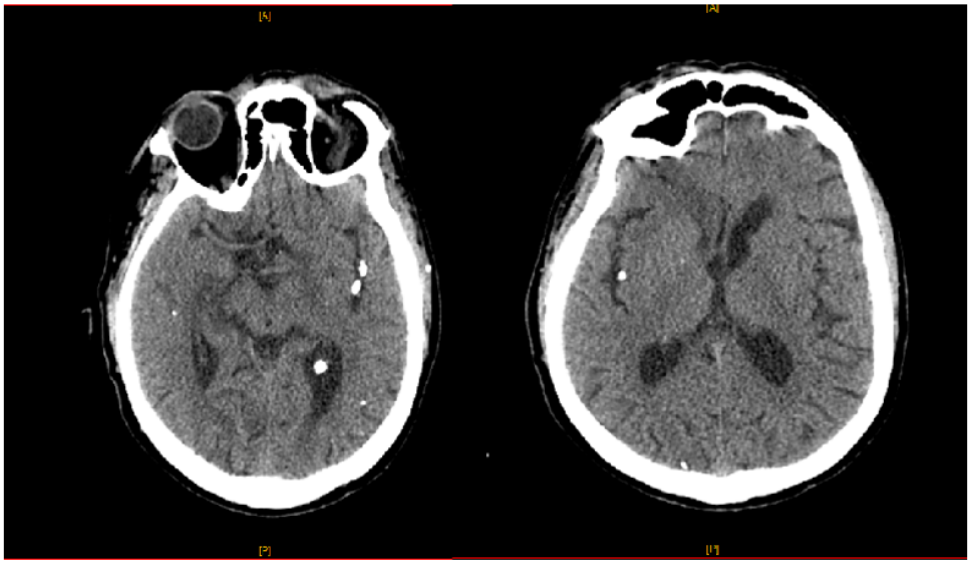

Figure 2: *Unenhanced cerebral CT with axial reconstructions showing calcific emboli in both middle cerebral arteries. These arteries were permeable.*

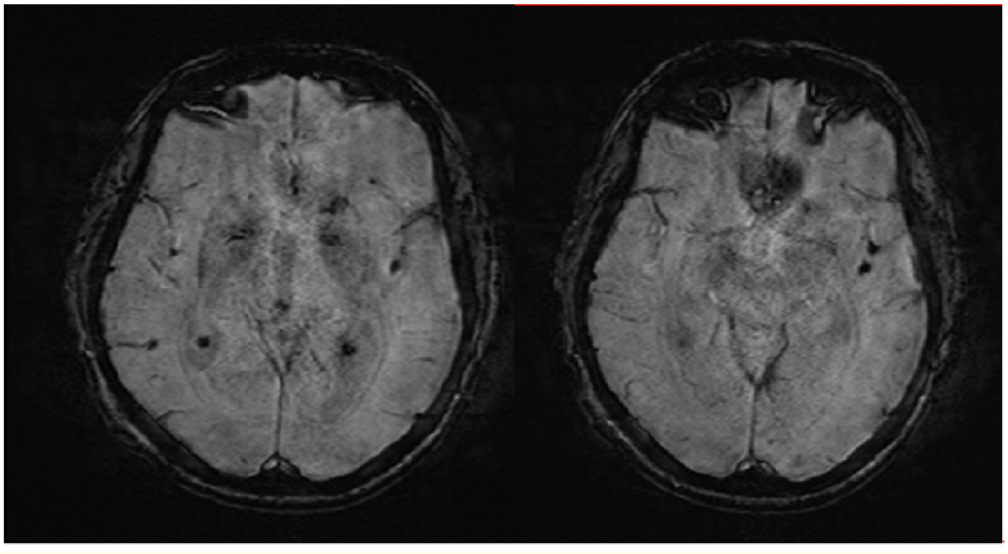

Figure 3: *Susceptibility weighted cerebral magnetic resonance sequences showing absence of signal inside both middle cerebral arteries.*

Cervical arteries Duplex showed normal carotid and vertebral arteries.

Coronary angiography was normal.

A vitamin K-antagonist (acenocoumarol) was prescribed, targeting an INR of 2.5 - 3.5 and replacement of mitral valve using a mechanical prosthesis was performed with a good short term and long term outcome. The patient did not experience ischemic stroke during a 1-year follow-up.

Discussion

In our patient, transient ischemic attack (TIA) was certainly due to atrial fibrillation (AF) and cerebral imaging leaded to the discovery of calcific emboli.

Approximately 6 - 31% of TIA are caused by a cardiogenic cerebral embolism (cardioembolic TIA) [4,5]. Determining TIA etiology is important before administering therapy. Permanent or paroxysmal, valvular and non-valvular, AF is associated with a 3-5-fold increased risk of TIA and stroke [6,7]. Cardiogenic cerebral embolization is common among patients with any cause of AF but particularly in AF resulting from rheumatic and arteriosclerotic heart disease [8]. It is recommended to prescribe these patients oral anticoagulant therapy in case of valvular AF, and according to CHA2DS2VASc score in case of non-valvular AF.

Calcified cerebral emboli are an infrequent but increasingly recognized cause of TIA and ischaemic stroke, although recognition amongst general radiologists and clinicians can be limited. Unenhanced CT and computed tomography angiography (CTA) are the imaging technique of choice for the diagnosis [9,10]. First described on CT by Yock in 1981, calcified cerebral emboli were previously thought to be unusual, and to most commonly arise following instrumentation of calcified cardiac valves or direct aortic/carotid artery manipulation [11,12]. However, there is growing evidence that spontaneous calcified cerebral embolism is more common, with a recent study and review of published cases reporting a 2.7% prevalence amongst a group of patients presenting with suspected stroke over a 1-year period. In this report, the middle cerebral artery was the site of 83% of calcified emboli. Cardiac valvular disease was more common than carotid atheromatous disease, with calcific aortic stenosis 3 times as common as mitral valve disease as the embolic source [2].

Iatrogenic calcific embolus following cardiac surgery or catheterization is common [3,13,14]. These emboli are presumed to occur because of valve trauma. According to a most recent post-mortem analysis of iatrogenic embolization cases, the source of calcific cerebral emboli was attributed to dislodgment and displacement of calcified material from calcified aortic valves and ulcerated aortic atherosclerotic plaques during therapeutic and investigative procedures [15].

There are some reported cases of cerebral calcific emboli following open heart mitral valvotomy and percutaneous mitral valvuloplasty. Mitral calcification accounts for fewer than 1% of cerebral cardiac embolism, and in all described cases, it was secondary to mitral valve intervention [15]. However, there isn't any case report published describing spontaneous calcific cerebral emboli in the context of calcific rheumatic mitral stenosis.

In case of stroke secondary to calcific emboli, the role of thrombolysis remains uncertain as there are conflicting reports regarding its efficacy in this setting [16-18]. There is debate and very limited experience regarding the place of mechanical thrombectomy [17,19]. Subsequent imaging evaluation of this subgroup of ischaemic stroke patients requires caution because the calcific nature of the embolus may be obscured on CT angiography or MRI. Clinical evaluation should include consideration of potential proximal source of calcific material and recent cardiac intervention or aortic/carotid manipulation. Although there is no data showing benefit of valve replacement, most authors advocate valve replacement to remove the source of emboli.

Regarding anticoagulation therapy, there is no evidence of its efficacy in the context of stroke secondary prevention in the absence of another indication. In this case, anticoagulation therapy using vitamin K antagonist was indicated because of valvular atrial fibrillation.

Conclusion

Spontaneous calcified cerebral emboli secondary to mitral valve leaflets calcifications is an extremely rare condition. Replacement of the valve is the only option to prevent recurrent emboli and outcomes are still to be determined.

Conflict of Interest

None.

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