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## Case Report

# Transient ischemic attack due to multiple spontaneous calcified embolus of the cerebral arteries on a calcified mitral and aortic stenosis <sup>☆</sup>

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## ABSTRACT

We report a case of calcified mitral and aortic stenosis revealed by a reversible ischemic stroke. A 59-year-old male patient, with background of hypertension, kidney failure, diabetes, and dyslipidemia, presented with neither acute onset of right-sided hemiparesis without aphasia nor any loss of consciousness. Head computed tomography (CT) revealed multiple rounded and amorph calcified high-density calcifications within the distal segments of both sylvian and posterior cerebral arteries. Angiographic CT of the carotids didn't reveal any stenosis or atherosclerotic plaques. Thoracic CT showed massive mitral and aortic valvular calcifications with a left ventricular hypertrophy.

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## Introduction

Embolic stroke is a major cause of mortality and morbidity in Morocco. To date, it represents the principal cause of admissions in the emergency room of the Ibn Rochd University Hospital in Casablanca. Cerebral emboli can originate from many sites and vary in their histopathologic composition [1]. Calcified embolus of the cerebral arteries is a rare and controverted cause of ischemic stroke. Mitral or Aortic annular fibrocalcification is a chronic process involving lipid deposition,

fibrosis and calcifications of the rim [2]. Frequently found in patients with cardiovascular risk factors background, this condition has never been specifically examined whether it predicts the incident of calcified embolus in the cerebral arteries.

## Case presentation

A 59-year-old, with a background of hypertension, episodes of transient ischemic attacks, kidney failure, diabetes and dys-

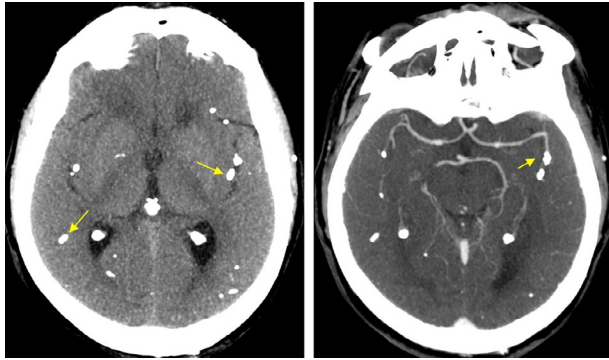
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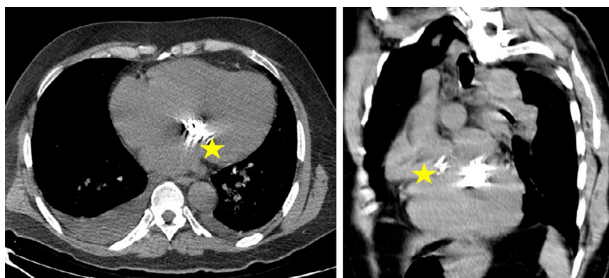
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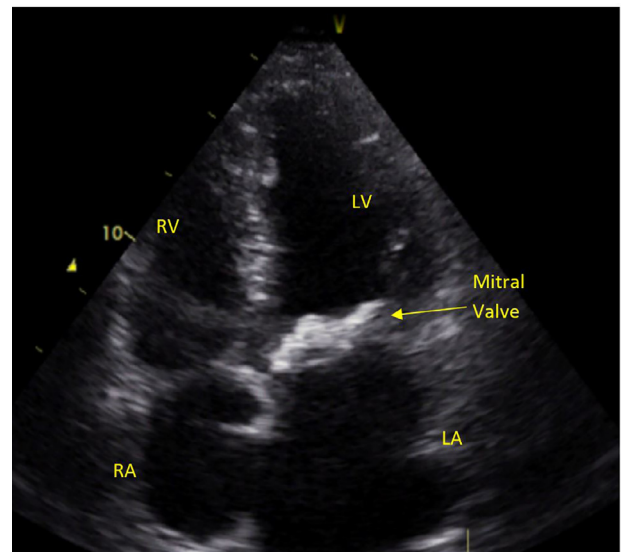
**Fig. 1 – Axial brain CT before (a) and after injection of iodine contrast media in maximum intensity projection (b), showing multiples calcifications (arrows) on the path of cerebral arteries' branches. However, we still notice a satisfying perfusion after the angiographic phase.**



**Fig. 2 – Axial (a) and coronal (b) chest CT acquisition showing multiples high density calcifications of the mitral and aortic valves (stars). Notice the bilateral pleural effusion secondary to his cardiac condition.**

lipidemia, presented with acute onset of right-sided hemiparesis graded at 3 of 5, without facial drop or dysphasia. A head CT with angiogram injection of iodine contrast media revealed multiple calcific attenuations in the territories of the distal M3 and M4 segments of both middle cerebral arteries and distal branches of the posterior arteries (Fig. 1). The anterior cerebral arteries were normal. There were no signs of cerebral infraction, with normal gray-white matter differentiation in the sylvian and posterior cerebral arteries. Angiogram acquisition from the left ventricle to the circle of Willis showed calcific attenuation following the branches of the middle and posterior cerebral arteries which were poorly opacified distally. No stenosis or atherosclerotic surcharge was found on both internal carotid arteries. The patient regained his motrice capabilities less than 24 hours after the stroke, keeping a slight confusion.

A chest CT was performed and revealed multiple calcifications of the mitral and aortic valves with a left ventricular hypertrophy without any thrombus. It has also showed a dilatation of the left atrium and the pulmonary veins (Fig. 2).



**Fig. 3 – Four cavities ultrasound slice of the heart showing a calcified mitral stenosis.**

The fact that the previous scans showed topography and different number of its calcifications, made us rule out the diagnosis of primary calcifications of the cerebral arteries.

Echocardiography revealed a thickened aortic and mitral valve with multiple amorph calcifications with high risk of embolism and stenosis with an area of 1 cm<sup>2</sup> on the mitral valve (Fig. 3). It has also showed a right ventricular dysfunction with a left ventricular concentric hypertrophy, without any patente foramen ovale or right-left shunt.

The supra-aortic trunks and transcranial sonography were normal with a discreet atherosclerosis.

A follow-up noncontrast brain CT didn't show any signs of hemorrhage infarction or aggravation. The patient was placed under antiplatelet therapy and made an excellent recovery regaining a grade 4 of 5 power in both right upper and lower limbs after few days of surveillance.

## Discussion

Embolic stroke is a major cause of mortality and morbidity. Cerebral emboli can originate from many sites and vary in their histopathologic composition [1]. Calcified emboli of the cerebral arteries are a rare and controverted cause of ischemic stroke. Mitral or aortic annular fibro-calcification is a chronic process involving lipid deposition, fibrosis, and calcifications of the rim [3].

There are multiple explanations to the finding of calcifications in the brain parenchyma. One should differentiate intraparenchymal lesions and vascular calcifications. Also, calcified arteries may be seen in acute strokes with the hyperdense artery sign [4], that translate into an early thrombosis, and intracranial calcification in the internal carotids [5].

Intraparenchymal lesions often represent calcified granulomas, of cavernomas or neurocysticercosis [6]. In opposite,

calcified cerebral emboli can be seen in the paths of major vessels as it is the case here, or sitting on the brain surface. They are more frequently mural and eccentric intravascular calcification, most commonly seen in atherosclerotic disease.

It's possible that the kidney failure of the patient was a risk factor for developing such valvular and vascular calcifications like it was described in Braun et al's study [7].

Calcified embolus is round shaped, localized in the path of a cerebral artery with a calcic attenuation [6]. Calcifications of the cerebral arteries do not usually cause strokes, unless there is significant arterial stenosis or occlusion. The angiographic images didn't find any significant stenosis. We can impute de multiple episodes of transient ischemic attacks to those calcifications seeing their topography.

Brain MRI shows multiple blooming lesions on the T2\* and SWI-weighted images. It can also reveal ischemic territories in T2, FLAIR, and DWI images.

With time, those calcifications may change in size and in density. The evolution conduct in a spreading through the distal and cortical segments of the cerebral arteries branches [6].

The migration of valvular calcification is rare and can be spontaneous or secondary to chest traumatism [8].

Previously, calcified cerebral emboli have been described secondary to a valve disease. However, Oliveria-Filho et al's prospective study demonstrated that the risk of embolus was not increased [2]. Thus, calcified emboli was more frequently due to carotid manipulations rather than aortic or valve disease [9,10]. In our case, the likely source of the embolus occurred from the migration of a mitral or aortic calcification.

## Conclusion

The diagnosis of calcified cerebral embolus should be considered after an onset stroke deficit, a calcified attenuated structure in a vessel of the brain and many asymptomatic calcified embolus in other territories. Such abnormalities should conduct to furthermore investigations in search of a atherosclerotic disease.

## Patient consent statement

Written and informed consent for publication of the case was obtained from the patient.

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