

CASE REPORT

Calcified embolism: a rare cause of cerebral infarction

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SUMMARY

Calcified cerebral emboli (CCE) are a rare cause of stroke and these emboli can be identified on a CT scan of the brain performed for the initial evaluation of stroke. In this report we present a patient who developed a CCE following cardiac catheterisation that lodged in the left middle cerebral artery with resultant right hemiparesis and aphasia. The calcified embolus was seen on CT but could not be identified on MRI. Predisposing factors for CCE include angiography and valve or vessel wall calcification. The natural history and response to standard therapy in patients with CCE as compared with stroke of other aetiologies have not been studied until now. Increased awareness and ability to identify calcified emboli will help us to have an improved understanding of strokes resulting from CCE.

BACKGROUND

Emboli in the cerebral circulation are a common cause of stroke. Cerebral emboli are most often dislodged material from a thrombus which has formed either in the heart or in a diseased artery. Rarely, emboli may consist of materials other than thrombus; such as bits of plaque containing cholesterol and/or calcium, air, fat or tumour fragments. Calcified cerebral emboli (CCE) are a rare cause of cerebral embolism and moreover are unique in that they can be diagnosed with fair certainty from the initial CT of the brain, which is usually performed as a first step in the evaluation of a patient with stroke. Through this case report we would like to demonstrate how to identify CCE on CT and discuss the predisposing factors and the possible implications on therapy that identification of this condition may have.

CASE PRESENTATION

An elderly gentleman with no history of hypertension or diabetes mellitus presented with a 2-month history of angina on exertion. Examination was normal except for blood pressure of 190/100 mm Hg. His ECG was normal and transthoracic echocardiography (TTE) showed normal left ventricular function, trivial mitral regurgitation and mild left ventricular hypertrophy. He underwent coronary angiography for his symptoms; the procedure was uneventful and showed only mild coronary artery disease and he was planned for medical management. Around 6 h after angiography while in the coronary care unit he became restless, agitated and his speech was found to be slurred; blood pressure recorded at this time was 200/100 mm Hg, for which he was started on

intravenous antihypertensives. The next day morning on awakening from sleep he had developed dense right hemiplegia, global aphasia with gaze preference to the left.

INVESTIGATIONS

A plain CT scan of the brain was performed which showed an evolving hypodensity involving the left temporo-parietal region, a circular hyperdense lesion in the left sylvian fissure suggestive of a calcified embolism involving the M2 segment of the left middle cerebral artery (MCA) and calcification of the wall of the intracranial right internal carotid artery (ICA; figure 1). MRI with diffusion-weighted images of the brain performed 1 day after CT confirmed the left MCA territory infarction with sparing of the basal ganglia consistent with occlusion of the M2 segment (figure 2), however the MRI itself did not show any signal changes to suggest a calcified embolism.

TREATMENT

The patient was started on antiplatelets and was not thrombolysed as he was beyond the window period.

OUTCOME AND FOLLOW-UP

The patient's neurological condition remained status quo, however he developed urinary tract infection which rapidly progressed to septicaemia. Despite antibiotic therapy he passed away shortly thereafter.

DISCUSSION

Intracranial calcifications may be physiological which include calcifications of the pineal gland, choroid plexus, basal ganglia and dura mater; or pathological which include infections either acquired/congenital, inflammatory lesions, tumours, phakomatoses, metabolic disorders, dystrophic and vascular calcification.¹ The presence of calcification in the path of major vessels is suggestive of vascular calcification²; in the intracranial compartment, these are most commonly seen involving the carotid siphon secondary to atherosclerosis.³ Calcification is rarely noted in the MCA or anterior cerebral artery.³ Calcified emboli may rarely be seen over the surface of the brain secondary to being lodged in the superficial smaller arteries.² The presence of a calcified lesion in the MCA and resultant ischaemia of the vascular territory distal to the lesion would suggest that the calcified lesion was responsible for the neurological deficit in this patient. Previous reports of calcified cerebral

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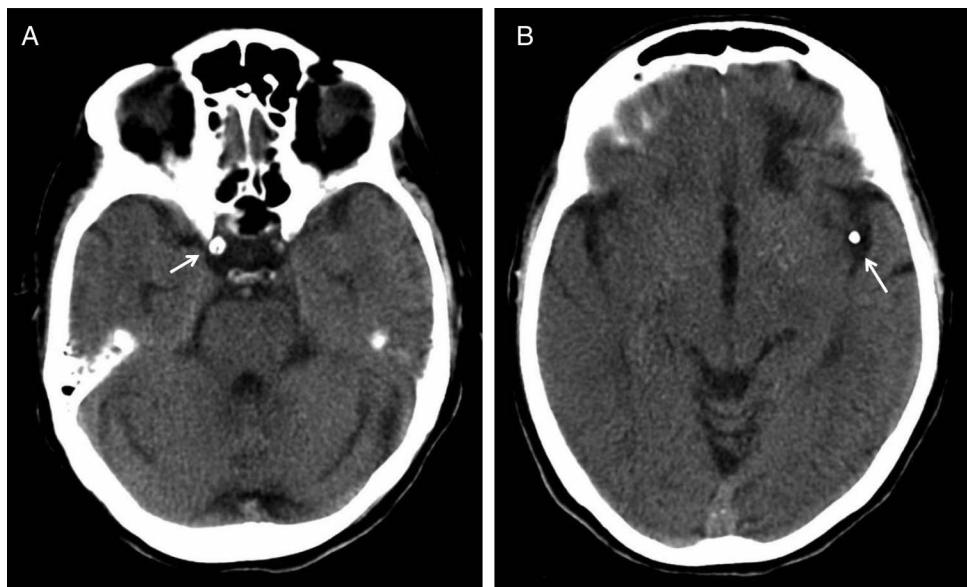


Figure 1 CT of the brain showing calcification of the intracranial right internal carotid artery (A, arrow) and a calcific speck in the left sylvian fissure corresponding to the M2 segment of the left middle cerebral artery (B, arrow)

embolism have been attributed to angiography,^{2 4 5} mitral annular calcification,⁶ calcified aortic stenosis,^{7 8} aortic and carotid vessel wall calcification.² Our patient developed a calcified cerebral embolism following coronary angiography. His TTE prior to and after the procedure did not reveal calcification of the mitral or aortic valve and therefore we believe that the calcified embolism originated from a vessel wall. No imaging of the neck vessels was performed as the patient developed other complications, however brain imaging did show calcification of the intracranial right ICA suggesting the possibility that he may have had calcification of other major vessels. The site of origin of embolism in this case is not evident. One of the possibilities is embolism originating from the arch of aorta secondary to coronary angiography, the odd feature is the onset of neurological symptoms several hours after angiography however this has to

be considered in view of the temporal association between the procedure and the neurological event; another possibility is spontaneous embolism from the carotid artery in the neck.

Our patient was not thrombolysed as he was outside the therapeutic window. Current guidelines do not make a distinction between calcified and non-calcified emboli with regard to thrombolysis. The presence of calcification in an embolus may play a role in determining the effect of thrombolytic therapy with some isolated case reports showing a poor response^{9 10} and others demonstrating a good response.²

These images highlight a rare variety of cerebral embolism and demonstrate the ability of CT to detect calcified emboli which would not have been detected by an MRI and thereby re-emphasises the advantages of CT in the setting of acute stroke.

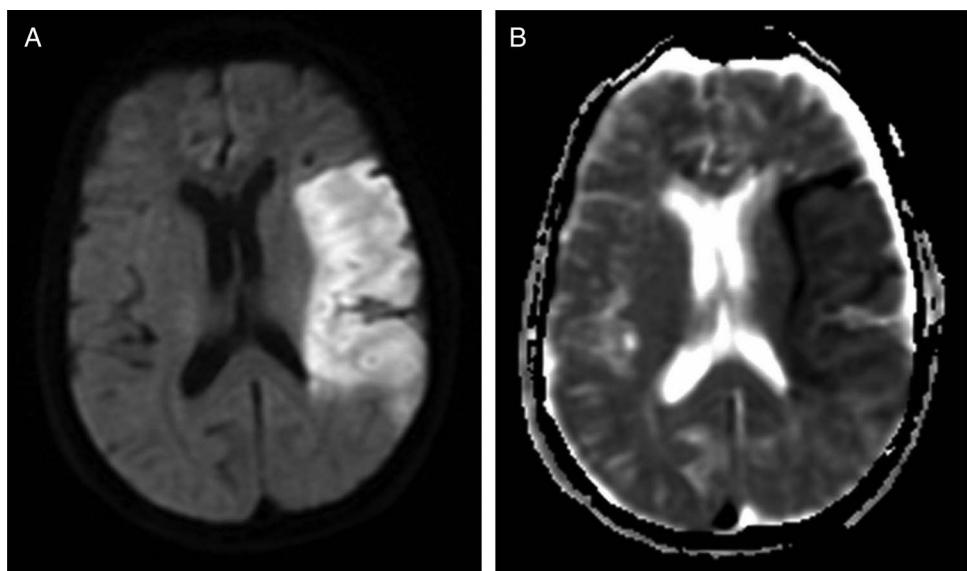


Figure 2 Diffusion-weighted images of the brain with left temporoparietal hyperintensity (A) which is restricted on apparent diffusion coefficient maps (B) suggesting an acute infarct.

Learning points

- ▶ Calcified cerebral emboli are a rare cause for cerebral infarction.
- ▶ Identified by the presence of calcification along the path of major vessels and rarely over the surface of the brain.
- ▶ Predisposing factors include cardiac catheterisation, mitral annular calcification, calcified aortic stenosis and aortic or carotid vessel wall calcification.
- ▶ CT is superior to MRI in identifying calcified cerebral emboli.

Contributors All the authors were involved in the conception, design, drafting, revision and the final approval of the case report.

Competing interests None.

Patient consent Obtained.

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