Calcified Cerebral Emboli

SUMMARY: Intracranial calcifications may represent calcified cerebral emboli. Calcified emboli may be overlooked even though cerebral CT is widely used as a stroke assessment. We report 4 cases of calcified cerebral emboli and demonstrate the value of CT in the diagnosis and temporal evaluation of such emboli.

Case Reports

Case 1
A 66-year-old man presented with acute onset of right-sided hemiparesis, right facial droop, and dysphasia. A noncontrast CT of the brain revealed a calcific attenuation in the region of the distal M1 segment of the left middle cerebral artery (Fig 1A). There was no evidence of cerebral infarction, with normal gray-white matter differentiation identified in the left cerebral hemisphere (Fig 1B). A CT angiogram (Fig 1C) was obtained from the level of the aortic arch to the circle of Willis, which showed a calcific attenuation in the distal left M1 segment, consistent with a calcified cerebral embolus. High-grade stenosis of the left internal carotid artery with eccentric calcified plaque was also identified (Fig 1D). Because the patient presented more than 3 hours after the onset of symptoms, thrombolysis was not administered. A follow-up noncontrast CT showed a large area of ischemic infarction in the distribution of the left middle cerebral artery with loss of gray-white differentiation and local mass effect (Fig 1E). The patient was then placed on antiplatelet therapy and made an excellent recovery, regaining grade 4 of 5 power in both right upper and lower limbs after 3 months. The patient’s dysphasia also dramatically improved with intensive speech therapy. Some residual weakness persisted in both right upper and lower limbs, and the patient also had residual word-finding difficulties. Left carotid endarterectomy was successfully performed 6 months after the initial presentation.

Case 2
A 70-year-old woman was admitted to our institution with syncope and bradycardia. Investigations included a cardiac catheterization study, which was complicated by sudden onset of right-sided weakness and dysphasia after the procedure. A left middle cerebral territory infarct was suspected, and a noncontrast CT of the brain was performed (Fig 2A, -B). A calcific attenuation was identified in a Sylvian branch of the left middle cerebral artery (Fig 2A). An image from the same CT examination at a higher level showed a calcified gyral attenuation in the cortex of the right frontal lobe (Fig 2B). There was no evidence of cerebral hemorrhage. The patient was treated with intravenous thrombolysis and made an excellent clinical recovery, with complete resolution of the right-sided weakness and dysphasia. Normal power was noted in both upper and lower limbs. A repeat noncontrast brain CT was performed 1 day after thrombolysis. The previously noted calcific attenuation in the region of the left Sylvian fissure was no longer seen within the proximal left middle cerebral artery branch (Fig 2C). At a higher level, the previously noted right-sided cortical calcified attenuation was unchanged (Fig 2D), but 2 new calcified densities were seen in the region of the left precentral gyrus, consistent with downstream migration and fragmentation of the previously identified calcified cerebral embolus.

Case 3
A 61-year-old woman with a history of insulin-dependant diabetes mellitus and multiple medical problems presented with renal failure. The patient also had a history of multiple left hemispheric transient ischemic attacks and a left frontal lobe cerebral infarct. Six months before this admission, the patient had undergone successful angioplasty of the supraclinoid portion of the left internal carotid artery. During the current admission, the patient suddenly developed a new right hemiparesis and slurred speech. A noncontrast CT of the brain was performed and revealed a calcified attenuation in the region of the left posterior cerebral artery (Fig 3A). An axial image obtained at a higher level showed a long-standing infarct in the deep white matter of the left frontal lobe (Fig 3B). Thrombolysis was not administered because a renal biopsy had been performed the day previously. The patient’s neurologic status improved a few hours after the CT examination, with residual right upper and lower limb weakness (grade 3 of 5), and rehabilitation was started. The previously noted slurring of speech resolved with intensive speech therapy. Echocardiography showed a normal aortic valve. A CT angiogram showed calcification at the origin of the left vertebral artery and calcified plaques in both carotid bulbs.

A repeat noncontrast CT, performed on a subsequent admission (5 months later), showed an infarct in the left temporal lobe (Fig 3C). The previously identified calcified attenuation was noted to have migrated into the left occipital lobe (Fig 3D). The findings were consistent with migration of a calcified cerebral embolus within the left posterior cerebral artery circulation.
Case 4
An 86-year-old woman presented with acute onset of central chest pain and breathlessness. Electrocardiography and laboratory investigations revealed an acute inferior wall myocardial infarction. The patient received intravenous thrombolysis and was transferred to the coronary care unit. Transesophageal echocardiography demonstrated a sclerotic aortic valve and extensive atheroma in the aortic arch. A chest x-ray film showed extensive calcification in the aortic arch.

The patient became confused on day 3 after admission, and a CT brain scan was obtained (Fig 4A). Note was made of a tiny gyral calcification in the posterior right frontal lobe, interpreted as a tiny granuloma or a calcified cavernoma. Five days later, the patient had a ventricular fibrillation cardiac arrest and was resuscitated successfully with DC cardioversion. The patient received several rounds of manual chest compressions before successful cardioversion. Following the cardiac arrest, the patient was noted to be confused, with a decreased level of consciousness. No focal neurologic signs were elicited on clinical examination. A repeat CT of the brain was performed, and no evidence of hemorrhage or infarction was identified. A new calcific gyral attenuation was noted in the anterior right frontal lobe (Fig 4B), likely representing a second calcified embolus to the right cerebral hemisphere.

Discussion
Many causes of physiologic intracranial calcification exist. Intraparenchymal cerebral calcifications often represent lesions such as calcified granulomas or calcified cavernomas. Although calcified cavernomas can be seen anywhere in the brain parenchyma, calcified cerebral emboli can be seen in the paths of major vessels (cases 1 and 3) or sitting on the brain surface (cases 2 and 4). Mural and eccentric intravascular calcification is commonly seen secondary to atherosclerotic disease. However, as we have demonstrated, intracranial calcifications may be secondary to calcified cerebral emboli that can change in site, size, and attenuation with time.

Calcified cerebral emboli have been previously described secondary to aortic valve disease. However, a prospective study in a large cohort of patients with aortic valve calcification demonstrated that the risk of embolic stroke was not increased. Calcified cerebral embolism has been reported secondary to direct carotid manipulation. CT findings in spontaneous calcified cerebral emboli secondary to a carotid source have been reported twice before. In our case 1, the presumed source of the calcified embolus was from calcified plaque in the ipsilateral stenotic internal carotid artery. Calcified cerebral emboli originating from the aortic arch have also been previously reported. In our case 2, the calcified cerebral embolus occurred after coronary artery catheterization, a scenario that has been previously described. The source of the...
calcified embolus in our case 3 may have been from the calcified plaque seen at the origin of the left vertebral artery. This patient had a history of widespread atheromatous disease and bilateral carotid stenosis. In our case 4, the likely source of the calcified emboli was from the calcified aortic arch, dislodged by vigorous manual chest compressions. To our knowledge, calcified cerebral emboli in the posterior cerebral circulation have not been reported previously.

All noncontrast images shown in this Case Report are axial 2.5-mm cuts from routine CT head scans. Intracranial calcifications seen on routine noncontrast CT of the head may represent calcified cerebral emboli. In contrast to a previous case report, this study documents that intravenous thrombolysis can be effective in the treatment of acute stroke secondary to calcified cerebral embolus. Therefore, an acute calcified cerebral embolus seen on a brain CT in the setting of a patient with acute stroke is not a contraindication for administering intravenous thrombolysis. This information is important when assessing a patient’s likely outcome when commencing thrombolytic therapy.
Conclusions

The presence of calcified cerebral emboli should prompt evaluation of the carotid arteries, the aortic arch, and the heart. The presence of calcified cerebral emboli does not indicate a contraindication to intravenous thrombolysis. Calcified cerebral emboli should not be overlooked when using cerebral CT for stroke assessment.

References