Showered Calcific Emboli to the Brain, the ‘Salted Pretzel’ Sign, Originating From the Ipsilateral Internal Carotid Artery Causing Acute Cerebral Infarction

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Background and Purpose—Unenhanced head CT has become the first line imaging study in the evaluation of suspected acute cerebral ischemia. It is important to identify subtle findings of acute ischemia on this exam to direct appropriate patient management.

Summary of Case—We report the first case of multiple pial surface distal internal carotid artery territory calcified emboli causing multifocal cerebral infarctions, likely from a carotid bifurcation source.

Conclusions—Visualization of multiple pial surface calcifications on unenhanced head CT, the ‘salted pretzel sign’, should raise suspicion for acute infarction from showered calcific emboli. (Stroke. 2009;40:e319-e321.)

Key Words: acute care ■ brain infarction ■ cerebrovascular accident ■ CT ■ MRI ■ radiology ■ stroke in evolution

Emboli are a well-known cause of cerebral ischemia being attributed to a significant number of infarctions. However, calcified emboli as a cause of cerebral infarction are rare with only a few reported cases in the literature, all of which document only central large arterial vascular emboli. We present the first case of multiple small pial surface-calcified emboli showered to the distal anterior cerebral artery and middle cerebral artery (MCA) territory, likely from the carotid bifurcation, as the cause of multifocal internal carotid artery territory infarction. This is the most thoroughly imaged case of calcified cerebral embolus to date, including complete CT, MRI, and CT angiographic evaluation.

Case Presentation

A 63-year-old man with a history of hypertension, cardiac disease, and chronic obstructive pulmonary disease presented to the emergency department with right hemiparesis and aphasia. He was outside the window for thrombolytic therapy. Initial unenhanced CT of the brain showed multiple punctate calcific densities along the pial surface of the left anterior cerebral artery and MCA distribution (Figure 1) without other evidence of infarction. MRI performed on hospital Day 3 showed multifocal areas of diffusion restriction within the left cerebral hemisphere (Figure 2). The apparent diffusion coefficient map (not shown) revealed low signal intensity corresponding to the regions of diffusion restriction consistent with acute cerebral infarction. On hospital Day 6, a CT angiogram obtained from the level of the aortic valve to the circle of Willis revealed multifocal left cerebral hypodense regions (not shown) corresponding to the prior regions of diffusion restriction and a large densely calcified plaque producing high-grade stenosis at the origin of the left internal carotid artery (Figure 3). The CT angiogram also demonstrated the punctate calcific densities on the pial surface of the left cerebral hemisphere were continuous with cerebral arteries (Figure 4) with several arteries being truncated at the level of the calcification. No cardiac valve calcifications were present on the CT angiogram, and a cardiac echocardiogram performed on hospital Day 2 showed no cardiac embolic source.

Discussion

Attributed sources for calcific emboli causing cerebral infarction span from the heart to the neck/cerebral vasculature. Previous cases in the English literature attribute sources for calcified cerebral emboli to the aortic valve, origin of the left vertebral artery, brachiocephalic trunk, atherosclerotic aortic, cardiac thrombus, calcified mitral valve, as a complication of coronary angiography and as a complication of carotid intervention.

To our knowledge, this is only the fourth reported case, since 1981, of cerebral infarction with the source attributed to spontaneous calcific emboli from the ipsilateral carotid bifurcation and the only example of calcific emboli showered throughout the distal anterior cerebral artery and MCA...
distribution. All previous cases were characterized by large calcific emboli within the proximal intracranial circulation. Different from noncalcified more proximal MCA thromboemboli previously described as the hyperdense MCA and MCA dot signs, these calcified peripheral emboli in pial surface arteries give the appearance of salt on a pretzel. This is the only case confirmed by diffusion restriction MRI and CT angiography.

This patient presented with pial surface calcifications throughout the left internal carotid artery circulation without

Figure 1. Axial 5-mm image of the brain from the initial unenhanced CT scan shows multiple punctate calcific densities along the pial surface of the left cerebral hemisphere, the “salted pretzel” sign. No brain parenchymal abnormality is appreciated.

Figure 2. Diffusion-weighted image of the brain shows scattered areas of diffusion restriction within the left cerebral hemisphere. Note the patchy distribution of infarcted tissue within the left MCA territory in the region of the calcifications seen on the CT. Diffusion restriction was also documented in the anterior cerebral artery territory (not shown).

Figure 3. Sagittal oblique maximum intensity projection image of ipsilateral internal carotid artery origin from a CT angiogram from the level of the aortic valve to the circle of Willis shows densely calcified plaque at the origin of the left internal carotid artery.

Figure 4. Sagittal maximum intensity projection image of the brain shows multiple punctate calcific densities (arrowheads) within the arterial vasculature of the left cerebral hemisphere, several of which show truncation at the calcification.
brain parenchymal abnormality on initial unenhanced CT imaging. Because most causes of intracranial calcification are attributed to nonemergent, often chronic, phenomena, it is important to recognize the near pathognomonic unenhanced CT appearance of this entity. Although much less likely in the setting of suspected stroke, other etiologies for multiple peripheral pial surface or superficial gray matter calcifications include congenital infections, tuberous sclerosis, distal intracranial atherosclerotic disease, remote trauma, and prior intrathecal administration of Pantopaque contrast media.

A significant treatment consideration is the potential decrease in efficacy of thrombolytic therapy in patients presenting within the therapeutic window. The 2 documented experiences with thrombolytic treatment of calcified embolic cerebral infarctions have shown mixed results. The patient in this case presented outside the therapeutic window.

In the setting of potential stroke, acute calcific embolus should be considered in patients presenting with intracranial calcifications in the expected location of intracranial arteries on CT. This case suggests the presenting CT imaging findings of stroke along with the hyperdense MCA, MCA dot, and insular ribbon signs should include calcifications within a distal intracranial arterial vascular territory, the “salted pretzel” sign.

Disclosures
None.

References
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