Calcific Cerebral Emboli and Aortic Stenosis: Detection of Computed Tomography

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SUMMARY Embolism with brain infarction rarely complicates calcific aortic stenosis (CAS). We report a case with severe CAS where the patient experienced multiple embolic strokes immediately following retrograde heart catheterization. Calcific emboli in the cerebral arteries were demonstrated by computed tomography (CT).

IN THE ABSENCE OF coexistent mitral stenosis or infective endocarditis, clinical brain embolism is uncommonly associated with CAS.1-3 In a single previous report, occlusion of a femoral artery by a large calcium embolus followed left heart catheterization in a patient with severe CAS.4 We report a patient who suffered several embolic strokes following retrograde heart catheterization for CAS, with intraarterial calcific emboli and cerebral infarction demonstrated by CT.

Case Report
A 66-year-old black male underwent aortography and left heart catheterization for evaluation of aortic valvular stenosis and suspected coronary artery disease. Prior to angiography, M-mode echocardiography had revealed a heavily calcified, stenotic aortic valve (fig. 1). The mitral valve annulus was minimally calcified; there was no mitral stenosis. Retrograde heart catheterization was done via a transfemoral approach with some difficulty encountered in the retrograde passage of the catheter through the aortic valve which was severely stenotic with a 0.6 cm² valve orifice.

Two days following cardiac catheterization, the patient developed a left hemiparesis, ataxia and confusion. The following day, the weakness progressed to a left hemiplegia, and a left hemianopsia was detected. A CT scan of the head done without contrast enhancement showed a focal calcific density along the course of the right middle cerebral artery (MCA) just proximal to the usual location of the MCA trifurcation and another in the proximal perimesencephalic segment of the right posterior cerebral artery (fig. 2). A low attenuation area was seen in the right basal ganglionic area and the adjacent corona radiata, that evolved in density on subsequent scans and was consistent with an acute cerebral infarction (fig. 3). Careful fundoscopic examination failed to reveal calcific retinal emboli.

Heparin therapy was briefly instituted, but was discontinued when hemiplegia persisted for 48 hours. No neurologic worsening occurred after the fifth day postcardiac catheterization. A follow up CT scan eight months later showed no change in the position of the calcific emboli and no extension of the area of infarction.

Discussion
While the underlying pathology of CAS is usually a congenitally bicuspid aortic valve or an inflammatory fibrocalcific process (including rheumatic valvular disease), the continuing disease process is one of organizing microthrombi on disrupted valvular endothelium.

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Circumflex branch of the left coronary artery by a presumably calcific embolus has also been described during thoracic aortography by Arvidson. Considering the difficulty that is encountered in traversing the aortic valve in tight AS during retrograde catheterization, a higher incidence of calcific embolization would be expected than suggested by the above reports.

CT has been instrumental in the diagnosis of cerebral infarction, based on the density changes in the infarcted parenchyma, which first appear several hours to days post-ictus. Earlier diagnosis can sometimes be facilitated by direct visualization of the embolus in a major cerebral artery, usually the middle cerebral. This high density secondary to clotted blood is probably not useful in separating embolism from thrombosis. Calcified emboli have been demonstrated by CT in the proximal middle and anterior cerebral arteries, with good evidence to support the origin of these emboli in calcified carotid plaques (2 cases) and from mural thrombi in the atrium (1 case) and left ventricle (1 case).

Embolism in our patient was presumably precipitated by cardiac catheterization with dislodgement of calcific deposits. It is therefore not surprising that there is a substantial incidence of spontaneous embolization from CAS. In Holley’s autopsy series of 156 patients with CAS, there were 45 instances of calcific coronary and systemic emboli in 31 patients. However, spontaneous clinically significant calcific embolization is rare. This disparity between the rare recognition of clinical embolism in CAS and the more frequent detection of calcific emboli at autopsy and also on fundoscopic examination of the retina suggests that most of these calcific emboli are either too small to be clinically diagnosed or that ischemic events produced by them are erroneously attributed to hemodynamic phenomena.

Aortic valvular surgery is associated with a higher incidence of calcific embolization than occurs spontaneously as shown by Holley in a separate autopsy series where he found 82 instances of embolization in 38 of the 62 patients who underwent closed valvotomy or aortic valve replacement and died at various intervals after surgery. A single case report of a calcific embolic occlusion of a femoral artery following retrograde left heart catheterization of a patient with CAS is the only evidence of a similar process occurring with angiographic intervention. Embolic occlusion of the circumflex branch of the left coronary artery by a presumably calcific embolus has also been described during thoracic aortography by Arvidson. Considering the difficulty that is encountered in traversing the aortic valve in tight AS during retrograde catheterization, a higher incidence of calcific embolization would be expected than suggested by the above reports.

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Figure 1. The M-mode echocardiogram shows heavily calcified aortic valve leaflets (arrow) with diminished valvular motion.

Figure 2. This non-enhanced CT scan of the head shows calcific emboli along the course of the right middle cerebral (black arrow) and right posterior cerebral (short black arrow) arteries. The left middle cerebral is seen within the sylvian fissure (white arrow).
Patients with CAS who experience ischemic stroke should be carefully evaluated for coexistent cerebrovascular disease or other cardiac sources of emboli before attributing the stroke to aortic valve disease. CT demonstration of calcific densities along the course of major intracranial arteries adjacent to a brain infarct is a useful radiographic finding that implicates CAS in stroke pathogenesis as a source of calcific emboli.14

Addendum

Post mortem examination ten months following stroke confirmed the persistent obstruction of intracranial arteries by calcific emboli.

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