Spontaneous Calcific Cerebral Embolus From a Calcific Aortic Stenosis in a Middle Cerebral Artery Infarct

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Calcific emboli from a calcific aortic stenosis is an uncommon event, usually following local trauma, as from cardiac surgery or left heart catheterization or as a sequel to bacterial endocarditis. We report what we believe to be the first case of a spontaneous calcareous embolus demonstrated by cranial computed tomography. In this patient, systemic hypertension and mild aortic insufficiency may have caused increasing mechanical forces acting on the aortic cusps and may have precipitated embolism. (Stroke 1989;20:691-693)

Calcific embolus (CE) is an uncommon complication of vascular calcified disease such as calcific aortic plaques, mural cardiac thrombi, or calcific aortic stenosis (CAS). Recently, Kapila and Hart1 have reported a case of cerebral CE following left heart catheterization in a patient with a CAS. Cranial computed tomography (CT) is helpful for proving such a phenomenon. We report what we believe to be the first case of a spontaneous stroke due to CE from CAS demonstrated by cranial CT.

Case Report

A 73-year-old right-handed man had the sudden onset of aphasia and right hemiparesis. He had been treated for hypertension over the previous 10 years. One year earlier, a pulsed Doppler echocardiogram carried out for effort-related cardiac symptoms of angina pectoris, dyspnea, and syncope showed moderately tight CAS. An aortic outlet of approximately 1 cm², with minimal aortic insufficiency and left ventricular hypertension, was demonstrated.

On admission 3 days after his stroke, right hemiparesis was no longer present. Neurologic examination showed a fluent Wernicke's aphasia, with dysgraphia and dyslexia. Electrocardiogram showed sinus rhythm and the sequelae of a posterolateral myocardial infarction. Electroencephalogram showed persistent left temporal abnormalities with monomorphic theta waves compatible with an ischemic stroke. Cranial CT scan showed a small, high-density image, interpreted as a CE, within the left sylvian region and a parenchymatous low-density area suggestive of acute infarction in the posterior territory of the middle cerebral artery (MCA) (Figure 1).

Twelve days after his stroke, left carotid angiography confirmed a left MCA partially obstructed just distal to the MCA bifurcation in the M2 segment (Figure 2), at a point closely correlated with the location of the CE on CT scan. Fundoscopy and fluorescein angiography provided no evidence of retinal artery CE.

The patient improved while receiving heparin therapy. Three weeks after his stroke, his aphasia was reduced to a semantic impairment.

Aortic valvular replacement was performed 4 months after the stroke under extracorporeal circulation to avoid recurrence of CE, although the risk of recurrence was uncertain and CAS was moderate by Doppler echography, with a mean aortoventricular pressure gradient of 34 mm Hg. After low aortotomy, examination of the aortic cusps showed the leaflets to be completely calcified, with perforation of the noncoronary cusp. The cusps were excised, and calcific anulus deposits were removed. A bovine prosthesis (Mitroflow No. 23, Symbion, Inc., Salt Lake City, Utah) was inserted. The postoperative course was uneventful after transient cardiac insufficiency.

On follow-up examination 10 months after his stroke, the patient spoke normally in usual conversation, with a mild semantic impairment and slow writing ability. Bioprosthesis function remained satisfactory.

Discussion

CAS is a common valvulopathy in elderly patients.1-6 However, arterial intracranial CE with
brain infarction occurs rarely. In a series of 103 patients with retinal artery occlusion and cardiovascular disease, CAS was discovered as the most common cardiac lesion in 11 of 29 cases. The incidence of spontaneous CE from CAS is doubtless underestimated. Forty-five instances of spontaneous CE were found in 37 of 165 patients with CAS who were examined anatomically; 32 CEs were observed in the coronary vessels, 11 in the renal vessels, one in the central retinal artery, and one in the MCA. In the latter case, although the MCA was incompletely obstructed, no neurologic deficit was reported and no infarct could be identified. In another autopsy series of 88 patients with CAS, six spontaneous CEs were identified: two in the cerebral vessels, two in the lower extremities, one in the coronary vessels, and one not specified. In a series of 81 patients with CAS studied postmortem, systemic CEs were found in one third. However, only one fifth of the CEs occurred spontane-

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