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

Gérald Rancurel, MD, Laure Marelle, MD, Didier Vincent, MD, Martin Catala, MD, Alexis Arzimanoglou, MD, and André Vacheron, MD

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 emboli 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 emboli (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 hypertrophy, 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 postero-lateral 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 spontaneous.
ously and most of these were observed after cardiac surgery, left heart catheterization, or bacterial endocarditis. A single pathologic case of CE from a CAS has been reported as showing a cerebral infarction; the calcific material had been displaced during cardiac surgery.

Intracranial CEs from CAS are better known in the ophthalmologic literature. Occlusion of collateral vessels of the central retinal artery by a CE may be observed by fundoscopy, appearing as irregular white bodies on the course of the artery in contrast to bright cholesterol crystals and soft, pliable, rapidly passing fibrin-platelet plugs. The small size of CEs explains their silent occurrence in cerebral arteries. In our case, however, fundoscopy did not show retinal CE nor retinal infarction.

Calcific occlusion might be completed by thrombosis. In one CAS case occurring after left heart catheterization, the femoral artery was obstructed by a CE, with poststagnation thrombus visualized by angiography.

Rarely, acute embolic occlusion by crucial material may be revealed on cranial CT scan as a spontaneous transitory high-density area in some major cerebral arteries. Five cases of CE have been demonstrated on CT scan by a calcific high-density area in the proximal cerebral arteries. These CEs originated from two carotid plaques, two mural thrombi (atrial or ventricular), and one calcific aortic cusp following left cardiac catheterization. In the latter case, CEs were demonstrated in the right posterior cerebral artery and in the right MCA, with acute brain infarction in the right MCA territory. CEs are easily distinguished from other vascular calcifications never seen on CT scan in these arterial territories.

The mechanisms of spontaneous migration of calcific deposits from a heavily calcified valve may be ulceration, friability, and disintegration combined with hemodynamic forces acting on the aortic cusps, such as violent ventricular contraction, tightness of the aortic orifice, high systolic blood pressure, and a high systolodiastolic pressure gradient. All these factors may account for the occurrence of spontaneous CE from CAS. In our case, CE occurred spontaneously, but associated hypertension and aortic insufficiency may have been contributory. Echocardiography did not reveal any other calcific source, and the left carotid artery was normal on angiography.

Treatment remains speculative. Heparin therapy does not seem to be a logical treatment of CE; heparin therapy may be hypothetically useful, however, for the treatment of associated poststagnation thrombus. The risk of recurrent clinical CE is unknown, but most postmortem studies have shown multiple arterial CEs. Hence, the necessity for valvular surgery is open to discussion once spontaneous clinical embolism has occurred, but there appear to be grounds for concern. Of course, hemodynamic features of CAS (such as blood pressure gradient, left ventricular hypertrophy, or associated aortic insufficiency) and the clinical symptomatology of the patient have to be taken into account in the surgical decision.

CAS is probably an underestimated cause of CE. CT demonstration of spontaneous CE in an artery adjacent to a brain infarct, without another source of CE, implies CAS as a mechanism in the stroke.

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References


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