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 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 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 Hart 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 posterolateral myocardial infarction. Electroencephalogram showed persistent left temporal abnormalities with monomorphic theta waves compatible with an isch-
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-

Figure 1. Cranial computed tomogram without enhancement showing high-density calcific embolus within left sylvian valley (left) and left posterior parenchymatous infarct (right).

Figure 2. Left carotid angiograms showing partial obstruction of middle cerebral artery just distal to bifurcation. Left: face; right: profile.
Calcific cerebral emboli 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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