Sonographic Confirmation of the Association Between Calcified Cerebral Emboli and Mitral Annular Calcification

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Mitral annular calcification (MAC), a noninflammatory chronic degenerative change of the fibrous support structure of the mitral valve, occurs more commonly in elderly women and patients with chronic renal failure. Although the causative relationship remains unclear, MAC is considered to double the risk of stroke and is known to be an uncommon cause of cardioembolic infarction. In this case, serial sonographic findings showed that MAC was a definite embolic source in a patient with recurrent calcified cerebral emboli (CCEs).

Case Report

A 76-year-old woman who was being treated for chronic heart failure and hypertension was admitted to our hospital because of a consciousness disturbance (day 1). Two months previously, she had brain infarction in the territory of the posterior circulation of the brain. Her neurologic symptoms improved completely, and she was discharged from our hospital taking aspirin. On physical examination on day 1, her pulse was regular (66 beats per minute), and her blood pressure was 120/70 mm Hg. Neurologic examination revealed drowsiness, nystagmus, left-sided ataxic hemiparesis, and hypoesthesia of the left leg. Her National Institutes of Health Stroke Scale score was 5. Laboratory examinations revealed normal calcium and phosphorus metabolism and kidney function. Abnormal results included brain natriuretic peptide, 1685 pg/mL (normal, 0.0–20.0 pg/mL), and D-dimer, 2.49 µg/mL (0.00–0.99 µg/mL).
On day 1, non–contrast-enhanced brain computed tomography (CT) revealed spots with calcific attenuation in the vessels in areas of both the anterior and posterior circulations, which had not been shown 2 months previously (Figure 1, A–D). Brain diffusion-weighted imaging did not show any acute infarctions. Multiplanar reconstruction images of electrocardiographically gated 64-detector row CT with contrast showed severe MAC (Figure 2). Transthoracic echocardiography (TTE), performed on day 2, showed a mobile component (7 × 7 mm) attached

Figure 1. Serial changes of CCEs (arrowheads) on CT of the brain. Calcified cerebral emboli, which had not been shown 2 months previously (A and B) are shown in areas of both of anterior (C) and posterior circulations (D) on images obtained at day 1. New CCEs are seen in the top of the basilar artery and the territory of the superior cerebellar artery (E) on images obtained at day 8 and in the territory of the right middle cerebral artery (F) on an image obtained at day 32.
to the ventricular aspect of the MAC (Figure 3A and Video 1). Transesophageal echocardiography failed to show the mobile component because of the acoustic shadow of the MAC, and no other cardiac embolic source was shown. These findings led to the diagnosis of CCEs that originated from a mobile component attached to the MAC.

To prevent recurrence, the patient received a heparin infusion, with the aim of maintaining the activated partial thromboplastin time at around 60 seconds. However, on day 8, sudden onset of the “top of the basilar artery syndrome,” with a consciousness disturbance, anisocoria, and left oculomotor paralysis, developed transiently (8 hours). Brain CT revealed new CCEs in the top of the basilar artery and the territory of

Figure 2. Multiplanar reconstruction images (A, axial; B, longitudinal) of electrocardiographically gated 64-detector row CT with contrast material show severe MAC (arrowheads).

Figure 3. Serial changes of TTE findings. On the apical 4-chamber view, a mobile component (7 × 7 mm; arrowhead) attached to the ventricular aspect of the MAC is seen in an image from day 2 (A), but it is reduced (3 × 3 mm; arrowhead) on an image from day 8 (B). It has expanded again (5 × 5 mm; arrowhead) on an image from day 62 (C).
the superior cerebellar artery (Figure 1E). Diffusion-weighted imaging showed acute infarction in the territory of the posterior cerebral artery (data not shown). Transthoracic echocardiography showed a reduced mobile component attached to the ventricular aspect of the MAC (3 × 3 mm; Figure 3B and Video 2). These facts confirmed that the mobile component attached to the MAC was the definite embolic source of CCEs.

Surgical resection of the mobile component was not considered suitable because of the patient's severe heart failure. It was decided that the antithrombotic therapy required intensification because the patient developed a transient consciousness disturbance and left-sided unilateral spatial neglect associated with new CCEs in the territory of the right middle cerebral artery (day 32; Figure 1F), and there was enlargement of the mobile component on the MAC (5 × 5 mm, day 62; Figure 3C and Video 3). Ultimately, triple antithrombotic agent (aspirin, clopidogrel, and warfarin sodium) therapy stabilized the clinical symptoms and the radiologic findings. The patient was able to walk with a cane (National Institutes of Health Stroke Scale score of 2 and modified Rankin Scale score of 2), and she was discharged from the hospital on day 72. The patient continued taking triple antithrombotic agents with strict blood pressure control. At a 1-year follow-up, the patient had no further ischemic events or adverse effects. Although a mobile component (5 × 2 mm) remained on the MAC, no further CCEs were seen on brain CT.

Discussion

Calcified cerebral emboli are considered to occur as a result of emboli from a calcified lesion near the heart, especially heart valves, to the cerebral circulation. They can occur spontaneously or as a complication of coronary or cerebrovascular angiography. There have been 5 cases reported as spontaneous CCEs that originated from calcific material of an MAC. All patients were female, ranging in age from 22 to 86 years. A mobile component attached to the MAC was shown only once before, but to our knowledge, there have been no descriptions of the serial changes of the mobile component in such a case. Thus, this report shows sonographic changes, before and after CCEs, of the mobile component attached to the MAC. Whether MAC contributes causally to the risk of stroke remains unclear, but this case might suggest that, although it is rare, there is a definite causal relationship between some embolic strokes and MAC.

The prognosis seems poor in patients with CCEs originating from MAC: of the 5 above-mentioned cases, only 1 had a good prognosis, whereas the other patients had recurrent cardiovascular events or died. Use of multiple antithrombotic agents, which is not recommended commonly because of the high risk of bleeding events, might have to be considered in patients with repeated CCEs. In fact, our patient was given 3 antithrombotic agents, and she had no further CCEs and a good prognosis. Such an effect indicates that CCEs might produce occlusion by fragments composed of thrombi and calcific material of the MAC.

References