Two Cases of Protruding Atherosclerotic Plaque with Mobile Projections in the Aortic Arch

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We recently studied two patients with cerebral embolism, in whom transesophageal echocardiography revealed protruding atherosclerotic plaques with freely mobile projections in the aortic arch. Ultrasonic imaging showed that the carotid artery was normal, and transthoracic and transesophageal echocardiography did not reveal a cardiac embolic source in either case. In one patient, we observed that an atherosclerotic plaque became ulcerated and developed mobile projections over the course of a year. More consideration should be given to the thoracic aorta as a source of embolism in patients with unexplained stroke.

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Introduction

Patients with unexplained stroke are often referred for transthoracic echocardiography (TTE). Although the heart is widely recognized as a source of systemic emboli, the thoracic aorta is not. We recently encountered two patients with unexplained stroke, in whom transesophageal echocardiography (TEE) unexpectedly revealed large protruding atherosclerotic plaques at the aortic arch with mobile projections that may have been a source of cerebral embolism.

Case Reports

Case 1

A 71-year-old man with angina pectoris, hypertension and diabetes mellitus visited the outpatient department of our hospital once a month. He was on anticoagulant and antiplatelet therapy. He was also a smoker. This patient was hospitalized with the sudden onset of left hemiparesis and dysarthria. On admission, his pulse was 58/min (regular) and blood pressure 148/84 mmHg. Physical examination revealed no carotid bruits, the lungs were clear, and the heart appeared normal. On neurological examination, the patient was alert and well oriented. His cranial nerves were normal. The tendon reflexes were increased on the left side, but no pathological reflexes were present. Tactile sensation was disturbed in the left upper and lower limbs. Cerebellar ataxia was not present. The electrocardiogram revealed left ventricular hypertrophy and sinus bradycardia. Chest X-ray films showed a prominent aortic knob. The fasting plasma glucose level was 89 mg/dl (normal: <110 mg/dl) and the HbAlc level was 7.0% (normal: 5.5–

Fig. 1. CT scan of the head showing a low-density area (arrow) in the right pons in case 1. This lesion is an infarct of the right pons.
6.9%). Serum lipid levels were within normal limits. Computed tomography (CT) of the head revealed infarction of the right pons (Fig. 1). TEE performed one year previously had incidentally demonstrated a protruding plaque (20mm x 10mm) in the aortic arch, which contained a cystic area and had small mobile projections on its surface (Fig. 2). TEE performed during hospitalization revealed that this lesion had become ulcerated and was associated with partially detached mobile portions of the tunica intima (Fig. 3). Ultrasonic examination of the extracranial carotid and vertebral arteries revealed no evidence of atherosclerotic obstruction or plaque. TEE revealed no evidence of a cardiac source of emboli nor left atrial spontaneous contrast (Moya-moya echo).

**Case 2**

A 65-year-old woman with hypertension, hyperlipidemia and diabetes mellitus was hospitalized due to the sudden onset of athetoid movements of the left arm. She was a nonsmoker. On admission, the pulse was 53/min (regular) and blood pressure 148/68 mmHg. Physical examination showed that no carotid bruit was present, the lungs were clear, and the heart was normal. On neurological examination, the patient was alert and well oriented, but had athetoid movements of the left upper and lower limbs and the mouth. Her cranial nerves were normal, as were her tendon reflexes. There was no laterality and no pathological reflexes were present. Vibration sense was disturbed in the lower extremities, but no cerebellar ataxia was present. The electrocardiogram was considered within normal limits except for sinus bradycardia. Chest X-ray films showed elongation and tortuosity of the descending aorta. The fasting plasma glucose level was 292 mg/dl (normal: <110 mg/dl) and the HbA1c was 15.1% (normal: 5.5–6.9%). The serum
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Fig. 4. CT scanning and magnetic resonance imaging of the head in case 2. Left: CT scan of the head showing a speckled high-density area in the right putamen (arrow). Right: T1-weighted magnetic resonance imaging of the head showing a high-intensity area in the right putamen (arrow). These findings indicate hemorrhagic infarction of the right putamen.

Fig. 5. Transesophageal echocardiograms of the aortic arch in case 2 showing a protruding atherosclerotic plaque with mobile projections (arrow). Top: Transverse view, Bottom: Sagittal view.

Triglyceride level was 190 mg/dl (normal: 30–160 mg/dl), but the other serum lipids were within normal limits. CT scanning and magnetic resonance imaging of the head indicated a hemorrhagic infarct of the right putaminal region (Fig. 4). Ultrasound examination of the extracranial carotid and vertebral arteries did not reveal any evidence of atherosclerotic obstruction or plaque, nor did TTE reveal any potential cardiac source of emboli. However, TEE revealed large protruding atherosclerotic plaques in the aortic arch with thin projections about 19 mm long which moved freely in the lumen with blood flow (Fig. 5). There was no left atrial spontaneous contrast (Moya-moya echo).

Discussion

The thoracic aorta has previously been described as a possible source of cerebral and peripheral emboli, but published studies on the etiology of stroke have not focused on the thoracic aorta as a source of embolism, perhaps due to the lack of reliable noninvasive methods for evaluating this vessel. However, TEE enables us to repeatedly investigate the thoracic aorta in a noninvasive manner. In fact, several studies focusing on the thoracic aorta as a possible embolic source have been published recently (1–8).

Amarenco et al (1) defined aortic ulceration as a disruption of the intimal surface that was visible on macroscopic examination. They studied the prevalence of ulcerated plaques in the aortic arch in 500 consecutive autopsy cases with cerebrovascular and other neurologic diseases. They reported that ulcerated plaques were present in 62 (26%) of 239 patients with cerebrovascular disease, but in only 13 (5%) of the 261 patients with other neurologic diseases. They also reported that the
prevalence of ulcerated plaques was 61% among 28 patients without a defined cause of cerebral infarction, as compared with 22% of the 155 patients having a known cause of cerebral infarction.

Karalis et al (4) defined intraaortic atherosclerotic debris as 1) disruption or marked irregularity of the intimal surface together with focal increase echogenicity and thickening of the adjoining intima, and 2) overlying shaggy echogenic material extending >5 mm from the aortic wall into the lumen. They reported that intraaortic atherosclerotic debris was identified in 38 (7%) out of 556 patients who underwent TEE, and that embolic events occurred in 11 (31%) of 36 patients with such debris. They also reported that the incidence of embolic events was higher when the debris was pedunculated and highly mobile [8 (73%) out of 11 patients] compared to layered and immobile debris [3 (12%) out of 25 patients].

Between March 1989 and September 1992, we performed TEE on 66 patients with cerebral infarction and 10 patients with transient ischemic attacks (TIA) in order to evaluate possible cardiac embolic sources and aortic plaques. The subjects consisted of 50 men and 26 women with a mean age of 65 years (range: 39 to 88 years). We defined atherosclerotic plaques as a focal increase in echogenicity along with intimal thickening extending >3 mm. Atherosclerotic plaques were present in the aortic arch in 15 (20%) out of 76 patients. Two of these 15 patients had protruding plaques with highly mobile projections in the aortic arch. In one patient, we observed a protruding atherosclerotic plaque that developed into an ulcerated plaque with highly mobile projections after cerebral infarction. In the two present patients, TEE revealed the presence of atherosclerotic plaques with intimal disruption and mobile projections in the aortic arch, while no potential embolic sources were detected in the heart or in the extracranial carotid and vertebral arteries.

We cannot definitely conclude that the ulcerated plaque with mobile projections was responsible for the cerebral emboli in both of these patients. As Karalis et al have stated (4), when atherosclerotic plaques are pedunculated and highly mobile, the incidence of an embolic event is high. In the present two cases, the atherosclerotic plaques were pedunculated and highly mobile, and we could not find any other cause of cerebral embolism. Therefore, these lesions may have been responsible for the cerebral emboli in our patients. Especially in case 1, the protruding atherosclerotic plaques in the aortic arch became ulcerated plaques with highly mobile projections after cerebral infarction, another finding suggesting that this lesion was responsible for the infarct.

Both of our patients were given anticoagulants. The use of anticoagulants in such a situation is controversial (9, 10). It has been suggested that anticoagulation may worsen the clinical syndrome, possibly by causing further plaque hemorrhage and thrombus superimposition (9). These patients have had no subsequent embolic events over a follow-up period of 2 years and 1 year, respectively.

Further studies are needed to assess the link between aortic arch plaque and cerebral infarction. TEE is an excellent method for detecting atherosclerotic plaques and for evaluating the thoracic aorta. Accordingly, the use of TEE is recommended in patients with unexplained stroke, even in those with a normal TEE.

References