A 41-year-old man was admitted to the emergency department for sudden-onset right hemiparesis. He had no history of diabetes mellitus, hypertension, arrhythmias, or stroke. However, he had a 20-year history of smoking, dyslipidemias, and hypothyroidism (laboratory test on admission revealed elevated thyrotropin [13.16 μIU/mL; reference, 0.27 to 4.2], normal T3 [0.84 ng/mL; reference, 0.58 to 1.59], and normal free T4 [0.81 ng/dL; reference, 0.70 to 1.48] levels). Magnetic resonance imaging of the brain revealed newly developed left cerebral infarction (Figure 1A and 1B). Brain computed tomographic (CT) perfusion imaging demonstrated perfusion defect in the left middle cerebral artery territory (Figure 1C), and CT angiography revealed total occlusion of the left internal carotid artery (Figure 1D). Chest radiography and ECG did not show any abnormalities. Transthoracic echocardiography was unremarkable except for mild aortic regurgitation. Laboratory tests for hypercoagulability and vasculitis were all negative. Two-dimensional transesophageal echocardiography (TEE) performed on admission day 7 demonstrated an 18×4.4-mm highly mobile echogenic mass attached to a minimally atherosclerotic wall of the distal aortic arch (Figure 2A and Movie I in the online-only Data Supplement). Real-time 3-dimensional technology with the use of an X7-2t TEE transducer and a commercially available ultrasound system (Phillips iE33, Andover, Mass) provided realistic movement of the mass and
the precise site of attachment on the aortic arch (Figure 2B and 2C and Movies II and III in the online-only Data Supplement). There was no evidence of thrombus in the left atrial appendage or patent foramen ovale on TEE. The patient was treated with intravenous unfractionated heparin and then warfarin (overlap with heparin for 3 days initially). Follow-up TEE performed after 9 days of anticoagulation revealed complete disappearance of the echogenic mass in the aortic arch (Figure 3 and Movies IV through VI in the online-only Data Supplement). On the basis of disappearance of the mass after anticoagulation therapy, we concluded that the echogenic mass was compatible with thrombus. After follow-up TEE, abdominal CT with contrast enhancement to exclude renal infarction as a cause of proteinuria showed no filling defect in the aorta and other organs (Figure 4). Six weeks later, after uneventful partial recovery, the patient was discharged with warfarin and levothyroxine therapy.

Laperche and colleagues\(^1\) reported 23 cases of mobile thromboses of the aortic arch without aortic debris from 27 855 TEE examinations with a monoplane, biplane, or omniplane probe. Even though very rare in normal or mild aortic atherosclerosis, we need to consider aortic thrombus as a source of sudden, otherwise unexplained embolism, especially in relatively young patients. Real-time 3-dimensional TEE for the evaluation of aortic thrombus or plaque may play a supplemental role because real-time volumetric image acquisition can provide 3-dimensional motion per se or an additional imaging plane for necessary information with high spatial resolution.

**Disclosures**

None.

**Reference**

Figure 4. Contrast-enhanced CT of the abdomen after follow-up TEE showed no filling defect in the spleen (A) and the kidney (B).
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