Acute Carotid T Occlusion in a Young Patient
Cryptogenic Origin?

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Case Description
A 32-year-old man without previous medical problems had acute global aphasia and right-sided hemiplegia (National Institutes of Health Stroke Scale [NIHSS] score 16 points) shortly after carrying a heavy mirror. Smoking (8 pack-years) was his only vascular risk factor. Computed tomographic angiography demonstrated a left carotid T occlusion (occlusion of the carotid artery, middle and anterior cerebral artery). Ninety minutes after symptom onset, intravenous thrombolysis was initiated with 80 mg recombinant tissue-type plasminogen activator, but successful reperfusion (Thrombolysis in Cerebral Infarction scale 3) was achieved only after thrombectomy with a Solitaire Stent (puncture 236 minutes and reperfusion 263 minutes after symptom onset). The thrombus was 20 mm long. Immediately after the intervention, his symptoms started to improve, and head MRI on the day after showed a residual 3.5 cm×1.5 cm×1.5 cm lesion in the left-sided basal ganglia on diffusion weighted imaging and that the recanalized carotid T stayed open (Figure 1). Secondary prevention with aspirin 100 mg/d and atorvastatin 40 mg/d was initiated. The sudden onset of physical straining and the large thrombus were suspicious for a cardioembolic source. Admission and follow-up ECG, 24-hour ECG recordings, and transesophageal echocardiography were normal. The only abnormalities of ancillary investigations were borderline-elevated high-sensitivity troponin T (hs troponin T; 0.015 ng/L; normal value <0.014 ng/L) and >3-fold elevated creatine kinase levels (750 μg/L; normal value <190 U/L). Creatine kinase levels declined, and troponin T levels were normal at follow-up examinations. Neurological signs continued to improve, and on day 4, clinical examination was essentially normal. The next night, transient aphasia and weakness of the right arm recurred for 2 hours. MRI did not show any new diffusion weighted imaging lesion but new irregularities of the left proximal and distal M1 segment of the middle cerebral artery, which were suspicious for spontaneously recanalized recurrent embolization (Figure 1). On transcranial Doppler on day 5 after stroke, there were microembolic signals in the left intracranial carotid artery and left anterior cerebral artery but not on the right side. Therefore, we decided to perform a cardiac MRI (CMR) on day 6 to search further for a cardiac source of emboli. CMR showed regional left ventricular (LV) apical wall thinning with dyskinesia and an apical thrombus (0.9×1.8 mm in size; Figure 2). LV ejection fraction was slightly reduced to 58%, and there was transmural late enhancement of the LV apex indicating subacute myocardial infarction (MI). Unfractionated heparin in therapeutic dosage was initiated. Coronary angiography on day 15 confirmed a tight proximal stenosis of the left anterior descending coronary artery and revealed multiple communicating linear filling defects in the distal segment of the left anterior descending suspicious for a spontaneously recanalized occlusion. Therefore, balloon angioplasty of the stenosis was performed, and a drug-eluting stent was deployed on day 15. Clopidogrel 75 mg/d was started, and heparin was switched to oral anticoagulation. We decided against a triple therapy with aspirin, clopidogrel, and oral anticoagulation to reduce intracerebral bleeding risk. On day 16, the patient was discharged home. At discharge, his NIHSS score was 3. In retrospect, the patient reported recurrent chest pain for 6 months, especially while jogging and when his heart rate exceeded 120 bpm.

Discussion
Undetected cardiac embolism is considered as a major mechanism underlying cryptogenic stroke.1 It can be challenging or even impossible to detect a cardiac source of embolism with the currently available diagnostic tests. MI is such an example. It is known that MI can cause abnormal LV wall motion, activate coagulation, and lead to regional thrombus formation and subsequent embolization and infarction of several organs including the brain.2 LV thrombus typically forms within 3 months after MI and most often after ST-segment–elevation myocardial infarction with severe disturbance of ventricular wall motion. Without antithrombotic medication, the risk of embolization in patients with LV thrombus is as high as 10% and persists for several months.1
MI typically starts/presents with chest pain, ECG abnormalities, and elevated cardiac enzymes. Because chest pain

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can be atypical or even absent and ECG nonspecific, the diagnosis often relies on cardiac biomarkers. Traditionally, creatine kinase (CK) and muscle–brain type CK levels were used for diagnosis, but their diagnostic accuracy is far from ideal. Accuracy improved with the introduction of Hs troponin T. Hs troponin T is highly sensitive and much more specific for myocardial damage than total serum CK levels or CK isoforms. Therefore, even when hs troponin T levels reach borderline levels, MI should be considered. Nevertheless hs troponin T is not absolutely specific. Mild elevations are encountered after rigorous physical exercise, with hypertensive crisis, acute LV failure, tachycardiac atrial fibrillation, pulmonary embolism, myocarditis, aortic dissection, or renal failure. In patients with stroke, interpretation of elevated troponin T, CK, and ECG changes is not straightforward. Catastrophic cerebral events such as subarachnoid or intracerebral hemorrhage or ischemic stroke can raise cardiac enzymes and provoke infarct signs in ECG through brain–heart interactions, presumably because of cardiac myocytolysis, a dissolution of myocytes that is associated with sympathoadrenal activation. Elevated troponin T levels are present in ≤10% of patients with stroke without underlying coronary artery disease. To date, little is known about the mechanism of this brain–heart interaction; however, involvement of the insular cortex increases the likelihood of it occurring. It is a matter of debate whether bigger infarct volumes are associated with an elevation of troponin. Three studies found an association of troponin levels and baseline NIHSS as an indirect marker of infarct size, but the only study that analyzed the infarct volume using the Alberta Stroke Program Early CT (ASPECT) score found no association.

In our patient, ancillary electrocardiographic and echocardiographic investigations did not show any signs of MI or other potential source of cardiac emboli. CK was only mildly elevated. Hs troponin T levels were borderline and could have been attributed to rigorous physical exercise, myocardial pathology resulting from the brain–heart interaction from stroke, or undetected primary myocardial damage. Because of the high level of suspicion for a cardiac source of emboli, we decided to proceed to CMR. CMR imaging is reported to have the highest sensitivity to detect LV thrombi and should be considered in patients with suspected cardioembolic origin of stroke. In addition, CMR can assess the size and structure of the heart including global and regional dysfunctions. Transthoracic echocardiography is also superior to visualize LV thrombi than transesophageal echocardiography and might also have been considered. In our patient, it detected an LV apical thrombus that had not been seen on transesophageal echocardiography. Whether transesophageal echocardiography missed the apical thrombus or whether the thrombus embolized during the first cerebral ischemic event and a new thrombus developed that was seen afterward by CMR remains unclear.

According to currently available clinical trials and the American Heart/Stroke Association guidelines, we treated our patient with oral anticoagulation (target international normalized ratio, 2.5; range, 2.0–3.0) for his LV thrombus. According to these guidelines, treatment should be for ≥3 months to reduce the risk of embolization (class I, level
of evidence B). Alternative antithrombotic agents for this situation have not been studied to date. Clopidogrel was added to anticoagulation because of the stent our patient had received.

**Disclosures**

Dr Arnold has received speaker’s fee from Covidien, Boehringer Ingelheim, and BMS and advisory board honoraria from Boehringer Ingelheim, Bayer, and BMS. The other authors report no conflicts.

**References**


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