A 56-year-old woman presented with acute confusion. Medical history was unremarkable, and she was not on any regular medication. On examination, she was hemodynamically stable and in sinus rhythm. She had global dysphasia, right-sided upper motor neuron facial paralysis, and hemiplegia as well as right hemineglect. Cardiovascular examination was unremarkable, and no carotid bruits were audible. The MRI of the brain revealed a large left middle cerebral artery territory infarct considerable mass effect (Figure 1a), and the magnetic resonance angiography revealed a thrombus within the mainstem of the left middle cerebral artery. A transeosophageal echocardiogram revealed a massive intracardiac thrombus extending through a patent foramen ovale (PFO) into the left atrium (Figure 2a). Atrial septal aneurysm was not evident. Carotid ultrasound of her neck was unremarkable. Multiple filling defects consistent with pulmonary emboli were seen in the right main pulmonary artery on a computed tomography pulmonary angiogram (Figure 1b). No intra-abdominal/pelvic mass was seen on computed tomography of the abdomen and pelvis.

Hypercoagulable and autoimmune states were excluded. She had global dysphasia, right-sided upper motor neuron facial paralysis, and hemiplegia as well as right hemineglect. Cardiovascular examination was unremarkable, and no carotid bruits were audible. The MRI of the brain revealed a large left middle cerebral artery territory infarct considerable mass effect (Figure 1a), and the magnetic resonance angiography revealed a thrombus within the mainstem of the left middle cerebral artery. A transeosophageal echocardiogram revealed a massive intracardiac thrombus extending through a patent foramen ovale (PFO) into the left atrium (Figure 2a). Atrial septal aneurysm was not evident. Carotid ultrasound of her neck was unremarkable. Multiple filling defects consistent with pulmonary emboli were seen in the right main pulmonary artery on a computed tomography pulmonary angiogram (Figure 1b). No intra-abdominal/pelvic mass was seen on computed tomography of the abdomen and pelvis.

Hypercoagulable and autoimmune states were excluded. She was anticoagulated with intravenous heparin. Repeat transeosophageal echocardiogram carried out 2 weeks later showed the patent PFO (mean diameter of PFO is 4.9 mm) with no residual thrombus (Figure 2b). A functional shunt was demonstrated by early passage of injected, aerated, saline microbubbles from the right-to-left atrial chambers. The PFO was subsequently closed percutaneously with a Premiere device (Figure 2c).

PFO has been reported to be present in a considerable number of individuals, with a prevalence of approximately 30% among patients with ischemic strokes. Several studies have suggested PFO as a significant risk factor for cryptogenic strokes. The mechanism underlying this phenomenon is postulated to be secondary to paradoxical embolism. This case clearly demonstrated paradoxical embolism through a PFO as a mechanism of ischemic stroke with evidence of cerebral embolism without a left-sided source, presence of pulmonary embolus, and demonstration of a right-to-left shunt.

In a meta-analysis of 9 case-control studies involving 566 patients and 458 nonstroke controls, young patients with a stroke had an odds ratio of 3.1 for having a PFO. The recurrent stroke rate among young cryptogenic stroke patients with a PFO is modest, but higher than normal for their age. About 1 in 42 young cryptogenic stroke patients with PFO alone will have recurrent stroke over a 4-year period (see review in Reference 4). Coexisting atrial septal aneurysm, albeit absent in this case, is a substantial potentiator of stroke risk in patients with PFO. PFO size, degree of functional shunting, and coexisting hypercoagulable state likely are additional risk factors.

Although there is no established treatment for patients with stroke and PFO, therapeutic options include antiplatelet agents, anticoagulants, surgical closure, or percutaneous closure devices. The completion of ongoing, randomized clinical trials comparing percutaneous closure devices with medical management will clarify whether the risks of the former are outweighed by a long-term reduction in recurrent vascular events.

This case illustrates the importance of investigating the existence of a PFO in patients with cryptogenic strokes, particularly in a younger population. In the presence of evident paradoxical embolism, percutaneous closure of PFO with antiplatelet/anticoagulant therapy was undertaken for secondary prevention of stroke.

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Disclosures
None.

References
Figure 1. a, MRI of the brain showed a left middle cerebral infarct with mass effect. b, Computed tomography pulmonary angiogram illustrated multiple pulmonary emboli in the right main pulmonary artery.

Figure 2. a, Transesophageal echocardiogram demonstrated a massive intracardiac thrombus extending through a PFO into the left atrium. b, Transeosophageal echocardiogram after anticoagulation revealed PFO with no residual thrombus. c, Transeosophageal echocardiogram confirmed closure of PFO with a percutaneous endovascular device (Premiere device) (See Data Supplement Movie).
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