A 57-year-old white woman, with history of hydatidiform mole 2 years earlier, was evaluated at our stroke center for the acute onset of intense cephalgia, vomiting, and aphasia. Recent medical history was significant for a vertebrobasilar transient ischemic attack 1 month earlier with a negative etiologic evaluation and negative vessel imaging and transesophageal echocardiography (TEE). A treatment by aspirin 160 mg was then started. On admission, neurological examination revealed a global aphasia and right homonymous hemianopsia. The National Institutes of Health Stroke Scale score was 8. She had no fever. Cardiovascular examination revealed a systolic murmur. Pulmonary auscultation was normal. A computed tomographic brain scan showed a left lobar temporal intracerebral hematoma with a subarachnoid hemorrhage in the ipsilateral sylvian fissure. Cerebral angiography and arteriography showed no vascular abnormality. Brain MRI showed a hemorrhagic infarction in the left middle cerebral artery territory and revealed several subacute infarctions in other vascular territories (Figure 1). Magnetic resonance angiography was normal. Coagulation tests involving fibrinogen, prothrombin time, activated partial thromboplastin time were normal. These multiple infarcts raised concern for a cardioembolic etiology to her stroke. The cardiac monitoring showed no arrhythmia. The transthoracic echocardiography showed an echogenic structure in the left atrium that was not clearly visualized (Figure 2). TEE revealed a 3-cm thrombus in the left atrium extending into a pulmonary vein (Figure 3, Movie 1 in the online-only Data Supplement). Anticoagulation by unfractionated heparin was started. Chest computed tomography and angiography confirmed the presence of the thrombus in the right inferior pulmonary vein contiguous to an invasive pulmonary mass of the right inferior lobe infiltrating the right inferior pulmonary vein (Figure 4). Abdominal and pelvic computed tomography were normal. Because the patient had a past medical history of hydatidiform mole, serum β-human chorionic gonadotrophin level is <2 IU/L. Those investigations led to the diagnosis of a pulmonary metastasis of a choriocarcinoma. A transbronchial lung biopsy was performed, but the biopsy material was not specific. Because increased β-human chorionic gonadotrophin is a very sensitive and specific test for the diagnosis of choriocarcinoma, histopathologic proof of choriocarcinoma is not always required to initiate therapy. The patient was then transferred to the oncology department where combination chemotherapy (etoposide, methotrexate, actinomycin D, cyclophosphamide, vincristine) was initiated. One month later the patient was functionally independent (modified Rankin Scale=2) and her National Institutes of Health Stroke Scale score was 3. At 3 months, the control TEE showed a persistent thrombus in the left atrium and the MRI was stable. Anticoagulation treatment was then pursued. Surgery was not proposed for several reasons: first, choriocarcinoma is very sensitive to chemotherapy, and, second, the thrombus was controlled after the initiation of anticoagulation. Surgical removal of choriocarcinoma is only recommended in cases of hemorrhage in patients with isolated tumors not responding to chemotherapy. Finally, the patient had a high surgical risk because of the recent stroke and cerebral hematoma.

Choriocarcinoma is a malignant neoplasm from the trophoblastic tissue. Increased β-human chorionic gonadotrophin levels, secreted by trophoblastic tissue, confirm the diagnosis. Hematogenous dissemination to the lungs, brain, liver, and vagina is common. Metastasis to the cerebral blood vessels may cause thrombosis or aneurysm formation and result in ischemic or hemorrhagic stroke. Choriocarcinoma is generally observed within a year after pregnancy, but can also be rarely found in postmenopausal women a long latent period after previous pregnancies. There are only a few case reports of metastatic choriocarcinoma as a cause of stroke, and, to our knowledge, none have been reported among postmenopausal women. Hypercoagulability and marantic endocarditis are other common mechanisms of stroke in patients with cancer. In the present situation, both initial normal vessel imaging and hypercoagulability testing were normal, whereas TEE disclosed the presence of a local thrombus in the vicinity of an infiltrative pulmonary mass. Therefore, thrombotic embolus was considered as the most plausible diagnosis, and
anticoagulation treatment was pursued in conjunction with chemotherapy.

In conclusion, this case report emphasizes the yield of TEE for the detection of an unusual cardioembolic cause of brain infarction. Furthermore, if it reveals left atrial thrombi extended into a pulmonary vein, pulmonary tumor must be sought.

**Disclosures**

None.

**References**


**Figure 1.** A, MRI axial T2*, recent temporal lobar hematoma (solid arrow) and subarachnoid hemorrhage in the sylvian fissure (dashed arrow). Axial diffusion-weighted imaging of restricted diffusion in the right middle cerebral artery (B), in the left posterior cerebral artery (C), and in the right posterior inferior cerebellar artery territories (D).

**Figure 2.** Two-dimensional transthoracic echocardiography (apical 4-chamber view) showing an echogenic structure in the left atrium (white arrow). LA indicates left atrium.

**Figure 3.** Two-dimensional transesophageal echocardiography (long axis) showing a 3-cm floating lesion within the left atrium, extended into a pulmonary vein (white arrow). LA indicates left atrium.

**Figure 4.** Computed tomography pulmonary angiography shows filling defects within the pulmonary veins (white arrow) and a pulmonary mass of the right inferior lobe, weakly enhanced by contrast that infiltrates the right inferior pulmonary vein.
Stroke Caused by a Pulmonary Vein Thrombosis Revealing a Metastatic Choriocarcinoma
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