A 25-year-old woman presented with acute unconsciousness. Examination revealed bilateral Babinski signs, fluctuating pupillary abnormalities (alternately widely dilated to miotic), upgaze paralysis, and multidirectional nystagmus.

Brain MRI sequences at 3 hours after onset revealed bilateral hyperintense signals in the thalami, consistent with infarcts (figure, A), as well as in the distal basilar trunk and posterior cerebral arteries (figure, B), consistent with intravascular thrombi. MR angiography confirmed occlusion of both posterior cerebral arteries.

No hypercoagulable state, cardioembolic source, or dissection was identified. There was a family history of hemorrhagic telangiectasia (Osler-Weber-Rendu syndrome), and chest CT scan demonstrated pulmonary arteriovenous fistulas (figure, C). Deep venous thrombosis was not identified.

Paradoxical embolization is considered the likely predominant mechanism of cerebral ischemia in patients with pulmonary arteriovenous fistulas.1

REFERENCE

Figure Brain MRI 3 hours after onset (A, B) and chest CT (C)

(A) Axial diffusion-weighted MRI shows high-intensity signals in the bilateral medial thalami. (B) Axial FLAIR MRI shows high-intensity signals in the distal basilar and posterior cerebral arteries. (C) Chest CT demonstrates two left pulmonary arteriovenous fistulas.
Teaching NeuroImage: Pulmonary arteriovenous fistula and top of the basilar infarction
Bruno Barroso
Neurology 2008;71:e42
DOI 10.1212/01.wnl.0000327603.42444.01

This information is current as of October 13, 2008