Bicaudate infarcts in the setting of congenital absence of A₁ segment

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A 51-year-old woman with a history of hypertension and smoking presented with sudden onset of confabulation, delusions, and blunted affect. She was oriented to time, place, and person with intact language, strength coordination, and sensation. Gait was slow and wide-based. Serum and CSF yielded no abnormalities. Brain MRI showed bilateral caudate infarcts (figure 1). Cerebral angiography did not provide evidence of dissection or stenosis of the internal carotid artery or major atherosclerosis intracranially or involving the aortic arch. However, it showed an embolus in the right A₁, with congenital absence of the left A₁ segment; there was a thrombus at the origin of the right internal carotid artery (figure 2). No source of thromboembolism was identified on transthoracic echocardiography. Infarction spared the more distal cortical branches of anterior cerebral artery territories because these benefited from a retrograde filling through middle cerebral artery and posterior cerebral artery pial collaterals, more developed on the left. This congenital vascular variant, one that clinicians should be cognizant of, leads to the bilateral nature of the infarcts.^{1,2}

Figure 1 Brain MRI

Brain MRI diffusion-weighted and apparent diffusion coefficient images (A–C) demonstrate restricted diffusion in bilateral caudate and putamen extending to the right frontal lobe with associated fluid-attenuated inversion recovery hyperintensity (D).

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Funding information and disclosures are provided at the end of the article. Full disclosure form information provided by the authors is available with the **full text of this article** at Neurology.org/cp.

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Figure 2 Cerebral angiogram

Cerebral angiogram showing adherent thrombus at the origin of right internal carotid artery (arrow, A), occlusive thrombus in right A₁ segment (arrows, B and D), and absence of left A₁ segment (C). Pial collaterals from the middle cerebral artery on right (E) and left (F).

REFERENCES

1. Kang SY, Kim JS. Anterior cerebral artery infarction: stroke mechanism and clinical-imaging study in 100 patients. *Neurology* 2008;70:2386–2393.
2. Yamaguchi K, Uchino A, Sawada A, Takase Y, Kuroda Y, Kudo S. Bilateral anterior cerebral artery territory infarction associated with unilateral hypoplasia of the A1 segment: report of two cases. *Radiat Med* 2004;22:422–425.

AUTHOR CONTRIBUTIONS

Pouya Tahsili-Fahadan and Noushin Yahyavi-Firouz-Abadi drafted the manuscript. Salah Keyrouz and Alan Pestronk edited the manuscript.

STUDY FUNDING

No targeted funding reported.

DISCLOSURES

P. Tahsili-Fahadan, N. Yahyavi-Firouz-Abadi, and S.G. Keyrouz report no disclosures. A. Pestronk serves on a scientific advisory board for The Myositis Association; is author on the following patents: (1) TS-HDS antibody 7,175,989, issued 2007; (2) GALOP antibody 6,121,004, issued 2000; (3) GM1 ganglioside antibody 6,077,681, issued 2000; and (4) Sulfatide antibody 6,020,140, issued 1995; has received speaker honoraria from Athena and The Myositis Association; receives/has received research support from Genzyme, Inmed, Knopp, Ultragenyx, ISIS, Sanofi, Cytokinetics, GSK, Biogen, CSL Behring, NIH, Washington University Neuromuscular Research Fund, CINRG Children's Hospital Washington DC, and Muscular Dystrophy Association; holds stock in Johnson & Johnson; and receives license fees from Athena and royalty payments for antibody testing patents. Full disclosure form information provided by the authors is available with the **full text of this article at Neurology.org/cp**.