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Delayed sudden coma due to artery of percheron infarction

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Delayed Sudden Coma Due to Artery of Percheron Infarction

A 52-YEAR-OLD MAN was noted to display "unusual behavior" with transient agitation and blurry vision after otherwise uneventful diagnostic cardiac catheterization. Several hours after same-day discharge from the hospital, he suddenly became comatose, requiring intubation and admission to the intensive care unit. Two days later, he regained consciousness and was noted to have vertical gaze palsy and dysarthria without other neurologic deficits. Magnetic resonance imaging demonstrated bilateral acute medial thalamic ischemic strokes (**Figure**). Magnetic resonance angiography did not display extracranial or intracranial arterial stenosis (not shown). At 3 months' follow-up, he had only mild residual dysarthria.

COMMENT

The thalamic vascular supply is categorized into the anterior, paramedian, inferolateral, and posterior territories.[1] Percheron[2] described 3 anatomic variations of the paramedian arteries: in the most common variant (type A), small perforating arteries arise from both P1 segments of the posterior cerebral artery. Type B, also called the artery of Percheron, is characterized by an asymmetrical common trunk arising from one P1 segment. Type C is defined by an arcade emanating from an artery bridging the two P1 segments. Occlusion of the artery of Percheron characteristically results in bilateral medial thalamic infarction with or without rostral midbrain involvement.[2] The clinical presentation may include disorientation, memory impairment, behavioral abnormalities, dysarthria, hypophonia, and dysprosody, often accompanied by eye movement abnormalities, including vertical gaze palsy.[1,3] Impairment of arousal with decreased and fluctuating levels of consciousness is a remarkable characteristic frequently observed within the first few days.[1] Coma may be the initial presenting symptom or it may develop progressively over time.[2,4] However, to our knowledge, ours is the first report of delayed sudden coma complicating bilateral thalamic infarction with an innocuous prodrome. The patient described herein recovered almost completely, likely because his stroke was limited to the bilateral paramedian thalamus not involving the midbrain, which has previously been associated with a favorable outcome.[4] His vertical gaze palsy may be explained by disruption of cortical inputs that traverse the thalamus on their way to the rostral interstitial medial longitudinal fasciculus.

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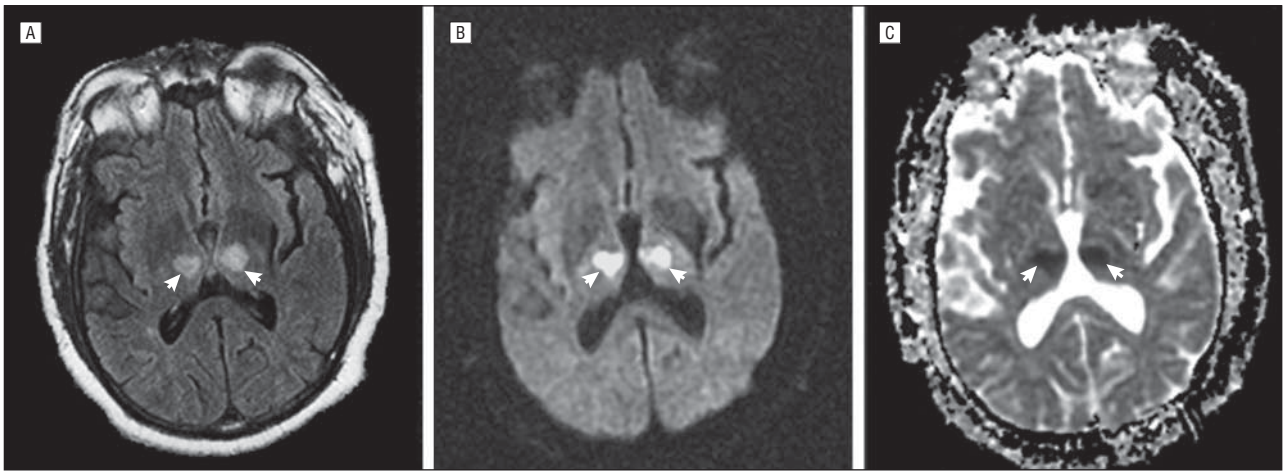

Figure. Magnetic resonance image of the brain indicating bilateral thalamic infarction. A, Fluid attenuated inversion recovery imaging showing high signal intensity in bilateral medial thalami (arrows). Axial diffusion-weighted image (B) and corresponding (C) apparent diffusion coefficient (ADC) map obtained 24 hours after symptom onset showing respective restricted diffusion and reduced ADC values (arrows), indicating acute bilateral paramedian thalamic infarction.