

Bilateral Thalamic Infarction

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ABSTRACT

Bilateral thalamic infarction is a rare cerebral vascular disease. The most common findings are acute impairment of consciousness, vertical gaze paresis, cognitive disturbances, and abnormal behavioral symptoms. In this paper, we report a case of bilateral thalamic infarction that resulted in abnormal behavior.

Keywords: Behavioral symptoms, unconsciousness, thalamic infarction

Introduction

Bilateral thalamic infarctions are a rare entity. A study demonstrated that they account for only 0.6% of all cerebral infarctions (1). The most common findings are acute impairment of consciousness and cognitive disturbances. Usually, they are accompanied with ocular signs. In some cases, corticospinal tract is not affected; thus, the loss of strength in the extremities does not arise. In this paper, we report a case of bilateral thalamic infarction with a history of hypertension, resulting in abnormal behavior.

Case Presentation

A 72-year-old woman presented with a sudden onset of abnormal behavior for a 1-week duration. She was not febrile but was hypertensive, with a blood pressure of 150/100 mmHg. She was somnolent but responded to verbal commands and showed no focal neurological deficits. She was disoriented to time, place, and person. Her eye movements were smooth and no nystagmus was observed. She spoke in a weak voice with dysarthria and could follow only one-step simple commands. She had been on irregular medications for hypertension. There was no history of injury, fever, or abnormal behavior.

Laboratory investigations were unremarkable. Drug screening for benzodiazepines, opiates, and barbiturates was negative. An unenhanced T1 magnetic resonance imaging (MRI) of the brain performed promptly revealed faint hypodensities in both paramedian thalami, and T2 MRI demonstrated hyperdensity in them (Fig 1). An ultrasound examination of the carotid artery and vertebral duplex was normal. Echocardiography showed no obvious valvular lesion or intracardiac thrombus. With the exception of hypertension, neither proximal source of embolism nor other underlying pathology was identified.

Discussion

The artery of Percheron is an uncommon anatomic variant where a solitary trunk originates from one of the posterior cerebral arteries (PCA) and provides arterial supply to both paramedian thalami and the rostral midbrain (2,3).

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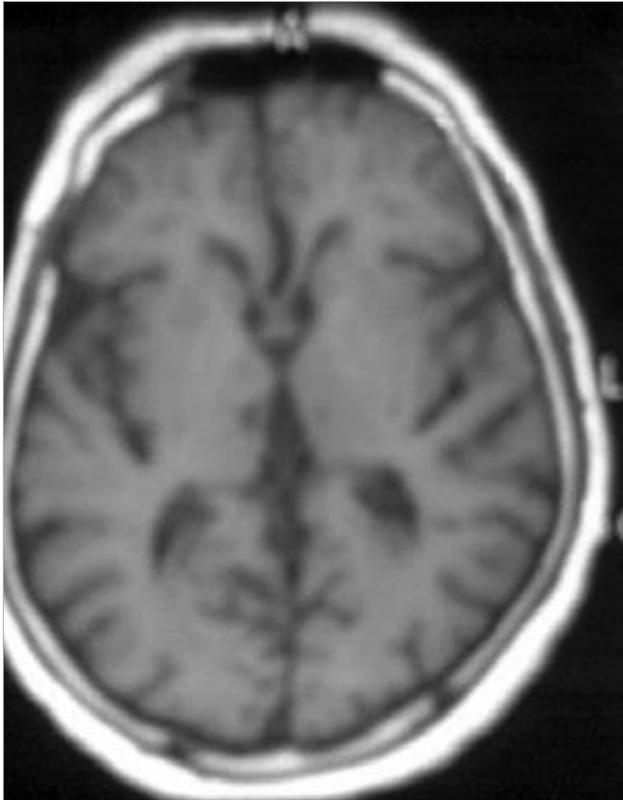


Figure 1. T1-weighted MR images revealed faint hypointensities at the both paramedian thalami

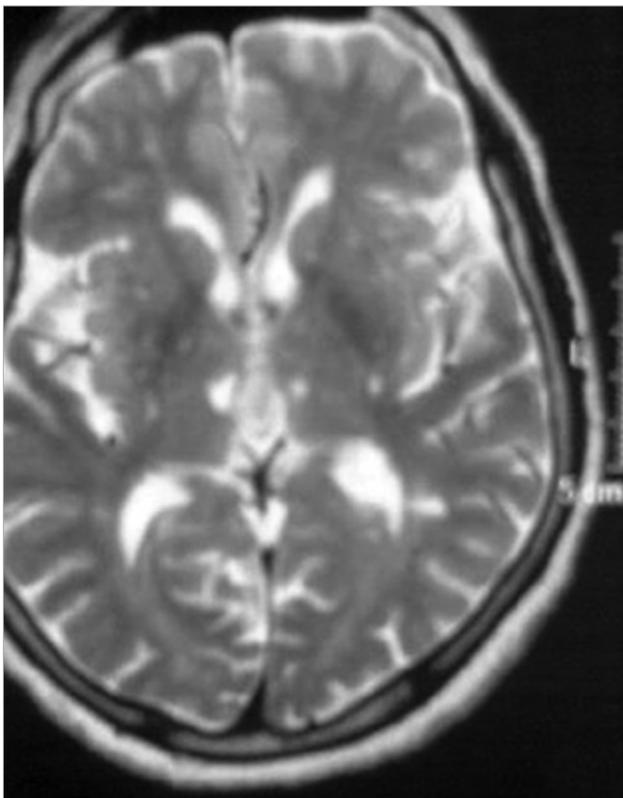


Figure 2. T2-weighted MR images demonstrated hyperintensity at the same region which were both consistent with bilateral thalamic infarct

Posterior circulation ischemic stroke often mimics other pathological processes including intracranial hemorrhage, infection, and inflammation, all of which were excluded in our patient by subsequent investigation. Her fever was normal, and laboratory tests were unremarkable.

We describe a patient with ischemic infarct involving both paramedian thalami, presenting with marked somnolence, abnormal behavior, and mild dysarthria as the only neurological signs. Occlusion of “top of the” basilar artery or of the Percheron artery, a developmental variant replacing the perforating medial thalamic arteries may result in acute infarction in both thalami. In our patient, the cerebral areas supplied by PCAs and superior cerebellar arteries did not show any evidence of ischemia on MRI. Similar clinical and neuroimaging features could be seen in patients with thrombosis of deep venous sinuses. MR venography of our patient revealed patent dural venous sinuses and major cerebral veins. Moreover, thrombophilia and vasculitic screen were unremarkable.

The patient’s clinical features were consistent with bilateral paramedian thalamic infarction, which typically involves altered mental status and memory impairment. The third feature of bilateral paramedian thalamic infarction is vertical gaze palsy (4,5). Altered consciousness is explained by the involvement of reticular activating system and disrupted connections between the thalamus and the anterior, orbitofrontal, and medial prefrontal cortices. In particular, vertical gaze palsy has previously been described in the absence of midbrain involvement (4).

Conclusion

Occlusion of the bilateral thalamic infarction usually occurs because of small vascular disease. Our patient also had hypertension. Echocardiography and duplex USG were normal. We wanted to emphasize that bilateral thalamic infarction rarely mimics other pathological processes such as psychiatric disorders. For the differential diagnosis in patients presenting with acute confusional states, bilateral thalamic infarction should be taken into account.

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