



Case Report

Bilateral thalamic infarct as a diagnosed conversion disorder

Abstract

Bilateral thalamic infarcts are an uncommon type of cerebral infarct. Bilateral paramedian thalamic infarctions may lead to a severe impairment of consciousness. The sudden onset of a lethargy or comatose state, in the absence of motor deficits, easily evokes the idea of a subarachnoid hemorrhage. Other patients present with behavior changes, disorientation in space and time, memory loss, or thought disorders. We believe that bilateral thalamic infarction is often missed in emergency department (ED) in relatively young patients, especially when magnetic resonance imaging is not performed. In these cases, the patient can be discharged with various psychiatric diagnoses. We suggest that bilateral thalamic infarct should be considered in patients in the ED with new diagnoses of conversion disorder.

The thalamus is an important strategic diencephalic structure of the human brain. Bilateral thalamic infarcts are an uncommon type of cerebral infarct [1]. They represent approximately 0.6% of first ever acute strokes. The infarcts are usually asymmetrical, and the paramedian thalamic region is the most commonly affected location [2,3]. The paramedian syndrome involves an acute loss or reduction of consciousness, often associated with oculomotor and neuropsychological disturbances [4].

The thalamus is supplied by many perforating arteries, which exhibit complex distribution with many variations. A rare variant is the artery of Percheron type II, which is a single arterial trunk that provides arterial supply to the paramedian thalami and rostral midbrain bilaterally [5]. Occlusion of this artery results in bilateral thalamic and mesencephalic infarcts [6,7]. The clinical manifestations include disorientation, confusion, hypersomnolence, deep coma, akinetic mutism, amnesic syndrome, and various neuropsychological phenomena. Speech and language dysfunctions, such as dysarthric and aphasic disturbances, hypophonia, and dysprosody may also be detected [8].

A 51-year old man was brought to the emergency department (ED) with impairment of consciousness. The patient's medical history included a mitral valve replacement, performed 20 years ago for which he had been taking warfarin sodium. He had abruptly discontinued the warfarin 1 month ago without consulting a physician. On physical examination, the patient appeared unwell; he was lethargic, apathetic, and agitated. His Glasgow coma score was 11 (eyes, 3; verbal, 4; motor, 4). His vital signs were as follows: blood pressure 125/76 mm Hg, pulse rate 53 beats per minute (regular), and axillary body temperature 37.6°C. Neurologically, the patient's pupils were isochoric, pinpoint, and not reactive to light bilaterally. Brain stem reflexes (oculocephalic, pharynx) were both intact. There were no motor deficits and no facial asymmetry. Hoffmann-Trömner and Babinski reflexes were bilaterally negative. Other system findings were within normal limits. Computed tomography of the head

showed a hypodense area, 15 mm in diameter, more prominent on the left than on the right side of thalamus. This imaging was consistent with a bilateral acute thalamic infarct (Fig. 1). Diffusion-weighted magnetic resonance imaging (MRI) showed that there is restriction of diffusion compatible with acute infarction within the bilateral thalami more prominent on the left side than on the right side of the thalamus (Figs. 2 and 3). Echocardiogram showed no thrombosis in the heart chambers. In addition, there were no pathologic or spectral findings identified by color Doppler imaging of the carotid vertebral artery.

The patient was admitted to the intensive care unit as with acute bilateral thalamic infarction. On the ninth day after admission, the patient became more conscious, and 16 days later, he was transferred to the neurology ward. The patient was discharged from the hospital with emotional lability and lethargy, and he was counseled to follow up regularly as an outpatient. In outpatient controls of our patient, the lethargy has been noted to be permanent.

The patient's history revealed that he was first seen at a city hospital where he was visiting a relative. While he was there, he experienced dizziness, lightheadedness, and altered mental status. The patient was admitted to the ED at this hospital and was diagnosed with conversion disorder. A 10-mg diazepam infusion was administered in 1 hour. After no improvement in the patient's symptoms after 8 hours of observation, the patient was referred to our hospital.



Fig. 1. Computed tomography of the head: Hypodense area 15 mm in size on anterior-inferior thalamus.

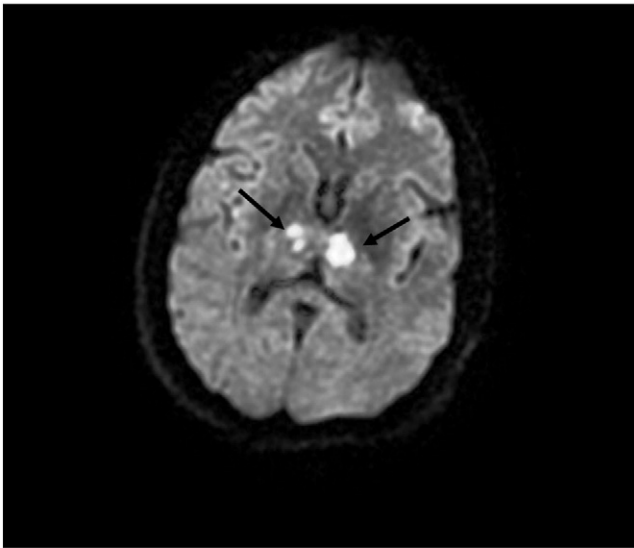


Fig. 2. Diffusion-weighted MRI: Restriction of diffusion, which is compatible with acute infarct at bilateral thalamic levels.

Thalamic infarctions result in markedly different clinical syndromes depending on the location of the infarction. Paramedian thalamic infarctions are characterized by supranuclear palsy, somnolence, neuropsychological deficits, and memory impairment. Bilateral paramedian thalamic infarctions may lead to a more severe impairment of consciousness [9]. Disorientation, confusion, hypersomnolence, deep coma, coma vigil or akinetic mutism, and severe memory impairment with perseveration and confabulation are prominent behavioral features, often accompanied by eye movement abnormalities. Apathy and anterograde and retrograde memory deficits, as exhibited by our patient, can be severe and persistent [3,8].

Previous reports have also described disorders of taste, smell, and appetite with bilateral paramedian thalamic infarctions, but these manifestations were not observed in our patient, who presented with isolated symptoms of altered of consciousness and somnolence.

Although isolated thalamic infarcts were reported for the first time more than 100 years ago by Dejerine and Roussy, they remain rare and account for only 11% of all vertebrobasilar infarcts. They may present only with alteration of consciousness and neuropsychiatric symptoms. Because the vessels are too small to be visualized by

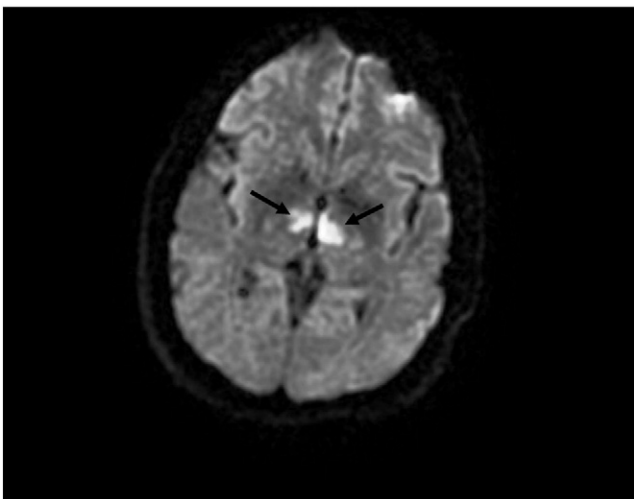


Fig. 3. Diffusion-weighted MRI: Bilateral thalamic infarcts, more prominent on the left side than on the right side of the thalamus.

angiography, conventional angiography is not indicated for demonstrating occlusion and establishing the diagnosis. If thalamic infarct is suspected, an MRI should be performed [1,3,6].

The paramedian nuclei are associated with alertness; nociception; sensation; motor functions; speech; regulation of cognitive function; and establishment of memory, mood, and motivation. Vascular lesions destroy these nuclei in different combinations and produce sensorimotor and behavioral syndromes depending on which nuclei are involved [8,9]. The chief cause of bilateral paramedian thalamic infarctions is small artery disease and is typically due to emboli (60%). The other main cause of thalamic infarctions is cardioembolism (40%), originating from the heart, aorta, and proximal cervicocerebral vessels, especially in young people [3,7,8]. In our case, the patient had been taking warfarin sodium for anticoagulation due after mitral valve replacement. However, he had discontinued warfarin without consulting a physician. Therefore, it is likely that the patient's infarct was the result of a cardioembolism.

The artery of Percheron, an asymmetrical common trunk arising from a P1 segment of the posterior cerebral artery, is a peculiar presentation of the 3 variants involved in the irrigation of the paramedian thalamic territory [4]. In type I, which is the most common variation, there are many small perforating arteries arising from P1 segment of posterior cerebral artery. In type II, also known as the artery of Percheron, a single perforating blood vessel arises from 1 P1 segment and supplies bilateral thalami. In type III, an arcade of perforating branches arises from an artery bridging the P1 segment of both posterior cerebral arteries. These vessels are too small to be visualized by angiography [8]. Because our patient had a bilateral thalamic infarction, we suspect that his thalamic vascular supply was a type II variation.

The medial thalamic nuclei lie in the territory of the artery of Percheron; these nuclei play a substantial role in sleep regulation. In our patient, their impairment caused a global sleep disruption, including disorders of the sleep/wake circadian cycle, and a disruption of sleep macrostructure and microstructure. In conclusion, small, "strategic" thalamic lesions, affecting the thalamic arousal system, may severely impair the main cortical activation networks, resulting in an impairment of consciousness and a severe "multilevel" disruption of sleep [4,10].

The vertical gaze palsy reported in cases of paramedian thalamic infarcts is due to associated involvement of the rostral midbrain tegmentum, including the interstitial nucleus of Cajal. Vertical gaze paresis localizes the lesion to this location. The sudden onset of a stuporous or comatose state, in the absence of motor deficits, raises concern for a subarachnoid hemorrhage. However, in other patients with thalamic infarct, symptoms include behavior changes, disorientation in space and time, memory loss, or thought disorders. These features are also suggestive of a metabolic or toxic encephalopathy [6,8]. Our patient did not present with vertical gaze palsy, suggesting that these nuclei were not affected. Because of the nonspecific clinical picture, our patient was diagnosed with conversion disorder by physicians in an outside hospital's ED.

Our case presented with no motor and sensory deficits, a deep sleep disorder, loss of consciousness, and normal blood tests. The computed tomographic imaging was assessed as within normal limits. Because of these findings, our patient had been diagnosed with conversion disorder by the initial hospital's ED and treated with diazepam. Our experience demonstrates that thalamic infarction should be considered in patients who present with abrupt changes in consciousness, sleep disorders, impaired response to environmental stimuli, speech disorders, and normal motor and sensory findings.

Bilateral thalamic infarcts are uncommon types of ischemic stroke. After the diagnosis is established by using adequate neuroimaging, further evaluation to define the cause should be conducted. Bilateral thalamic infarctions are characterized by disorders of consciousness, memory dysfunctions, various types of vertical gaze paresis, and psychologic changes. We believe that bilateral thalamic infarcts are

often missed in the ED in relatively young patients, especially if an MRI is not performed. In these cases, the patient is subsequently discharged with various psychiatric diagnoses. We suggest that bilateral thalamic infarcts should be considered in patients seen in the ED with new diagnoses of conversion disorder.

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