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CASE REPORT

Agitation and somnolence by bilateral paramedian thalamic infarct

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Key Clinical Message

Bilateral thalamic infarction in paramedian artery territory may present with severe acute illness, confusion, coma and memory impairment. However, subtle clinical presentation as in our case should alert the clinician to consider such a diagnosis as it can be associated with good prognosis.

Abstract

Bilateral thalamic infarct is a rare form of stroke. Mostly thalamic infarcts are unilateral. In most cases, bilateral thalamic infarction leads to cognitive dysfunction, opthalmoparesis, conscious impairment, behavioral disturbance, and corticospinal dysfunction. Here, we describe the case of a 75-year-old male patient who presented to the emergency department of our hospital with agitation and somnolence for one day. He had poorly controlled hypertension. There was no previous history of stroke, diabetes mellitus, hyperlipidemia, known cardiac disease, or smoking history. There was no seizure, recent headache, or visual disturbance. The patient was somnolent and not oriented to time, person, or place. Neurological examination did not show any focal weakness or vertical eye movement restrictions. Other systemic examinations, including those of the respiratory and cardiovascular systems, were unremarkable. Extensive laboratory investigations excluded potential metabolic, infectious, endocrine, or toxic etiologies. The patient did not have any recent history of drug misuse, including benzodiazepines. Brain MRI with diffusion-weighted imaging showed an acute bilateral thalamic infarct. Cerebral angiography was unremarkable. The patient was treated with low molecular weight heparin 60 mg subcutaneously, aspirin 300 mg daily, and haloperidol 5 mg twice daily for agitation. After two weeks of intrahospital treatment, his condition improved (consciousness and orientation massively improved).

KEYWORDS

agitation, bilateral thalamic infarct, cognitive dysfunction

JEL CLASSIFICATION
Neurology, Neurosurgery, Social Care

1 | INTRODUCTION

The thalamus is one of the most important units of the human brain and is made up of five major functional units that involve high-level cognitive processes, sensory and motor functions, language, as well as emotions and motivation.[1,2] Bilateral thalamic infarction is a rare form of cerebral infarction. Previous studies have shown that it accounts for only 0.6% of all forms of cerebral infarcts.[3,4]

The thalamus has a complex arterial supply. The arterial supply of the thalamus consists of an intricate arterial circuit arising from both the anterior and posterior circulations of the brain. The internal carotid artery supplies mostly the anterior segment of the thalamus, whereas the medial, lateral, and posterior areas receive their blood supply via the vertebrobasilar system.[5] The posterior cerebral artery's P1 portion gives rise to the paramedian arteries, which serve both sides of the thalamus. The paramedian thalami and the rostral midbrain both can receive vascular feed from the artery of Percheron, an anatomical variant that arises from one of the posterior cerebral arteries. As a result, an occlusion of the artery of Percheron can lead to a bilateral thalamic infarction with or without involvement of the midbrain.[4,6]

Although bilateral thalamic involvements are less prevalent yet, they cause more severe behavioral disturbances than those with unilateral involvement.[7] Patients with bilateral thalamic infarction may present to the emergency room with disorientation, confusion, hypersomnolence, a deep coma, or akinetic mutism. Other documented features include memory impairment, sensory loss, and motor weakness associated with restricted eye movements. Agitation or acute behavioral disturbances may be the only presentations.[6,8] In this paper, we report a case of bilateral thalamic infarction with a history of hypertension, presenting with agitation and somnolence without corticospinal tract involvement.

2 | CASE PRESENTATION

A 75-year-old male patient presented to the ER of our hospital with agitation and lethargy for one day. He had chronic hypertension and was on Amlodipine 5 mg daily, but this was not sufficient to control his blood pressure. There was no previous history of stroke or known cardiac diseases. Likewise, he had no history of diabetes mellitus, hyperlipidemia, or smoking. He had no known psychiatric illness. On examination, he was afebrile and hypertensive (his BP was 160/110 mmHg on admission). The patient was somnolent and not oriented to time, person, or place. His GCS (Glasgow Coma Scale) was 9/15. A neurological examination did not show any focal or lateralizing deficits. There was no seizure, recent headache, or visual disturbance. There was no apparent restriction of eye movements. Other systemic examinations, including those of the respiratory and cardiovascular systems, were unremarkable. Extensive laboratory investigations excluded potential metabolic, infectious, endocrine, or toxic etiologies. The patient did not have any recent history of drug misuse, including misuse of benzodiazepines. Non-contrast brain CT was unremarkable. Brain diffusion MRI showed acute bilateral thalamic infarction (see Figure 1A,B). Cerebral angiography was unremarkable. The ECG did not reveal atrial fibrillation or arrhythmia. Echocardiography did not show any potential cardioembolic source. The patient was treated with low molecular weight heparin 60 mg SC, aspirin 300 mg daily, and haloperidol 5 mg twice daily. After two weeks of intrahospital treatment, his condition improved (consciousness and orientation massively improved). He was discharged for outpatient neurology clinic follow-up.

At one-month post discharge, the patient was re-evaluated in the outpatient clinic, the agitation had disappeared, there has been a significant improvement in cognitive function of the patient and his memory was intact.

3 | DISCUSSION

Acute bilateral thalamic infarction is rare, accounting for about 0.6% of acute ischemic strokes. The majority of thalamic infarctions are unilateral.[9] The thalamic nuclei are complex and made up of five main functional units: reticular and intralaminar nuclei that involve consciousness state and nociception; sensory nuclei that connect sensory input to the cerebral cortex (medial geniculate nucleus (MGN; auditory), lateral geniculate nucleus (LGN; visual), and the ventrobasal complex); effector nuclei involving in language and motor processing; associative nuclei, which is crucial in advanced cognitive processes; and limbic nuclei that are involved in affective behaviors such as stress, anxiety, and agitation.[10]

The paramedian thalamic artery, which arises from posterior cerebral and communicating arteries, is the

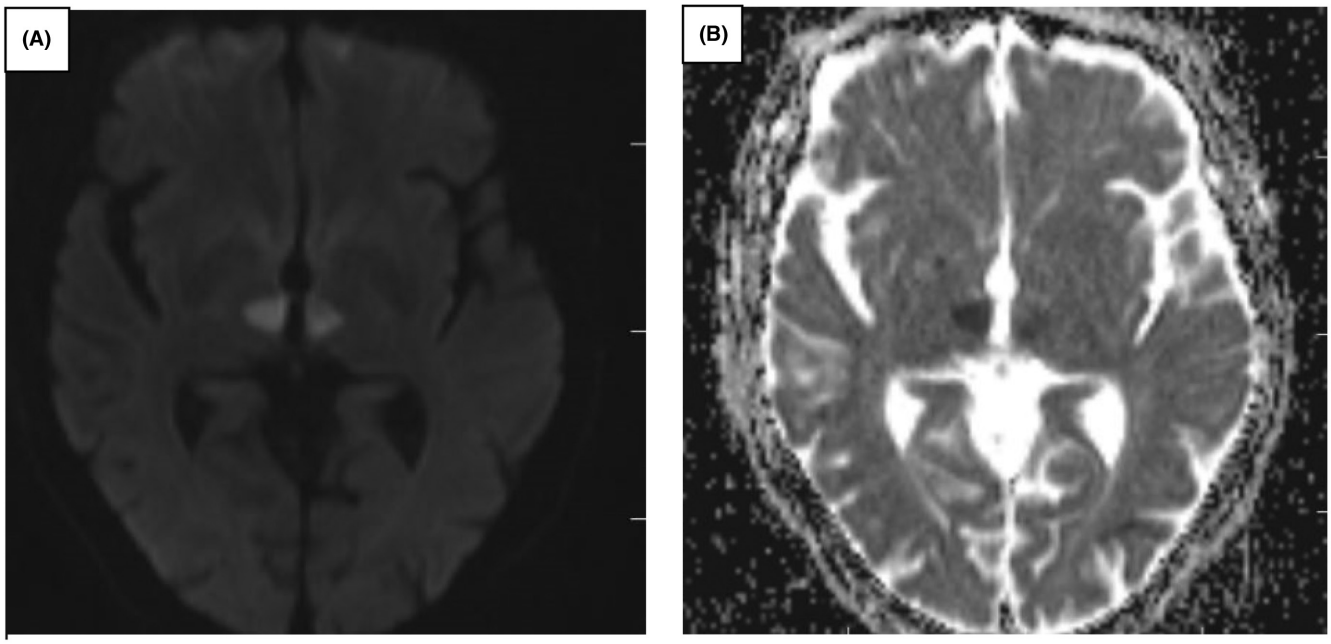

FIGURE 1 (A) Cerebral DWI showed hyperintensity in bilateral thalamus. (B) ADC sequence showed hypointensity at both thalamus; this is consistent with acute bilateral thalamic infarction.

major arterial supply of the paramedian region of the upper midbrain and thalamus, including the intralaminar nuclei and most of the dorsomedial nucleus. In certain patients, the artery of Percheron, which arises from the P1 segment of the posterior cerebral artery, supplies both sides of the paramedian territory of the thalamus.[6,11] In the present case, occlusion of the artery of Percheron was not observed in the cerebral angiography.

Because of the complex processes of the thalamus, infarction causes a variety of manifestations. These include conscious impairment (42%), vertical gaze palsy (65%), decreased memory (58%), and confusion (53%). If the corticospinal tract is involved, the patient may develop focal or lateralizing weakness. Because of its arterial connections with the brainstem, patients with bilateral thalamic infarcts may manifest cranial nerve palsies.[12,13] The patient presented to emergency room with agitation and somnolence for one day. He was not oriented to time, person, or place. His GCS was 9/15. Neurological examination did not show any focal or lateralizing deficits (indicating a preserved corticospinal system). There was no apparent vertical gaze impairment. He had no speech impairment or dysphagia. Pupils were isochoric and reactive to light.

MRI with diffusion sequence is the modality of choice for detecting acute thalamic infarct. Non-contrast CT is an alternative if MRI is not available. MRI/CT angiography may show occlusion of the posterior cerebral artery and the branching artery of the Percheron. In the present case, MRI diffusion of the brain showed bilateral hyperintensity of the thalamus, consistent with an acute infarct. Cerebral angiography did not show occlusion of posterior cerebral artery or its branching artery, the Percheron. The patient had poorly controlled hypertension. Hypertensive microangiopathy was considered the underlying etiology of this case.

Like other forms of stroke, the prognosis depends on the patient's clinical status, age, early application of thrombolysis, and the extent of the affected area.[14–16] In this case, despite the fact that the patient missed the thrombolytic treatment, his recovery was modest, and he achieved massive clinical improvement in two months of treatment.

4 | CONCLUSION

Bilateral thalamic infarct is a rare form of stroke. This is because the thalamus receives multiple arterial supplies from both the posterior cerebral artery and the posterior communicating arteries. Because of its neuronal complexity, a thalamic infarct may present with complex neurological symptoms. Neuroimaging is essential for diagnosis. Bilateral thalamic infarct should be considered in the differential diagnosis of patients who present with acute confusional state or agitation.

AUTHOR CONTRIBUTIONS

Mohamed Sheikh Hassan: Conceptualization; writing – original draft. **Nor Osman Sidow:** Data curation; methodology. **Abdiladhif Mohamed Ali:** Investigation; resources. **Mohamed Farah Osman:** Investigation;

resources. **Abdiwahid Ahmed Ibrahim:** Validation; visualization. **Said Abdirahman Ahmed:** Supervision; writing – review and editing.

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CONFLICT OF INTEREST STATEMENT

The authors declare no competing interests.

DATA AVAILABILITY STATEMENT

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

CONSENT

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the editor-in-chief of this journal on request.

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