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Acute bilateral paramedian thalamic infarct in the differential diagnosis of hyperactive delirium: A case report

Fettah Eren, Ayşegül Demir

University of Health Sciences Turkey, Konya Training and Research Hospital, Clinic of Neurology, Konya, Turkey

ORCID ID of the author(s)
FE: 0000-0001-6834-0827
AD: 0000-0002-5444-9837

Abstract

Acute bilateral paramedian thalamic infarct is a rare condition. Changes in consciousness, hypersomnia, mood disorders, cognitive problems and paralysis of vertical gaze may be observed in patients with acute bilateral paramedian thalamic infarct. Although it may have many causes, the most common is atherosclerotic small vessel disease. Some patients, who present with isolated neuro-psychiatric symptoms, may be misdiagnosed with a psychiatric illness. We herein present a patient presenting with the clinical features of hyperactive delirium who was diagnosed with acute bilateral paramedian ischemia. A 53-year-old female patient was admitted with complaints of changes in consciousness, agitation, and impairments in attention and memory, starting 2 days earlier and fluctuating during the day. Her history revealed hypertension and obesity. Levels of serum total cholesterol, high density lipoprotein (LDL) cholesterol and triglyceride were high, and the cardiac evaluation was within normal limits. Acute paramedian thalamic ischemia was detected through neuroimaging. Thalamic infarcts display different clinical features according to the localization of the lesions. Although many neurological and neuropsychological symptoms may be seen, thalamic infarcts may also present with psychiatric symptoms alone. Therefore, thalamic lesions should also be considered in acute or subacute behavioral changes in patients with no history of psychiatric disorders.

Keywords: Behavioral changes, Delirium, Paramedian thalamic ischemia

Corresponding Author
Fettah Eren
University of Health Sciences Turkey, Konya Training and Research Hospital, Clinic of Neurology, Konya, Turkey
E-mail: dreren42@hotmail.com

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The authors stated that the written consent was obtained from the patient presented with images in the study.

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Introduction

An oval-shaped mass of gray matter ideally situated at the core of the diencephalon, the thalamus conducts afferent impulses from the peripheral sensory receptors and transmits those from the cerebellum and basal ganglia to the motor cortex. There are many nuclei in the structure of the thalamus [1]. The medial and lateral geniculate nuclei play a role in visual and auditory functions, the pulvinar and lateral dorsomedial nuclei, in visual functions, the ventral posterolateral and posteromedial nuclei, in somatosensory functions, ventrolateral and ventroanterior nuclei, in motor functions, and medial dorsomedial nuclei, in autonomic and behavioral functions (2). In addition, the thalamus is involved in consciousness, sleep and paying attention. Vascular lesions of the thalamus can progress with different symptoms depending on the affected nucleus [1, 2].

The thalamus is fed by four major arteries, the tuberothalamic, inferolateral, paramedian and posterior choroidal arteries. Of these, bilateral infarct of the paramedian artery can cause confusion, hypersomnolence and changes in consciousness [3]. The etiology of bilateral thalamic infarct varies, and the main reason is small vascular disease developing because of atherosclerosis and cardiac embolism. In addition, thalamic infarcts are responsible for artery-to-artery embolisms due to large vessel atherosclerosis and constitute the etiology in migrainous stroke [2]. In the present report, a patient with acute bilateral paramedian thalamic ischemia with hyperactive delirium is presented.

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Case presentation

The patient signed the informed consent form for sharing her details and images for scientific purposes. A 53-year-old female patient presented with complaints of impaired recognition of surroundings, agitation and visual hallucination commencing suddenly around 3:00 am two days earlier. During this period, the patient could not recognize her spouse as well. However, the patient had partially stabilized when she woke up in the morning. The complaints recurred during the day with fluctuating symptoms. She especially claimed that she was with dead friends and relatives and saw such acquaintances in her daily life. The patient's sleep-wake cycle was impaired, and she suffered from insomnia. The patient was first admitted to the psychiatry outpatient clinic with these complaints, and later referred to the neurology outpatient clinic with a pre-diagnosis of hyperactive delirium due to the organic factors. Her history revealed that she had hypertension, for which she used ramipril 5 mg 1x1, and had been smoking 10 cigarettes per day over 20 years. The patient's body mass index (BMI) was 34.75 kg/m^2. BMI was calculated as weight in kilograms (kg) divided by height in meters (m) squared (kg/m^2). Neurological examination revealed that she was confused. The patient followed commands although place and time orientation were impaired. The patient's speech was rapid without dysarthria. In memory examination, her records were preserved while recall was impaired. Long-term memory was within normal limits with impaired attention and concentration. No cranial nerve deficits were found, and cerebellar tests were normal. She had no Babinski sign, and deep tendon reflexes were bilaterally normoactive.

The laboratory examinations were as follows: Leukocyte count: 7.44 103/mm^3 (4.99–12.68), hemoglobin: 12.8 g/dL (11.9–14.6), platelet count: 159x10^3/mm^3 (150-450), international correction rate (INR): 1.0 (0.8–1.2), glucose: 101 mg/dL (74–106), urea: 39 mg/dL (17–43), creatinine: 1.04 mg/dL (0.66–1.09), aspartate aminotransferase (AST): 18 U/L (0–35) and alanine aminotransferase (ALT): 12 U/L (0–35), serum total cholesterol: 287 mg/dL (0-200), triglyceride: 218 mg/dL (0-150), high-density lipoprotein (HDL): 46 mg/dL (40-60) and low-density lipoprotein (LDL): 180 mg/dL (70-130). The electrolytes, thyroid hormones, and blood gas values, along with serum copper and ceruloplasmin levels were normal. No intracerebral hemorrhage was detected on brain computed tomography (CT). In diffusion-weighted magnetic resonance imaging (MRI), while hyperintense areas were seen in the paramedian areas of the bilateral thalamus, a hypointensity was detected in the apparent diffusion coefficient (ADC) map (Figures 1A, 1B). No flow limitations suggesting venous occlusions were determined on cerebral MRI venography, and the electroencephalography (EEG) examination was normal (Figure 2A, 2B, 2C).

Considering current findings, an acute ischemic stroke was considered in the patient. The blood pressure (BP) was measured as 140/100 mmHg. On transthoracic and transesophageal echocardiography (echo), the left ventricular diameters and wall movements were normal, and there was no thrombus. The ejection fraction (EF) was 52% (>50%), and no arrhythmia was detected on 72-hour Holter electrocardiography (ECG). The carotid and vertebral artery examination with Doppler ultrasonography (USG) revealed an increased intimal-medial thickness in both Carotis Communis arteries (CCA). A 5x2 mm non-stenotic echogenic plaque was observed in the posterior wall of the right internal carotid artery (ICA). No stenosis was detected in vascular structures. Therefore, the metabolic, hematological, and genetic risk factors suggesting or leading to thrombocytosis were ruled out. The patient was administered olanzapine 10 mg/day and acetylsalicylic acid (ASA) 100 mg/day. On the 10th day of the treatment, orientation, and hallucination complaints, as well as insomnia began to improve. The patient was discharged with good health on the 17th day of treatment.

Figure 1: A) Hyperintensity in paramedian areas in bilateral thalamus on diffusion-weighted magnetic resonance imaging (MRI), B) Hypointensity in the same area on apparent diffusion coefficient (ADC) map

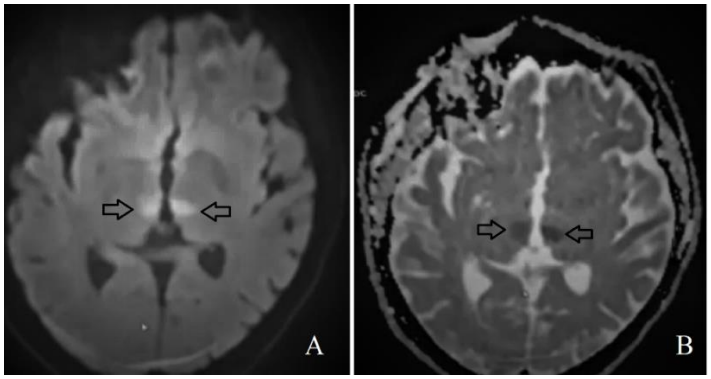
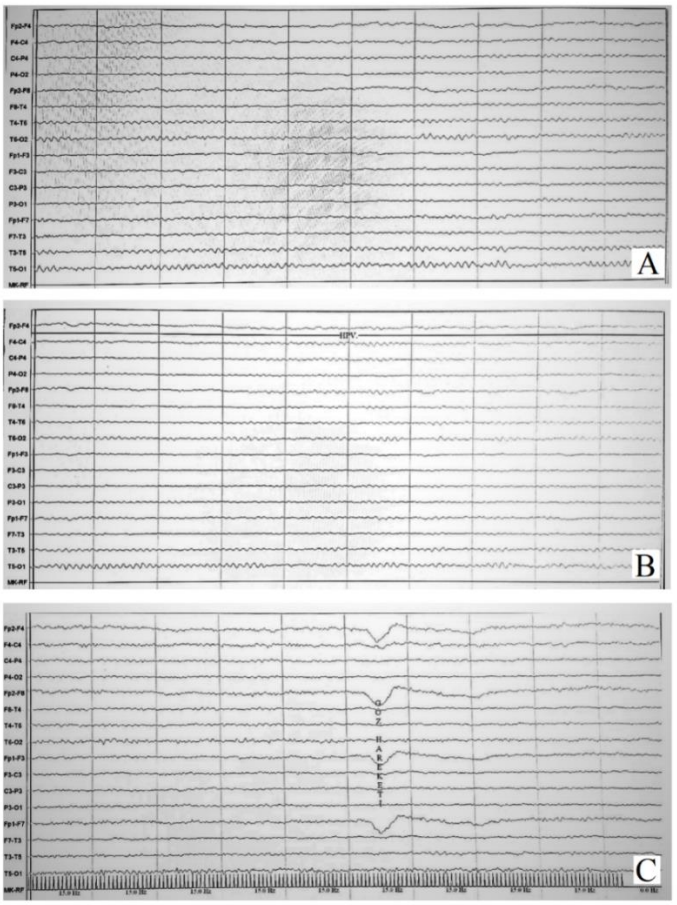


Figure 2: Electroencephalogram (EEG) A) Eyes opened, B) Hyperventilation, C) Intermittent photic stimulation (IPS): Electroencephalography findings consistent with normal bioelectrical activity



Discussion

Bilateral thalamic ischemia is an extremely rare entity and accounts for approximately 22-35% of all thalamic strokes. Atherosclerosis and cardioembolism are the most common etiological risk factors in bilateral thalamic strokes. Among other risk factors are atrial fibrillation, ventricular aneurysms, right-to-left shunts, left ventricular dysfunction and hypercoagulability [4]. Metabolic and toxic processes, infections and neoplasms can also mimic thalamic ischemia. Wernicke's encephalopathy occurs due to B1 (thiamine) deficiency, especially in patients with malabsorption. It presents with ataxia, changes in consciousness, antegrade amnesia and ocular dysfunction. The condition is especially seen among chronic alcohol users. On MRI, bilateral thalamic lesions, periaqueductal gray matter, tectum, mammillary bodies, and signal changes around the third ventricle are encountered [5]. Bilateral thalamic lesions may also be observed in the patients with central pontine myelinolysis, chronic kidney disease (CKD), liver disease, syndrome of inappropriate antidiuretic hormone secretion and diabetes mellitus (DM). Spastic hemiparesis, disorders of consciousness and pseudo-bulbar palsy are clinically observed with bilateral thalamic lesions [4, 5]. Diffusion restriction may be seen in the thalamus in Wilson's disease. Clinical symptoms, especially dysarthria, dystonia, ataxia, Parkinsonism, and psychiatric symptoms are often detected in young adults. Wilson's disease may also be accompanied by globus pallidus (GP), putamen and caudate nucleus lesions [6]. Creutzfeldt-Jakob disease is a neurodegenerative disease that can present with rapidly progressing dementia, ataxia, and myoclonus. In Creutzfeldt-Jakob disease, signal changes are observed in the putamen, caudate nucleus, and periaqueductal areas, in addition to the bilateral thalamic lesions [5, 6]. Bilateral thalamic glioma may also be encountered with similar MRI images and sometimes result in hydrocephalus. Patients with bilateral thalamic glioma are admitted to health facilities with personality changes or symptoms of dementia, they affect children or young adults, and their prognosis is poor since the lesions are deeply localized [5]. Bilateral thalamic lesions should be evaluated meticulously in differential diagnosis. The clinical findings in our patient had begun hyperacutely, and there was no alcohol use, history of malabsorption and chronic disease. The patient's current clinical, neurological, serological, and radiological findings prevented us from considering other differential diagnoses. In light of these findings, ischemic vascular disease was considered in our patient on the basis of atherosclerosis.

After strokes, delirium is quite frequent. However, stroke patients presenting with delirium are observed more rarely. The rate of delirium after a stroke was reported as 14.8%. Especially after acute ischemia affecting the left cortical and left subcortical areas, delirium develops at a higher rate. While left cortical ischemia was observed in 50% of stroke patients developing delirium, left subcortical ischemia was seen in 55.3%. No association was detected between brain stem and cerebellum involvement [7]. The thalamus, fusiform cortex, posterior parietal cortex, basal ganglion, and prefrontal cortex are the prominent brain regions related to emotions and memory. Emotional and memory disorders increase the risk of developing delirium by misinterpretation of the surrounding circumstances. In the literature, no difference was reported in terms of hypoactive or hyperactive delirium development and stroke localization [8]. However, acute bilateral paramedian thalamic ischemia was detected in our patient who was admitted with hyperactive delirium.

Although bilateral paramedian thalamic ischemia can be diagnosed through MRI, they are often diagnosed by means of clinical findings. Especially sensorial changes, paralysis of vertical gaze, and memory impairments are observed in patients with bilateral paramedian thalamic ischemia. Dysarthria, changes in consciousness and posterior cerebral circulation in motor paresis should suggest ischemia. In some patients, severe cognitive impairment, amnestic syndrome, and executive dysfunction may be seen even a few months after the stroke. Inappropriate behaviors, apathy, and mood swings may also occur in patients with delirium [9, 10]. Cases of delirium associated with bilateral paramedian ischemia are extremely rare [11]. Fluctuations of consciousness, hallucination and agitation suggested hyperactive delirium in our patient. With the combination of olanzapine and antiaggregant therapy, the patient's symptoms subsided within a short time.

Conclusion

Bilateral paramedian ischemia is a rare condition, and there are many mimicking disorders. Although cognitive and ocular problems are commonly observed in patients with bilateral paramedian ischemia, delirium is rare. Bilateral paramedian thalamic ischemia should be considered in the differential diagnoses in case of acute delirium, especially in those without any history of previous psychiatric disorders.

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