Case Report

Atypical Presentation with Good Outcome in a Bilateral Paramedian Thalamic Infarction

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Abstract

Background: Bilateral paramedian thalamic infarction is rare. We describe a patient without the previously reported cardinal features of vertical gaze paresis, decreased vigilance, mood changes, and memory problems that accompany bilateral paramedian thalamic infarction.

Case Study: A 41 year-old male with no previous medical problems presented with two short episodes of unconsciousness and transient ptosis. The patient returned to normal within several hours of presentation. The patient had no mood or personality changes after the event and bedside testing showed no evidence of cognitive changes or memory difficulties. A MRI scan of his brain showed an acute bilateral thalamic infarct.

Conclusions: This is only the second known described case with complete resolution of symptoms following bilateral paramedian thalamic infarction. This case also demonstrates that physicians should consider a bilateral thalamic infarct in the differential diagnosis when a patient presents with only transient unconsciousness and transient third nerve dysfunction.

Keywords: Bilateral thalamic, paramedian, infarct

Bilateral Paramedyan Talamik İnfarkt Olgusunda Atipik Prezantasyonla İyi Sonuç

Özet

Giriş: Bilateral paramedyan talamik infarkt enderdir. Biz daha önce bildirilmemiş bilateral paramedyan talamik infarktın eşlik ettiği vertical bakış paresi, uyanıklılıkta azalma, ruhsal değişiklikler ve bellek bozuklukları gibi kardinal özellikleri olan bir hasta tanımlamaktayız.


Sonuçlar: Bu olgu bilateral paramedyan talamik infarkt sonrası semptomların tamamen düzelme gösterdiği ikiinci bilinen olgudur. Bu olgu aynı zamanda sadece geçici bir biliş bozukluğu ve geçici üçüncü sinir bozukluğu geçiren bir hastanın ayırıcı tanısında bilateral talamik bir infarktın da düşündülmesi gerektiğini göstermektedir.

Anahtar kelimeler: Bilateral talamik, paramedian, infarkt
INTRODUCTION
Bilateral thalamic infarction is a rare entity. A study by Kumral et al. demonstrated that only 0.6% of stroke patients at their hospital had a bithalamic infarct over a seven year period.\(^{5}\) They found the most common thalamic infarct was in the territory of the paramedian artery.\(^{5}\) Despite its rarity, bithalamic infarction is a well-defined clinical syndrome. The clinical features that have been most commonly reported with a bilateral paramedian infarct are: vertical gaze paresis, extensive period of decreased vigilance (from drowsiness to coma), mood changes, poor motivation, and problems with memory.\(^{1,2,6}\)

Others have reported pseudobulbar palsy or loss of vergence control as the presenting feature of a bithalamic infarct.\(^{3,9}\)

Bilateral thalamic infarction most commonly occurs in patients who have an anomalous perfusion to the thalamus. The four arteries that supply blood to each thalamus are the paramedian, inferolateral, tuberothalamic, and posterior choroidal arteries. An anomalous paramedian artery is most commonly identified in people who suffer from a bilateral thalamic infarction. The paramedian artery arises from the P1 segment of the posterior cerebral artery.\(^{8}\) While each paramedian artery normally arises from each P1 segment, some people have both paramedian arteries anomalously arise from one common P1 trunk. This anatomic variant is known as the artery of Percheron.\(^{8}\)

Bilateral paramedian thalamic infarction is due to this rare anatomical variant in which a single lesion may affect both thalami. The lesion may be atherothrombotic or embolic in nature. The following case demonstrates a clinical variation of the usually described features and outcome of a bilateral paramedian thalamic infarct.

CASE PRESENTATION
A 41 year-old Mexican-American man, who had no previous medical problems, was watching television with his wife when he suddenly became unresponsive and slumped to the ground. His wife attempted to awaken the patient without success. She then reportedly attempted to perform cardiopulmonary resuscitation. A few minutes later the patient became alert, but was complaining of dizziness and faintness. At that time, his wife noted the patient had a right sided ptosis. The paramedics were called. On arrival, the paramedics reported the patient had a Glasgow Coma Scale (GCS) of 15. However, while transporting the patient to the hospital, thirty minutes after the initial event, the patient became unresponsive again. The paramedics described the patient having a GCS of 3 along with a heart rate of 120 beats per minute and a blood pressure of 134/83. The paramedics also observed that the patient had a dilated and unreactive left pupil. On arrival to the emergency department (ED), the patient was observed to have a left gaze preference and decreased withdrawal to pain on the left side. The ED physician intubated the patient after Versed sedation for airway protection. A neurologist saw the patient within one hour. The patient's vital signs were all within normal limits. He was unresponsive to voice while he was intubated and sedated with Versed. His left pupil was 3 mm and reactive to light while his right pupil was 1.5 mm and also reactive to light. The patient’s oculocephalic reflex was present in all directions. He had intact bilateral corneal reflexes and a normal gag reflex. There was no gaze preference at that time and his face appeared symmetric. The patient withdrew to painful stimuli in all four extremities. His deep tendon reflexes were 2+ bilaterally in the upper extremities and 3+ bilaterally in the lower extremities. The patient had a Babinski sign present bilaterally. One hour after Versed was discontinued, the patient was responsive to all simple commands and he demonstrated full motor strength in all extremities. The patient’s family reported he had no previous medical or surgical problems. He was not taking any medications and did not smoke or abuse drugs. In the ED, laboratory tests showed normal complete blood count, electrolytes, ammonia level, prothrombin time, partial thromboplastin time, and arterial blood gas. The patient also had a blood alcohol test and urine toxicology screen which were both negative. An EKG was performed which showed normal sinus rhythm and his cardiac enzymes were negative as well. Furthermore, the patient’s chest x-ray was also normal. A CT scan of the
patient’s brain was negative. A lumbar puncture was then performed. Cerebral spinal fluid analysis showed 4 white blood cells, 1 red blood cell, 29 mg/dl protein, 72 mg/dl glucose, VDRL negative, and gram stain and culture negative. Later, a CT angiogram of the arteries of the neck and circle of Willis was performed and showed no abnormalities. However, the paramedian artery was not visualized.

The patient was admitted to the intensive care unit (ICU) for monitoring. By the time he arrived in the ICU (3 hours from presentation), he was fully communicating and his neurologic exam was normal except his lower extremity reflexes remained brisk bilaterally and he continued to have bilateral Babinski signs. The following day, he was extubated without difficulty. He subsequently underwent an electroencephalogram (EEG) which was also normal. The patient continued to do extremely well while in the hospital without any episodes of alteration in consciousness. An MRI with diffusion weighted images was performed three days after the event. It showed a discrete increase in signal in the thalami bilaterally on both diffusion weighted images and fluid-attenuated inversion recovery (FLAIR) sequences. Also, a corresponding dark area on the apparent diffusion coefficient (ADC) map was present. These imaging findings were consistent with an acute bilateral ischemic infarct of the thalamus (Figure 1). The patient subsequently had an extensive evaluation which showed a normal sedimentation rate, thiamine level, cholesterol level, thyroid function, antinuclear antibody level, and syphilis studies. An echocardiogram was also performed which showed no abnormalities.

Figure 1: A) Diffusion weighted image showing restricted diffusion in both thalami B) ADC map showing corresponding dark areas in both thalami C) FLCIR image showing increased signal in both thalami
The patient continued to do well throughout his hospital course with a normal exam except for bilaterally brisk lower extremity deep tendon reflexes. During the hospitalization, the patient’s mood was pleasant and appropriate toward the staff as well as his family. Bedside cognitive testing revealed the patient had fluent speech and was able to follow all commands. His cognition appeared intact with a mini-mental status of 30/30. The patient had no difficulty in naming common and uncommon objects. Remote memory was tested and found to be intact (with assistance from his wife for verification). Furthermore, the patient’s fund of knowledge appeared normal after questioning him on current and past major political figures. He also successfully interpreted several proverbs. Lastly, his visual-spatial-constructional ability was determined to be normal after correctly drawing a clock figure.

The patient’s family felt there was no change in his personality or memory after the stroke. The patient was discharged home on aspirin therapy one week after his bilateral paramedian thalamic infarct in good condition. The patient was contacted six months after his hospital admission. The patient had no complaints and was working. The patient’s family reported no personality changes and no noticeable memory difficulties. Due to the fact that the patient was a member of a health maintenance (HMO) insurance plan, he was not able to be followed-up at our center for formal neuropsychological testing.

DISSCUSSION

This is, to our knowledge, only the second reported case of a complete recovery after a bilateral paramedian thalamic infarct. The first case was reported by Krolak et al. in 2000. Our case demonstrates that, in fact, recovery can be full. However, one limitation of our study was that we used extensive bedside testing for cognitive evaluation instead of formal neuropsychological testing. Our case contradicts Gentilini’s statement that a bilateral paramedian thalamic infarction has a prognosis that is “neither severe nor good.”

Our case presents several important clinical feature variations which differ from previously reported cases of bithalamic infarction. First, our patient did not have the vertical gaze paresis. Vertical gaze paresis has been noted in many prior case reports as being associated with the decreased level of consciousness in bilateral paramedian thalamic infarction. Instead, our case had a transient partial third nerve dysfunction in which there was a dilated pupil only. Second, our patient demonstrates that a bilateral paramedian infarct may present with loss of consciousness for a very limited time (minutes to few hours). This is much different than what previous case reports in the literature had reported. In fact, there have been many case reports citing that patients had difficulty in maintaining a normal conscious state for at least several days. The purported mechanism for the decreased level of consciousness is due to affected midline thalamic nuclei. These nuclei are a rostral extension of the reticular activating system. Third, our patient also demonstrates that a person with bilateral paramedian thalamic infarct may have a lack of pervasive retrograde or anterograde amnesia after the event and no subsequent behavior or personality changes. This differs from most prior case reports, such as the report by Winocur et al, who described anterograde amnesia with the syndrome of bilateral paramedian infarction. Finally, in contrast to the Kumral et al. case series in which 7 of the 8 cases with bilateral paramedian infarction had known causes (5 patients had longstanding diabetes or hypertension with presumed small artery disease while 2 patients had a potential cardiac source of embolism), our patient was relatively young, had no known risk factors, and had a negative workup. Furthermore, our patient did not have any less common conditions such as lupus, syphilis, cystercerosis, fungal infection, or thiamine deficiency. This is in contrast to the association made by Roitberg et al. who
reported these less common disorders may be an underlying cause of a bithalamic infarct. (7)

This case demonstrates a departure from many prior case reports with regard to the clinical manifestations of a bilateral thalamic paramedian infarct. As this entity can easily be missed by CT scan in the acute phase, it is important to consider this type of infarct in the differential diagnosis of patients with otherwise unexplained transient impairment of consciousness (even without the classical vertical gaze paresis). Physicians should be alert to the possibility that transient loss of consciousness coupled with a transient third nerve palsy may be a presentation of bilateral thalamic infarction.

REFERENCES

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Recieved by: Jun.21.2005
Revised by: Nov.21.2005
Accepted: Dec 12.2005