

Paroxysmal Sleep as a Presenting Symptom of Bilateral Paramedian Thalamic Infarctions

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Bilateral paramedian thalamic infarctions may result in hypersomnolence, supranuclear palsy, and an amnesic syndrome. We describe an 82-year-old man who presented with recurrent, transient episodes of unresponsiveness secondary to bilateral paramedian thalamic

infarctions. Electroencephalography was performed during one of these paroxysmal events and showed an abrupt transition to a stage 2 sleep pattern with spindle formation.

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REPORT OF A CASE

An 82-year-old man with a history of hypertension, benign prostatic hypertrophy, and gastroesophageal reflux disease presented with episodic, sudden unresponsiveness that lasted several hours. On the day of admission, the patient experienced 2 witnessed events characterized by sudden onset of unresponsiveness with no associated clinical manifestations. The patient remained motionless with flaccid tone, closed eyes, and no involuntary movements for approximately 2 hours. He was hemodynamically stable during the events. The patient then suddenly regained consciousness and maintained a normal state of alertness between events.

The patient had no history of cognitive decline, dementia, fever, rash, chest pain, shortness of breath, headache, apneic events, restless legs, seizures, recent trauma, recent medication change, or stroke. The patient had minimal snoring and took a daily nap for many years.

The patient's medications included furosemide, fosinopril sodium, and potassium supplementation. He did not take benzodiazepines or barbiturates. He consumed 1 or 2 alcoholic beverages per week and denied tobacco use.

Findings on neurologic examination showed amnesic syndrome, supranuclear palsy, and mild visual anosognosia. The amnesic syndrome was characterized by immediate short-term and long-term verbal memory impairment. The supranuclear palsy was most severe with down gaze. Findings on the neurologic examination were otherwise unremarkable.

Findings on the following laboratory studies were unremarkable: complete blood cell count, electrolytes, liver enzymes, coagulation parameters, troponin, ethanol level, vitamin B₁₂, folate, ammonia, thyroid, and arterial blood gas. The patient's erythrocyte sedimentation rate was mildly elevated at 41 mm/h.

Electroencephalography (EEG) performed on the day of admission showed a diffuse increase in beta frequency activity and intermittent, diffuse theta frequency activity. Follow-up EEG the next day captured the patient's third event. The patient was initially fully awake with an EEG background similar to the baseline of the admission study (Figure 1). The patient suddenly became unresponsive, and EEG revealed a pattern similar to stage 2 sleep with sleep spindles and delta frequency slowing (Figure 2). No rapid eye movements were present.

Computed tomography of the patient's head showed mild cerebral atrophy. Magnetic resonance imaging of the brain revealed abnormal T2-weighted and diffusion-weighted signals in the paramedian thalamic nuclei on both sides, consistent with subacute thalamic infarction (Figure 3). Magnetic resonance angiography revealed minimal irregularity of the distal left middle cerebral artery, a large left posterior communicating artery, and mild narrowing of the distal left carotid bulb.

A transesophageal echocardiogram showed moderate to severe atherosclerosis in the descending aorta with protuberant complex plaque. No other source of embolism was identified.

DISCUSSION

The dorsal paramedian thalamic nuclei receive afferent input from the frontal lobe, amygdala, inferior temporal cortex, centromedian thalamic nucleus, and zona incerta.¹ Efferent connections then project to the frontal lobe, lateral hypothalamus, and ventral anterior thalamic nucleus.¹

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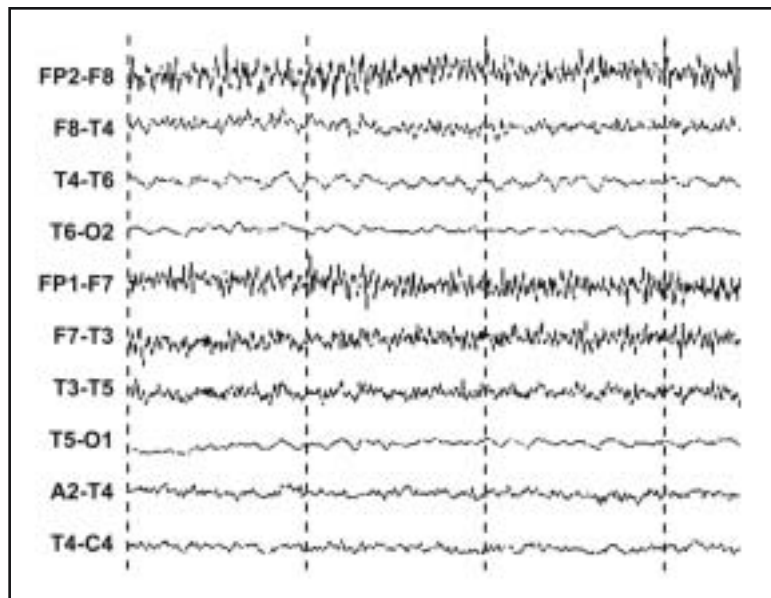


Figure 1. Electroencephalogram (EEG) obtained during the patient's waking state as patient responded appropriately to questions (sensitivity = 7 μ V/mm, high-frequency filter = 35 cycles/second, low-frequency filter = 0.53 cycles/second). The EEG is characterized by theta frequency slowing and muscle artifact.

These nuclei are involved with memory, emotion, and maintenance of consciousness. The vascular supply to the anterior portion of the medial thalamus is from the polar arteries. The polar arteries typically originate from the posterior communicating arteries. The remainder of the paramedian thalamic nuclei receive most of their vascular

supply from the paramedian thalamomesencephalic arteries, which originate from the posterior cerebral artery proximal to the ostium of the posterior communicating artery. The infarctions in our patient are in the territory of the distributed paramedian thalamomesencephalic arteries.

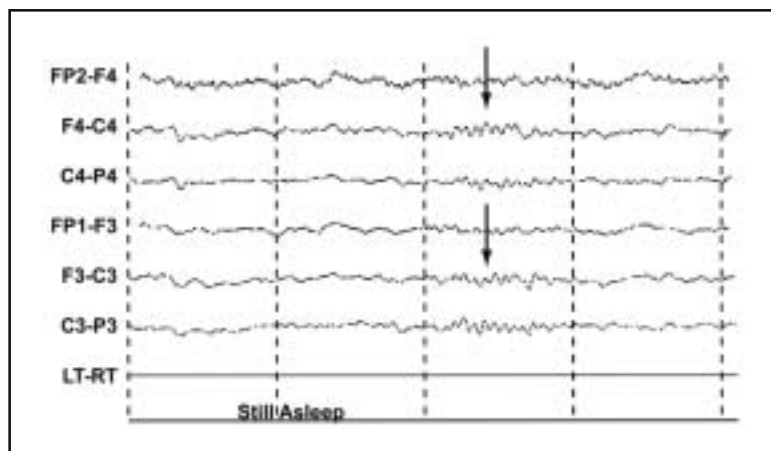


Figure 2. Electroencephalogram (EEG) obtained 9 minutes after Figure 1 EEG during the patient's episode of unresponsiveness. Note the 14-Hz sleep spindles prominent in the central channels (arrows) (sensitivity = 5 μ V/mm, high-frequency filter = 35 cycles/second, low-frequency filter = 0.53 cycles/second) and the absence of muscle artifact.

Thalamic infarctions result in markedly different clinical syndromes on the basis of the location of the infarction.^{2,3} Paramedian thalamic infarctions are characterized by supranuclear palsy, somnolence, neuropsychological deficits, and memory impairment. Bilateral paramedian thalamic infarctions may lead to more severe impairment of consciousness. There are also reports of bilateral paramedian thalamic infarctions that result in blepharospasm⁴ and disorders of smell, taste, and appetite.⁵

In 4 patients with paramedian diencephalic syndrome, initial symptoms were hypersomnolence and apathy followed by fluctuations in consciousness. However, discrete episodes of paroxysmal unresponsiveness were not observed.⁶ Otherwise, we found no previous reports of patients with bilateral paramedian thalamic infarctions who presented with episodic unresponsiveness and its EEG correlate.

Spindle coma is a condition characterized by a comatose state and 12- to 14-Hz sleep spindles on EEG. The spindles seen in spindle coma are similar in morphology to those seen in stage 2 sleep. However, the distribution of spindles in spindle coma often is more diffuse than that seen in our patient.

In our patient with bilateral paramedian thalamic infarctions, the presenting symptom was recurrent episodes of paroxysmal unresponsiveness, which had an EEG spindle pattern similar to that of stage 2 sleep. The magnetic resonance diffusion-weighted image was instrumental in identifying the infarctions that otherwise may have been overlooked by computed tomography. The paramedian thalamic nuclei play an important role in maintaining consciousness; therefore, it is not surprising that a lesion in this region might lead to impaired consciousness. However, recurrent paroxysmal unresponsiveness is an unusual presenting symptom for this lesion. A diagnosis of paramedian thalamic infarctions should be

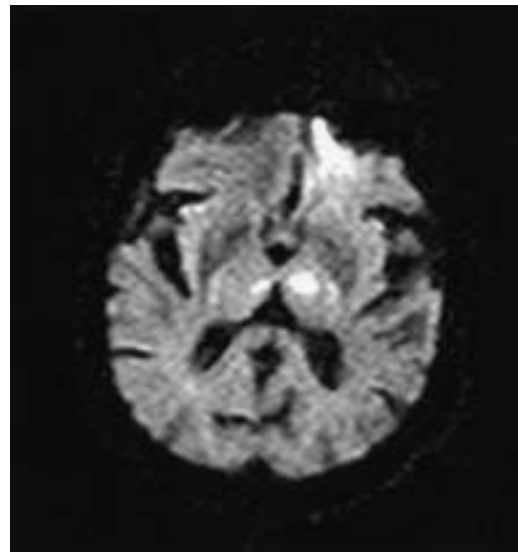


Figure 3. Axial diffusion-weighted magnetic resonance image of patient's head shows increased signal in the paramedian thalamic nuclei bilaterally.

considered in patients who present with recurrent episodes of unresponsiveness.

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