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Case Report

Oculomotor Palsy in a Subject with Thalamic Infarction: A Case Report

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Abstract

The thalamus is a highly complex nuclear structure comprising approximately 50–60 distinct nuclei, strategically positioned between the cerebral cortex and the midbrain. It plays a pivotal role in regulating sensory pathways, motor integration, consciousness, sleep, and cognitive functions. Through its vast interconnections, the thalamus serves as a key hub for neurocommunication, facilitating signal relay between cortical and subcortical areas.

This case report presents a rare instance of paramedian thalamic infarction selectively involving the dorsomedial nucleus of the thalamus, manifesting clinically as isolated oculomotor nerve palsy. Such presentations challenge conventional understanding of oculomotor dysfunction, which is typically attributed to midbrain lesions. The absence of brainstem involvement in this patient highlights the diagnostic importance of thalamic pathology in neuro-ophthalmologic syndromes. Our objective was to examine the visual and oculomotor manifestations associated with this atypical vascular event and assess clinical outcomes from an ophthalmologic standpoint. Key features analyzed include gaze abnormalities, ptosis, skew deviation, and pupillary asymmetry, all of which carry significant implications for quality of life and functional independence. The findings underscore the need for comprehensive neuro-ophthalmologic evaluation in cases of deep brain infarction, particularly those involving paramedian thalamic territory.

This report contributes to the growing recognition of thalamic strokes as important etiological factors in oculomotor dysfunction and provides insights into their clinical trajectory, prognostic markers, and rehabilitative strategies. Given their potential to mimic brainstem pathology, accurate early identification is essential for guiding neurorehabilitation and improving patient outcomes.

More Information

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Introduction

The thalamus is a major nuclear complex located in the diencephalon [1]. It serves as a relay centre for sensory, motor mechanisms, attention, and major neurocognitive processes. The thalamus is made up of a series of nuclei and is functionally divided into five components [2] and anatomically divided into three groups: relay nuclei, reticular nucleus, and intralaminar nucleus [3].

Stroke is an emerging cause of morbidity and mortality in developed countries and increasingly in low- and middle-income countries and contributing to the global health burden [4] thalamic infarctions account for 20% of all ischemic strokes and 14% of all lacunar infarctions [5,6].

Depending on the vascular regions and related nuclei, it causes a variety of neurologic symptoms. Thalamic infarction can cause a variety of visual and eye movement dysfunctions, either alone or in conjunction with infarction involving other regions, in addition to the traditional sensorimotor and amnestic syndromes [7-10].

There exists a lot of uncertainty around the underlying processes of oculomotor impairments, as well as possible future clinical courses and the most effective therapy strategy. Even in case series investigations of thalamic infarction with small samples, only a few have thoroughly addressed the neuro-ophthalmologic characteristics of the condition, despite the fact that numerous studies have been carried out to examine its clinical features [11,12].

The clinical diagnosis of thalamic infarction is challenging due to variations in anatomical location, volume, and lateralisation. This is a case report of paramedian thalamic artery infarction presenting with ophthalmoplegia.

Case report

A 60-year-old male presented with a history of right-

sided limb weakness accompanied by dysarthria. He had a known history of hypertension for six months. Magnetic resonance imaging (Figure 1) revealed an infarction in the left paramedian thalamus and the posterior limb of the internal capsule. MR angiography demonstrated an azygous anterior cerebral artery and a hypoplastic A1 segment.

Admission examination, the patient was drowsy but oriented. His blood pressure was elevated at 170/100 mmHg, and his Glasgow Coma Scale score was E4V4M5. General physical examination findings were within normal limits, and systemic evaluation was unremarkable. The electrocardiogram showed normal sinus rhythm.

Ocular Findings: Ocular examination revealed skew deviation with right-sided head tilt. Pupillary diameters measured 3 mm on the right and 4 mm on the left, with sluggish responses to light. The left eye showed restricted elevation, depression, and adduction, while the right eye was normal. Visual fields and posterior segment examination were within normal limits.

Neurological Findings: The patient exhibited central-type right facial nerve palsy without tongue deviation or limitation of tongue movement. No behavioral changes were noted. Limb power was rated at 2/5 on the right and 4/5 on the left. Babinski and Hoffmann reflexes were negative, and cerebellar function was intact.

A mild reduction in light touch sensation and proprioception was noted in the left upper and lower extremities. Brunnstrom recovery stages were graded as five for the left upper extremity, hand, and lower limb. However, manual muscle testing showed decreased strength in the left lower limb, with a grade of 3. Laboratory tests were within normal limits.

The patient was transferred to neurology care for further management.

Discussion

The thalamus functions as one of the most critical relay centres in the brain. Its nuclei are categorized into four groups based on arterial supply: tuberothalamic artery, thalamogeniculate artery, posterior choroidal artery, and paramedian artery.

Paramedian arteries, a branch of the posterior cerebral artery, originate at the junction of the basilar bifurcation and the junction of the posterior communicating artery and supply the thalamus. Rarely, a single paramedian artery can supply both sides. If a single paramedian artery supply is present and stroke occurs, such patients present with coma, abnormal eye movements, and behavioural abnormalities.

Oculomotor nerve palsy is a common symptom of a midbrain infarction, but there have been reports of a paramedian thalamic infarction inducing third-nerve palsy without a definite lesion on the brainstem, as in the present case [13].

Elevation deficit is likely due to the involvement of the frontofugal dorsothalamic bundle transversing the mediodorsal nucleus and internal medullary lamina of the thalamus on its way to the superior colliculus in the midbrain [12,14]. Along with thalamic lesion, coexisting lesion involving the rostral interstitial nucleus of the medial longitudinal fasciculus in the upper midbrain may also account for the vertical gaze palsy. In the absence of a definite midbrain lesion in this case, medial gaze palsy could be due to the involvement of oculomotor nuclei or fascicle, associated with thalamic lesion extension.

Skew deviation can occur with lesions anywhere in the vestibulo-ocular pathway, which is also believed to traverse the thalamus. As with several previous studies, skew deviation was found in this study in more than half of the patients with paramedian thalamic infarction [15,16].

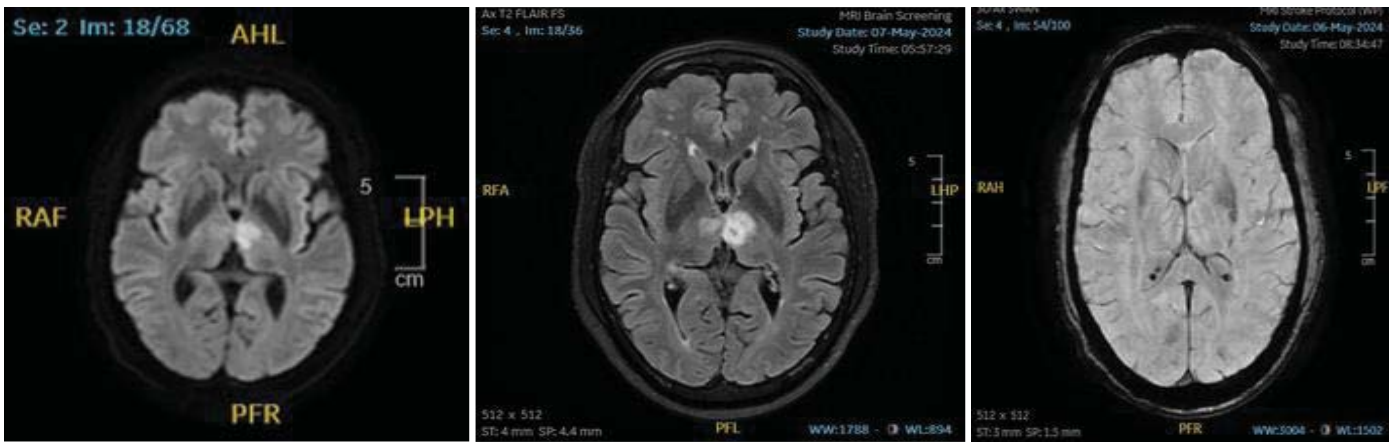

Figure 1: An MRI image showing an acute left paramedian thalamic infarction.

In addition to these features, ptosis [17-20], pseudo abducens palsy [21-23], Gaze-evoked nystagmus [24,25] have also been reported in previous studies.

A thalamic infarction not only causes motor impairments and sensory changes but also presents with oculomotor palsy, abnormal behavioural changes, and impaired executive function. Previous studies have reported sexually abnormal behaviour, sleep disturbance, amnesia, and the loss of self-activation in a paramedian thalamic infarction [11].

In the present case, ipsilateral oculomotor palsy appeared due to thalamic infarction, mimicking crossed hemiplegia observed in brainstem lesions. Although many oculomotor deficits resolve spontaneously within months, some may persist for years. Our patient received neurological care and showed gradual recovery over time—highlighting the importance of early assessment and targeted management in thalamic infarction.

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