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BILATERAL PARAMEDIAN THALAMIC INFARCTION PRESENTING WITH ATYPICAL PSEUDOBULBAR EFFECT: A DIAGNOSTIC CHALLENGE

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ABSTRACT

Bilateral thalamic infarction is a rare subtype of ischemic stroke often resulting from occlusion of artery of Percheron (AOP), diagnosed after excluding alternative arterial, venous & non-vascular etiologies. Altered sensorium and abnormalities in eye movements are common manifestations. We present the case of a 31-year-old woman attending our tertiary care hospital with presentation of sudden onset altered mentation associated with pseudobulbar affect and dysarthria. Her neuroimaging exhibited bi-thalamic infarcts. The clinical diagnosis of this entity remains challenging, particularly when classical symptoms are lacking.

Keywords: Artery of Percheron, Thalamic stroke, Pseudobulbar effect

INTRODUCTION

The thalami act as a relay station for all sensory inputs. They facilitate communication between subcortical regions and the neo-cortex.[1] Their blood supply is complex and arises from branches of the circle of Willis. Depending on the vascular territory involved, thalamic infarction can present as distinct vascular syndromes.[2] One of these is the paramedian thalamic infarction, which results from occlusion of the artery of Percheron (AOP)—a rare anatomical variant in which a single artery arising from the terminal basilar artery or proximal posterior cerebral artery supplies both paramedian thalami.[3]

The medial thalami function as components of the ascending reticular activating system which is the substrate for consciousness, and the upper midbrain is involved in control of eye movements. Bi-thalamic infarcts with or without the involvement of the midbrain produce alterations in consciousness, sleep, cognition, memory, behavior, ocular movements, and language; these varied manifestations are a source of difficulty in prompt recognition and diagnosis of such infarcts.[4]

We present here a case of bilateral paramedian thalamic infarction in a young woman manifesting with an unusual syndrome of somnolence, pseudobulbar symptoms, and dysarthria. This presentation explores the list of clinical symptoms associated with such infarction and may contribute to its prompt diagnosis for emergent treatment.

CASE PRESENTATION

A 31-year-old woman presented to the Emergency department (ED) of Sultan Qaboos Hospital, Muscat, Oman, with a one-day history of sudden onset altered behavior & change in speech associated with dysphagia. Notably, she underwent a spontaneous vaginal delivery three weeks prior. A day before presentation, she was observed to be sleepy and had to be woken up repeatedly to nurse her child also noted to laugh & cry inappropriately with any attempt of conversation with her. Her symptoms were initially attributed to the recent postpartum status, however later she developed slurred speech & difficulty in swallowing with severe episodes of choking on water. She was brought to the ED for further evaluation. On presentation the patient was sleepy & frequently yawning, though able to answer simple questions and was able to obey commands. She had heart rate of 67 b/min and a blood pressure of 131/77 mm Hg, with normal respiratory rate and oxygen saturation.

On neurological evaluation she had intact cognition, with withdrawn behavior, poverty of speech, and dull facial expression. She had a flat vocal tone with short one-word answers. She demonstrated inappropriate emotional responses, exhibiting both laughter and crying without clear provocation. Her extra-ocular movements were intact, with no ptosis. She was dysarthric with unclear syllables & had dysphagia for liquids. Tongue protrusion was weak without deviation. Cranial nerve examination revealed no further deficit except poor gag reflex. Further evaluation including tone, power, tendon reflexes & coordination was intact

in all extremities. Sensory examination an assessment showed no abnormality.

An urgent CT brain revealed hypo-densities in the paramedian regions of both thalami, being more prominent on the right side (Figure1a).There was no hemorrhage or hydrocephalus. A CT Angiogram of the head and neck (Figure 1b) performed subsequently revealed non-opacification of a short proximal segment (P1) of PCA, including the terminal part of the basilar artery and few bilateral thin arteries supplying the medial thalami. MRI of the brain (Figure 2) confirmed the presence of bilateral asymmetrical acute infarcts in paramedian thalami. Brainstem structures including the midbrain and pyramidal pathways along with cerebral venous sinuses were normal. These imaging findings were interpreted to suggest the presence of bilateral paramedian thalamic infarcts in the territory of the artery of Percheron.

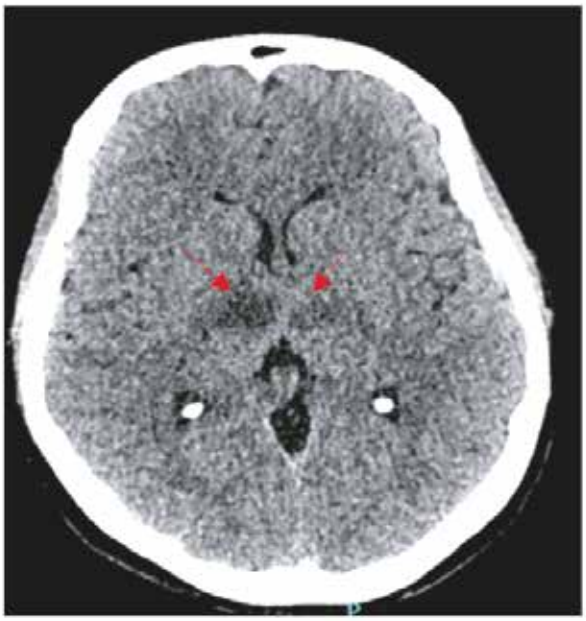




Fig 1. (1a) **CT brain without contrast** showed bilateral thalami hypo-densities.

(1b) **CT Angiogram** showed bilaterally slim origin and short proximal segment of P1 of PCA including the tip of basilar artery (AOP Origin).

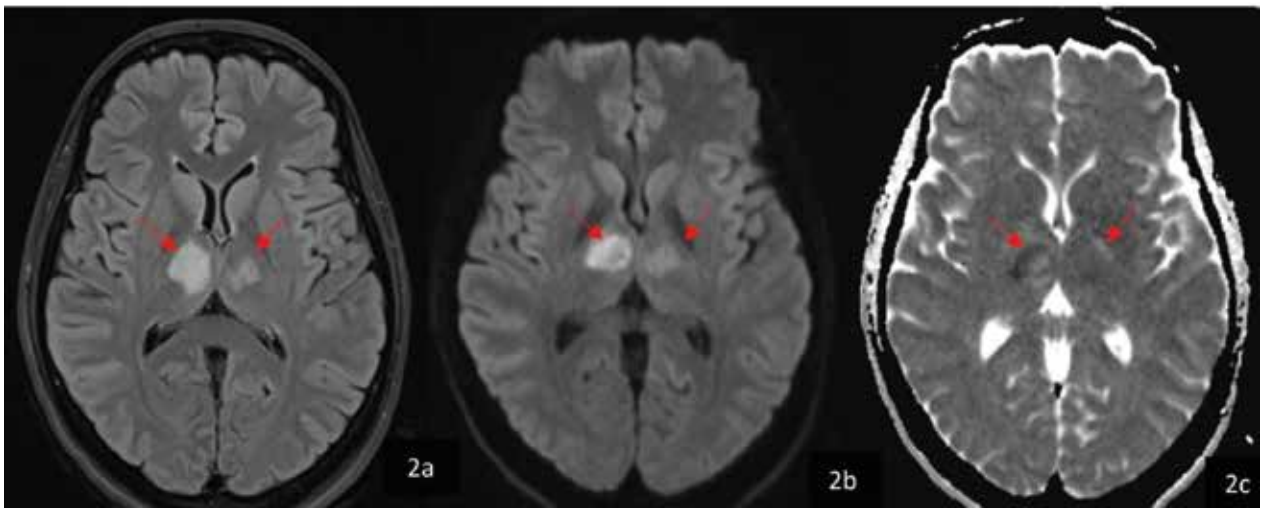


Fig 2: **MRI BRAIN** (2a) **T2/FLAIR** images showed bilateral asymmetrical thalami hyper intensities. (2b) Diffusion restriction seen in bilateral thalami on **Diffusion weighted images (DWI)** with corresponding a**pparent diffusion coefficient (ADC)** drop out signals (2c) indicating acute infarcts in bilateral medial thalami suggesting artery of Percheron territory infarcts.

Laboratory investigations including CBC, serum biochemistry, lipid profile, and B12 assay, were normal. ECG was normal. A comprehensive evaluation for the cause of ischemic stroke in a young patient was performed. Echocardiography with contrast bubble study and Holter study excluded any structural heart changes or arrhythmias. Autoimmune screening including ANA, ANCA, APLA, lupus anticoagulant & thrombophilia profile were negative. As were tests for HIV, varicella zoster, Syphilis, hepatitis, and Serum limbic/para-neoplastic antibodies. Normal CT chest, abdomen & pelvis excluded any underlying malignancy.

She received oral aspirin 100mg and clopidogrel 75mg (initially with loading doses of 300mg respectively) with 80mg atorvastatin. Nasogastric tube feeding was initiated to assist with feeding as well as medication. Her management continued along with rehabilitation in hospital. Over two weeks, she demonstrated improvement in her dysphagia and dysarthria. She received a short course of oral fluoxetine with which her emotional lability improved. She was discharged home on oral dual antiplatelet for three months along with atorvastatin without a nasogastric tube. At clinical follow-up after three months, she had fully recovered in all symptoms including behavior and dysarthria.

DISCUSSION

Thalamus is a highly intricate structure composed of numerous nuclei that facilitate the integration of sensory, motor, and behavioral signals from the cerebral cortex with various other neural pathways.[5] Overall, most authors recognize about four different patterns of AOP infarcts (summarized in table 1).[6]

Table 1: Patterns of ischemic damage due to an AOP stroke

Pattern	Frequency
Bilateral paramedian thalamus and the midbrain	(43%)
Bilateral paramedian thalamus without the midbrain	(38 %)
Bilateral paramedian thalamus, anterior thalamus, and midbrain	(14%)
Bilateral paramedian and anterior thalamus without midbrain	(5%)

The paramedian thalamic arteries can have anatomical variation of which one is known as the artery of Percheron (AOP). It appears as isolated terminal branch nascent in the PCA for the bilateral blood supply of paramedian thalamus and rostral midbrain The classic clinical triad of symptoms in the infarction of the AOP is characterized by consciousness impairment, vertical gaze palsy, and cognitive or behavioral disturbances.[7] Given the rarity and lack of comprehensive data on these specific manifestations, it's challenging to provide exact frequencies for each symptom. Bilateral thalamic infarction associated with pseudobulbar palsy is a rare but significant clinical condition, which typically arises from the occlusion of the artery of Percheron. Only a few cases of patients with bilateral paramedian thalamic infarction who develop acute pseudobulbar palsy have been reported in the literature.[7,8] Pseudobulbar palsy is characterized by dysarthria, dysphagia, facial and tongue weakness, with emotional lability. It is due to an upper motor lesion caused by bilateral disturbance of the corticobulbar tracts.[9] Clinical characteristics caused by bi-thalamic lesions & its intricate arterial supply is summarized in table 2 .[2,10-12]

Table 2: Clinical features associated with abnormalities in thalamic blood supply	
Thalamic blood supply	**Clinical features**
Tubero-thalamic Artery	•Fluctuating consciousness & Personality changes •Impaired learning, language and visuospatial impairment •Apathy & abulia •Emotional & limb apraxia
Inferolateral Artery	•Altered social skills, aggression & agitation •Sensory loss •Hemi-ataxia •Dejerine-Roussy •Auditory Impairment
Paramedian Artery of Percheron	•Decreased arousal (coma if bilateral) •Impaired memory & confabulation •Aphasia and spatial deficits
Posterior Choroidal Artery	•Visual field loss (hemianopia, quadrantanopsia) •Variable sensory loss, weakness, aphasia, memory impairment and delayed complex hyperkinetic motor syndrome

Long-term prognosis post thalamic infarction is commonly observed as being better compared with other cortical and subcortical strokes.[13] This usually implies with rapid recovery from a motor deficit, however cognitive and psychiatric manifestations may persist after tubero-thalamic or paramedian artery stroke due to hypothalamic dysfunction.[14]

CONCLUSION

Thalamic stroke syndromes are indeed known for their atypical combination of symptoms and signs, often contributing to misdiagnosis. The present case illustrates a brain with such infarcts manifesting with the unusual combination of emotional lability and dysarthria. An explanation for these symptoms could be bilateral pyramidal pathway dysfunction resulting in a partial pseudobulbar state. Awareness of this syndrome in the context of bilateral paramedian thalamic infarction could contribute to its prompt diagnosis and possible early intervention.

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Tahir Siddiqui; Case management, literature search, manuscript writing
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Muhammad Imran; Literature search, manuscript revision
Abdullah Al-Asmi; Case management, manuscript revision
Abdullah Al-Asmi; Case management, manuscript writing
Arunodaya R Gujjar; Case management, manuscript revision
All the authors have approved the final version of the article and agree to be accountable for all aspects of the work.

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