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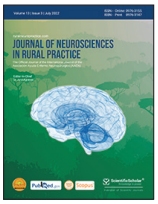

Case Report

Claude syndrome unveiled in a case of thalamic-midbrain infarction

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ABSTRACT

Claude syndrome is a rare mesencephalic syndrome characterized by third-nerve palsy and contralateral cerebellar symptoms. We present the case of a 52-year-old man with sudden diplopia, right-sided ptosis, drowsiness, and gait instability. Examination revealed anisocoria with a mydriatic right pupil that was non-reactive to light stimuli, left-sided dysdiadochokinesia and dysmetria, and restricted eye movements with impaired intorsion of the right eye, suggesting possible fourth cranial nerve palsy. Magnetic resonance imaging revealed an acute stroke in the right thalamus and ipsilateral midbrain. This is among the first cases in South America to describe the clinical-radiological correlation of a thalamic-midbrain stroke with Claude syndrome.

Keywords: Claude syndrome, Cranial nerve palsy, Posterior circulation brain infarction

INTRODUCTION

Posterior circulation infarcts account for 20–30% of stroke patients admitted to the hospital. The complex blood supply and numerous anatomical structures of the brainstem lead to various syndromes depending on the location of the infarction. Claude's syndrome is a brainstem stroke syndrome initially described in 1912, characterized by ipsilateral third cranial nerve palsy, hemiataxia, and contralateral cerebellar symptoms. This is a rare case of thalamic-midbrain infarction in a 52-year-old male that presents as Claude's syndrome with possible involvement of the fourth cranial nerve and some symptoms possibly attributed to the thalamic component of the infarction.[1]

CASE REPORT

On August 10, 2022, a 52-year-old man presented to the emergency room of Cayetano Heredia National Hospital, Lima, with a sudden onset of diplopia, right-sided ptosis, drowsiness, dizziness, and gait instability. The symptoms had appeared 12 h before his arrival to the hospital. Before the onset of these symptoms, the patient was in good health, except for a history of severe traumatic brain injury (TBI) in 2003, treated with frontal craniotomy, with no apparent sequelae. He had no prior history of stroke, diabetes, hypertension, or dyslipidemia. On examination, the patient was found to be drowsy with a Glasgow coma scale score of 14/15 (eye-opening: 3, verbal response: 5, and motor response: 6) and a normal blood pressure of 135/75 mmHg. His visual acuity, visual fields, language, and limb strength were normal. He exhibited right-sided ptosis and anisocoria, with a dilated right pupil that was non-reactive to both direct and indirect light stimuli. Left eye upgaze was limited, while right eye upgaze, downgaze, and adduction were restricted. Intorsion of the right eye during attempted adduction and depression of the pupil was limited, suggesting possible fourth cranial nerve palsy [Figure 1] [Video]. The patient exhibited an unsteady gait with a widened base of support and a negative Romberg test. He also had dysdiadochokinesia and dysmetria in the left upper and lower limbs. No extrapyramidal signs were observed. The remainder of the physical examination was unremarkable. The patient exhibited sinus bradycardia with pulse rates ranging from 50 to 62 beats/min, left ventricular hypertrophy, and ST elevation in the inferior leads, as observed in multiple consecutive electrocardiogram readings over the following days. An emergency echocardiogram revealed no cardiac dysfunction. No new symptoms appeared during hospitalization, except for a slight decrease in sensitivity in the left hemibody. Laboratory tests included a complete blood count, glucose level, rapid severe acute respiratory syndrome coronavirus 2 test, VDRL, HBsAg, and rapid human immunodeficiency virus test, all of which were negative or normal. His lipid profile revealed an LDL cholesterol level

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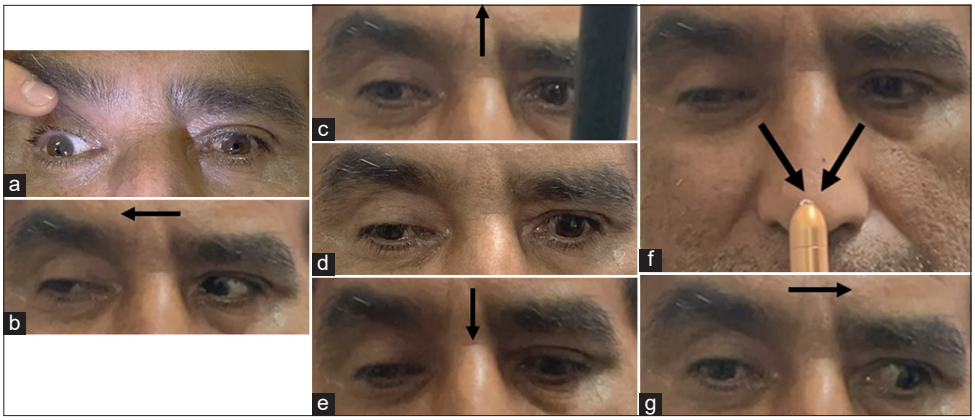
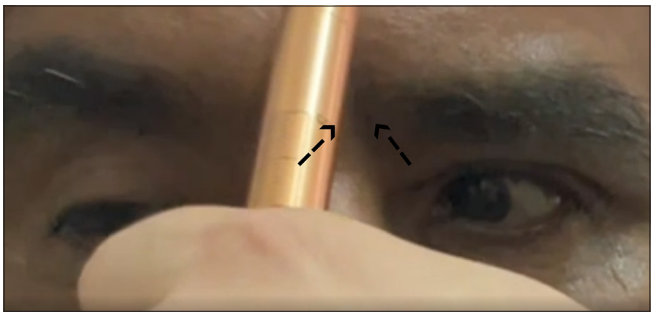


Figure 1: (a) Neurological eye examination exhibits clinical anisocoria with a dilated right pupil that was non-reactive to light stimuli. (b) Lateral gaze of the right eye was normal (arrow). (c) Bilateral upward gaze was restricted (arrow). (d) Partial right-sided ptosis was evident in the primary gaze position. (e) Right-eye downward gaze was restricted (arrow). (f) Right-eye intorsion during attempted adduction and depression was limited (arrow). (g) Right-eye adduction was restricted (arrow).

of 146.6 mg/dL. Magnetic resonance imaging (MRI) with T2, apparent diffusion coefficient, and diffusion-weighted sequences [Figure 2] revealed acute synchronous lesions in the anteromedial region of the right thalamus (16 × 11 mm) and in the paramedian region of the ipsilateral midbrain (8 × 7 mm), extending rostrally and to the ipsilateral superior cerebellar peduncle. In addition, chronic lesions were observed in both frontal lobes, attributed to his prior history of severe TBI. The patient was observed for 5 days before being discharged on aspirin 100 mg and atorvastatin 80 mg, to be taken every 24 h. He attended a follow-up appointment 2 weeks later but subsequently lost contact.

DISCUSSION

This case describes a thalamic-midbrain infarction with symptoms consistent with Claude's syndrome: Partial ipsilateral oculomotor paralysis, ataxia, and contralateral cerebellar syndrome with possible involvement of the IV cranial nerve. Ataxia, dysdiadochokinesia, and dysmetria in contralateral limbs are explained by damage to the corticopontocerebellar fibers of the dentatothalamic pathway, which is responsible for coordination between the cerebellar hemisphere contralateral to the lesion and the ipsilateral motor cortex. This pathway enters the brainstem through the superior cerebellar peduncle, crosses the midline, and ascends toward the contralateral ventrolateral and anteroventral thalamic nuclei before synapsing in the motor cortex. If no apparent lesion is evident in the lateral thalamic region, the damage is presumed to be located at the midbrain level, specifically in the inferomedial region of the red nucleus, posterior to the decussation of the dentatothalamic pathway.[2] Most patients with Claude's syndrome exhibit partial or incomplete involvement of the III cranial nerve, with preservation of the parasympathetic pupillary fibers, as their fibers run widely separated through the midbrain before converging at the interpeduncular fossa to form the oculomotor nerve.[2]



Video: Ocular motility test. Ptosis associated with difficulty in adduction and downward gaze of the right eye, corresponding to ipsilateral third cranial nerve paralysis. Limited bilateral upward gaze. Inward gaze of the right eye appears restricted, suggesting possible fourth cranial nerve palsy.

However, the extent of the midbrain infarction reported in this case results in nearly total involvement of the right III nerve fibers, associated with a rare involvement of the IV cranial nerve nucleus at the level of the inferior colliculi. IV cranial nerve involvement in patients with Claude's syndrome was initially reported in 1922 by Claude and Levy-Valensi and has been rarely reported in similar cases since.[3] In addition, in this case, the patient exhibited bilateral paralysis of upward gaze despite having a cerebral infarction on the right side. This can be explained because the motor fibers of the somatic lateral column of the III cranial nerve nucleus innervate the contralateral superior rectus muscle and the eyelid elevators of both sides.[1]

A series of cases has found that most midbrain infarctions are unilateral, paramedian, and tend to be associated with other lesions in the posterior circulation, especially in the thalamus.[4]

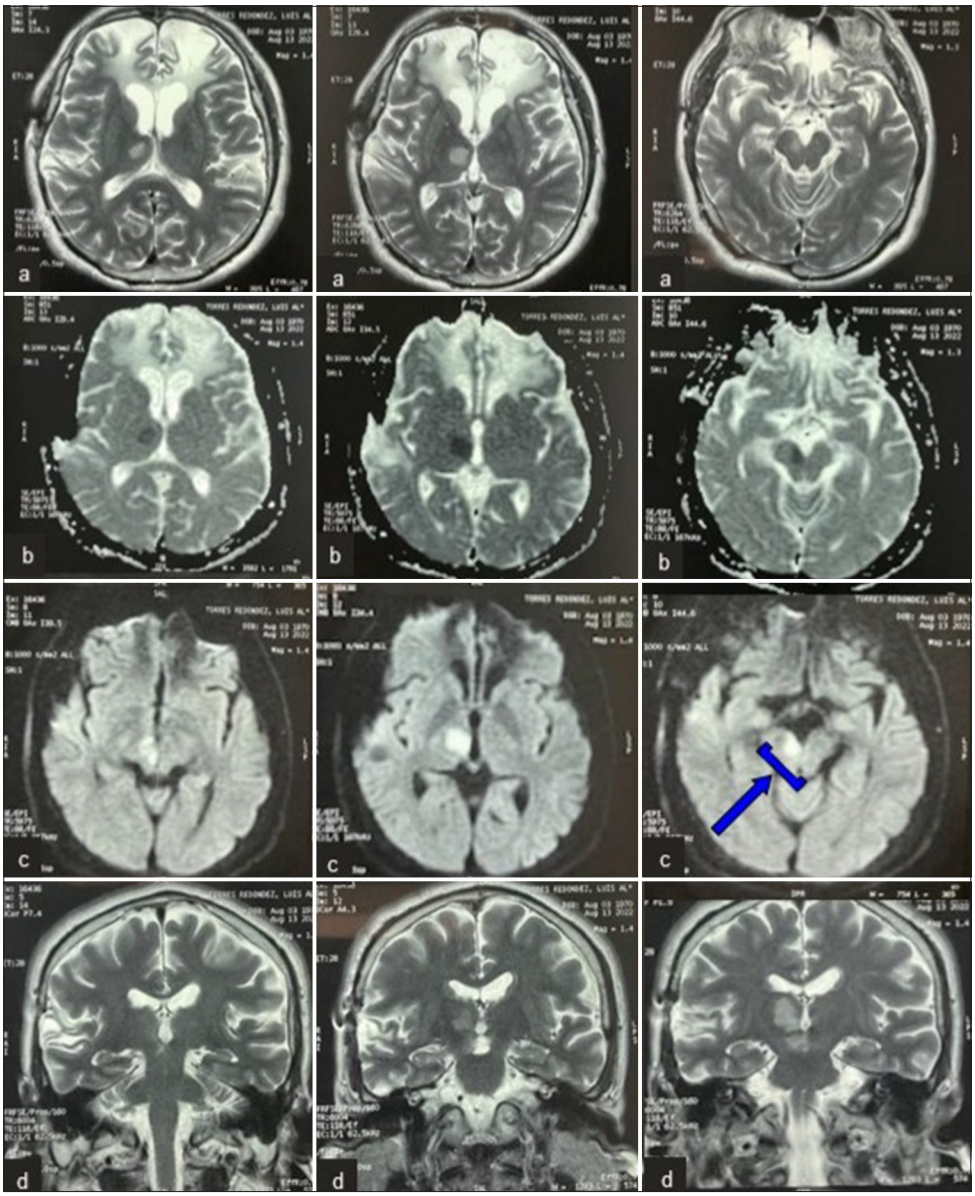


Figure 2: (a) Brain magnetic resonance imaging (MRI). The axial T2-weighted (T2W) image shows hyperintensity in the anteromedial region of the right thalamus, measuring 16 × 11 mm, and in the paramedian region of the ipsilateral midbrain, measuring 8 × 7 mm. (b) Low values on the apparent diffusion coefficient (ADC) map and (c) hyperintensity on the diffusion-weighted image (DWI) (blue arrow and bracket) suggests an acute ischemic lesion in both the thalamus and midbrain. This pattern differs from the lesions in the frontal lobes, which are hyperintense on T2W and hypointense on DWI and exhibit high values on the ADC map, corresponding to a chronic lesion due to prior head trauma. (d) The coronal T2-weighted image helps visualize the extent of the hyperintense lesions in the right thalamus and midbrain, consistent with an acute ischemic infarction.

Furthermore, unilateral thalamic-midbrain infarctions tend to cause drowsiness, while bilateral lesions are associated with coma from the onset of symptoms.[4] In this case report, the clinical presentation and thalamic extension of the infarction correlate with the pattern of paramedian thalamic infarction, which is characterized by an initial fluctuation in the level of consciousness, and as the condition evolves, changes in mood and behavior become evident.[1] The paramedian arteries are branches of the P1 segment of the posterior cerebral arteries and are responsible for the dual blood supply to the paramedian thalamic and midbrain regions.[5] These arteries also tend to supply the anterior thalamic regions due to the

high frequency of anatomical variations or inconsistencies in the polar arteries, which typically supply this area. This can lead to an anterior extension of paramedian thalamic infarctions, as reported in this case.[1,5]

The apparent association between sinus bradycardia and paramedian thalamic-midbrain infarction has been previously reported. Although sinus bradycardia is not a common finding in Claude's syndrome, it can occur in cases where the midbrain or thalamic infarction affects the autonomic pathways that regulate heart rate.[6] This case report describes one of the rare variants of Claude's syndrome with involvement of the IV cranial nerve, sinus bradycardia, and concomitant thalamic infarction. Therefore, this study is one of the first reports of Claude's syndrome with IV cranial nerve involvement in South America.

CONCLUSION

This patient presented contralateral cerebellar symptoms associated with damage to the ipsilateral third cranial nerve, possible involvement of the fourth cranial nerve, and other symptoms such as sinus bradycardia and drowsiness, possibly attributed to thalamic dysfunction. The most important lesson from this case is the need to correlate neuroanatomical and radiological MRI findings to understand the clinical presentation and symptoms of the infarction in question. In addition, the study highlights the importance of further investigating the role of the thalamus in regulating cardiac function.

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