

Case Report

Bilateral Claude Syndrome — A Rare Paramedian Midbrain Hemorrhagic Stroke Presentation

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Abstract

Background : Brainstem strokes can have protean ophthalmological manifestations including pupillary and extraocular muscle paresis. Incomplete paresis of oculomotor function is more common than complete paresis owing to the selective involvement of subnuclei or fascicles of various extraocular muscles. Here, we report a case of paramedian midbrain hemorrhage who presented with symmetric bilateral ptosis, complete ophthalmoplegia with ataxia. Also the anatomical as well as radiological correlation of this clinical presentation in relation to the structure of oculomotor nerve nuclei has been discussed.

Key words : Claude syndrome, Midbrain Hemorrhage, Bilateral Ptosis, 3rd Cranial Nerve, Ataxia.

The midbrain is the most cephalic portion of the brainstem, extending from the pontomesencephalic junction to join the diencephalon. Its anterior limits are given by the crus cerebri and the interpeduncular fossa, while the posterior limit is characterized by the presence of the superior and inferior colliculi¹. Claude syndrome is a rare midbrain stroke syndrome characterized by ipsilateral third cranial nerve palsy and contralateral hemiataxia². Although the red nucleus in midbrain has often been suggested as the site responsible for Claude's syndrome, a lesion of the superior cerebellar peduncle just below and medial to the red nucleus could be responsible for this syndrome. This case demonstrates neurological heterogeneity of midbrain infarction³. The most common causes of Claude's syndrome are cerebrovascular disease and malignancy⁴. As with peripheral lesions, the prognosis for these patients is generally good, and ocular motor dysfunction tends to improve or resolve within several months⁵. Isolated third nerve palsy secondary to a hemorrhagic stroke is rare⁶. A case of bilateral trochlear nerve palsy following a midbrain brainstem hematoma has been described⁷. We report the case of bilateral third nerve palsy & Ataxia secondary to midbrain hemorrhage.

CASE REPORT

A 63-year-old man, presented with 3 days history of

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Editor's Comment :

- Bilateral Claude syndrome is a rare manifestation of midbrain stroke.
- Prompt neuroimaging and focused neurological assessment are essential for accurate diagnosis.
- Recognition of this entity aids in better understanding and management of brainstem vascular disorders.

sudden onset giddiness, drooping of both eyelids such that patient was not able to open his both upper eyelids without any diurnal variation or fatigability & had associated limb ataxia. Neurological examination revealed bilateral, symmetrical ptosis with normal size pupils, symmetrical & reacting to light, but with both eyes abducted ie, deviated laterally. Extraocular movements showed palsy of both eyes in adduction, elevation, depression & extorsion with bidirectional horizontal nystagmus evident on attempting adduction of either eye. Deep tendon reflexes were preserved in all limbs with bilateral plantar response were flexor & gait was wide based & ataxic. NCCT brain revealed a hyperdensity in paramedian midbrain with intra-ventricular extension in 4th ventricle (Fig 1). NCCT head was repeated after 48 hours, which showed resolving hematoma without any signs of hematoma expansion or hydrocephalus. The patient was managed conservatively with antihypertensive and other supportive therapy. patient was managed by antihypertensive medication and supportive measures.

DISCUSSION

The nuclei of the 3rd cranial nerve originate at the level of the superior colliculus. The oculomotor fascicles sweep ventrally and laterally through the oculomotor complex, pass medial to the red nucleus and exit the brainstem medial to the cerebral peduncles⁶.

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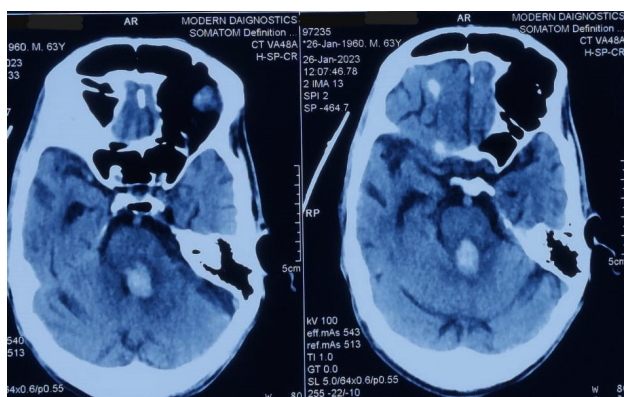


Fig 1 — Showing midbrain hemorrhage and IVE

To the best of our knowledge, no case of midbrain hemorrhagic stroke leading to bilateral third nerve palsy has been described in the literature. In our patient, the bilateral third nerve palsy can be explained by nuclear involvement and ataxia by involvement of superior cerebellar peduncle.

The sudden onset, preceded by high blood pressure (190/110mmHg), is suggestive of hemorrhagic stroke. The absence of an associated impairment of the descending and ascending tracts suggests a posterior bilateral midbrain lesion. This constellation of signs give a picture like bilateral claud's syndrome.

CONCLUSION

Bilateral complete third nerve palsy & ataxia secondary to a midbrain hematoma is an exceptional situation that must be considered because of the morbidity and therapeutic implications of the underlying disease. Stroke should be included in the differential diagnosis of an isolated oculomotor paralysis even when it is bilateral especially

when the onset is sudden or when the onset is unknown. One has to be vigilant in brainstem bleed because of high likelihood of deterioration as a result of complications, so that an excellent functional outcome can be achieved by adopting appropriate strategies timely.

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