Case Report

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ATYPICAL PRESENTATION OF PONTINE STROKE - A CASE REPORT

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ABSTRACT Brainstem strokes are traditionally characterized by the presence of "crossed neurological deficits," where cranial nerve involvement occurs ipsilateral to the lesion while motor or sensory deficits occur contralaterally in the body. Pontine infarctions, however, may present with variable and sometimes atypical clinical manifestations depending on the precise neuroanatomical structures involved. We report the case of an 80-year-old male who presented with acute onset swaying while walking and slurring of speech for one day. Neurological examination revealed left facial palsy and left hemiparesis, MRI brain suggestive of right pontine stroke. This report emphasizes that not all pontine infarcts present with classical brainstem syndromes, and careful clinical evaluation supported by neuroimaging is essential for accurate diagnosis. We also review the literature regarding atypical presentations of pontine infarction.

KEYWORDS : Brainstem stroke, Pontine stroke, Hemiparesis

INTRODUCTION

The brainstem plays a critical role in integrating motor, sensory, and autonomic pathways and houses multiple cranial nerve nuclei. Due to this complex anatomical organization, lesions in the brainstem often produce distinctive neurological syndromes that allow accurate clinical localization. One of the classical teaching principles in neurology is that brainstem lesions produce "crossed neurological deficits," in which ipsilateral cranial nerve dysfunction occurs in association with contralateral motor or sensory deficits in the limbs (1).Pontine strokes represent an important subset of posterior circulation strokes and account for a significant proportion of ischemic brainstem infarctions (2). The pons contains the corticospinal tracts, corticobulbar tracts, cranial nerve nuclei, ascending sensory pathways, and connections with the cerebellum. Because of this anatomical complexity, infarctions involving the pons can produce a wide range of neurological manifestations.

Several classical pontine syndromes have been described, such as Millard–Gubler syndrome, Foville syndrome, and Marie–Foix syndrome, which typically exhibit the classical pattern of crossed deficits. However, clinical observations and neuroimaging studies have demonstrated that pontine infarctions may also present with atypical or incomplete syndromes depending on the size and location of the lesion (3).

We present a case of acute right hemipontine infarction presenting without classical crossed hemiparesis, highlighting the variability in clinical presentation of pontine strokes.

CASE REPORT

An 80-year-old male presented to the emergency department with complaints of swaying while walking and slurring of speech for one day. The symptoms had an acute onset and were noticed by family members when the patient developed difficulty maintaining balance while walking and his speech became slurred. There was no history of headache, vomiting, seizures, loss of consciousness, visual disturbances, limb numbness, or facial sensory symptoms.The patient had no known comorbidities such as hypertension, diabetes mellitus, ischemic heart disease, or dyslipidemia. He was not on any long-term medications. There was no history of smoking, alcohol consumption, or other substance use.

Neurological examination revealed dysarthric speech. Cranial nerve examination demonstrated deviation of the mouth toward the right side, suggestive of a left facial nerve palsy. Motor system examination revealed left pronator drift, indicating subtle weakness in the left upper limb. Muscle strength in the left lower limb was mildly reduced (Medical Research Council grade 4/5). Muscle tone was slightly decreased on the left side compared with the right. Deep tendon reflexes were brisk in the left upper and lower limbs, while they were normal on the right side. The plantar response was extensor on the left side and flexor on the right.Given the acute onset focal neurological deficits, a provisional diagnosis of acute ischemic stroke involving the posterior circulation was considered.Magnetic resonance imaging (MRI) of the brain with diffusion-weighted imaging was performed. The imaging revealed an acute infarct in the right hemipons, confirming the diagnosis of right pontine infarction.The patient was managed by initiation of antiplatelet therapy, statin therapy, and supportive management.

The most notable aspect of this case was the absence of classical crossed neurological deficits, which are commonly associated with brainstem lesions.

Fig 1: Image of the patient showing weakness of left Upper limb with left facial palsy

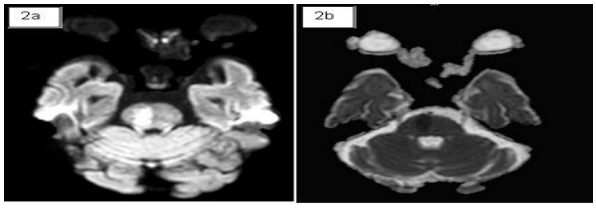

Fig 2: MRI brain - Intra axial images showing restriction diffusion with ADC reversal in the right Hemipons

DISCUSSION

Pontine infarctions account for approximately 7–10% of all ischemic strokes and are among the most frequently encountered brainstem infarctions . The clinical manifestations of pontine stroke are highly variable because of the diverse anatomical structures present in this region.Traditionally, brainstem lesions are associated with crossed neurological syndromes, in which cranial nerve deficits occur ipsilateral to the lesion while long tract signs occur contralaterally . Classical pontine syndromes include Millard–Gubler syndrome, characterized by ipsilateral facial nerve palsy and abducens nerve palsy with contralateral hemiparesis, resulting from involvement of the corticospinal tract and facial nerve nucleus .

However, several studies have demonstrated that pontine infarctions may present with atypical clinical features. Kim and colleagues reported cases of pontine infarction presenting as pure motor hemiparesis, which mimicked supratentorial lacunar stroke syndromes . In these cases, selective involvement of the corticospinal tract in the basis pontis resulted in contralateral motor weakness without cranial nerve involvement.Another well-recognized variant is ataxic hemiparesis, which occurs due to involvement of pontocerebellar fibers in addition to corticospinal tracts (4). These patients present with a combination of limb weakness and cerebellar ataxia on the same side. Pontine infarctions may also present with isolated cranial nerve deficits, such as isolated facial palsy. Several reports have described dorsal pontine infarctions presenting with facial weakness mimicking facial palsy of peripheral cause (5).In addition, cases of uncrossed facial weakness associated with pontine infarction have been reported, suggesting that selective involvement of corticobulbar fibers may produce atypical facial motor deficits (6).Other unusual presentations described in the literature include isolated hemiataxia, sensory disturbances, or dysarthria, depending on which neural pathways are affected (7,8).These observations indicate that the clinical manifestations of pontine infarction depend primarily on the topographic localization of the lesion rather than a uniform pattern of crossed deficits.

In the present case, the infarct likely involved the ventral portion of the right pons, affecting descending corticospinal and corticobulbar tracts while sparing the cranial nerve nuclei. This pattern explains the contralateral limb weakness and ipsilateral facial deviation observed in the patient.

Another possible explanation is that small paramedian pontine infarcts, usually resulting from occlusion of perforating branches of the basilar artery, may selectively affect certain tracts while sparing adjacent structures (9). Such selective involvement results in incomplete or atypical brainstem syndromes.From a clinical standpoint, recognizing such atypical presentations is extremely important. Posterior circulation strokes are often underdiagnosed or misdiagnosed, particularly when classical neurological signs are absent (10). Early neuroimaging with diffusion-weighted MRI plays a crucial role in identifying small brainstem infarcts that may not be visible on CT scans.

CONCLUSION

The present case of right hemipontine infarction presenting with contralateral facial palsy and contralateral limb weakness demonstrates that not all brainstem infarctions follow classical patterns.

While classical teaching emphasizes crossed deficits in brainstem lesions, clinicians must recognize that pontine infarcts can produce uncrossed or incomplete neurological syndromes.

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