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A Rare Case of Right Eight-and-a-Half Syndrome with Left-Sided Ataxia Secondary to Subacute Brainstem Infarction in a Diabetic-Hypertensive Male

Case Report

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Abstract

Eight-and-a-half syndrome is a rare neuro-ophthalmological condition caused by a focal brainstem lesion involving the paramedian pontine reticular formation (PPRF), medial longitudinal fasciculus (MLF), and ipsilateral facial nerve fascicle[1]. We report a 60-year-old diabetic-hypertensive male with right eight-and-a-half syndrome and contralateral cerebellar ataxia due to a subacute infarct in the right hemipons extending to the pontomedullary junction. The case illustrates a classic example of clinico-radiological correlation and emphasizes the role of early neuroimaging.

Introduction

Eight-and-a-half syndrome, first described by Eggenberger in 1998, combines one-and-a-half syndrome with an ipsilateral lower motor neuron (LMN) facial palsy [1]. The one-and-a-half syndrome results from damage to the PPRF or abducens nucleus along with the MLF on the same side, leading to horizontal gaze palsy and internuclear ophthalmoplegia [1-4] When the lesion also involves the adjacent fascicle of the facial nerve, an LMN facial palsy completes the clinical picture [1]. The most common causes are brainstem infarcts, demyelinating lesions, and tumors [2-5]. Due to the compact neuroanatomy of the pons, such clinical findings can assist with precise localization even before imaging [5].

Case Presentation

A 60-year-old right-handed male with a 10-year history of type 2 diabetes mellitus and systemic hypertension presented on day four of symptom onset with acute giddiness, facial deviation to the left, and unsteadiness while walking. He had no headache, vomiting, seizures, trauma, fever, or focal limb weakness. He had discontinued antihypertensives and oral hypoglycemics. No history of smoking, alcohol, or familial neurological disorders was noted.

On general examination, the patient had a blood pressure of 150/90 mmHg and a pulse rate of 82 beats per minute, which was regular. His fasting blood sugar was 230 mg/dL and postprandial

blood sugar was 389 mg/dL. There was no pallor, icterus, cyanosis, lymphadenopathy, or pedal edema.

Neurologically, he was alert and oriented to time, place, and person. Cranial nerve examination revealed conjugate right horizontal gaze palsy with preserved vertical gaze, right internuclear ophthalmoplegia characterized by failure of adduction of the right eye and abducting nystagmus in the left eye, and right LMN facial palsy, evidenced by flattened nasolabial fold and inability to puff the right cheek or close the right eye tightly. Other cranial nerves were intact.

Motor system examination showed normal tone and power (MRC grade 5/5) in all limbs. Deep tendon reflexes were brisk bilaterally, and plantar responses were flexor. Sensory examination revealed no abnormalities.

Cerebellar signs were evident on the left side, with limb ataxia, dysdiadochokinesia, and past-pointing. The patient also displayed truncal ataxia and walked with a wide-based gait, tending to veer to the left.

Systemic examination of the cardiovascular, respiratory, and abdominal systems was unremarkable.

Neuroimaging

MRI of the brain and orbits (dated 11/06/2025) revealed a subacute infarct involving both the dorsal and ventral aspects of the right hemipons, extending inferiorly to the pontomedullary junction. There was no susceptibility artifacts noted on SWI sequences, and no abnormal enhancement was seen on post-contrast T1 images. Mild cerebral atrophy was observed, with a global cortical atrophy (GCA) score of 2.

MR TOF angiography showed no significant vascular narrowing, aneurysms, or arteriovenous malformations in the intracranial circulation. The bilateral posterior communicating arteries were hypoplastic.

Carotid Doppler ultrasonography revealed 48% stenosis in the right and 56% in the left external carotid artery (ECA). The plaques were classified as Type II atherosclerotic, without ulceration or flow-limiting internal carotid artery obstruction.

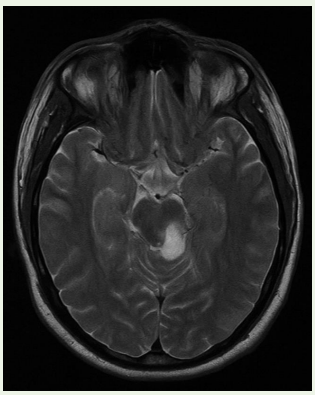

Figure 1: Impression: CEMRI study of the Brain with orbit reveals subacute infarct involving dorsal and ventral right hemipons extending to the pontomedullary junction. There is cerebral atrophic change (GCA score 2).

Table 1: Baseline Investigations

Parameter	Value	Normal Range
Hemoglobin (Hb)	12 g/dL	13.0–17.0 g/dL
Total Leukocyte Count	7909 /mm³	4000–11000 /mm³
Platelet Count	2.3 lakh /mm³	1.5–4.0 lakh /mm³
AST	40 U/L	< 40 U/L
ALT	36 U/L	< 45 U/L
Serum Creatinine	1.1 mg/dL	0.7–1.2 mg/dL
TSH	4.8 µIU/mL	0.5–5.0 µIU/mL
HbA1c	10.8%	< 5.7%
LDL	130 mg/dL	< 100 mg/dL
Triglycerides	230 mg/dL	< 150 mg/dL
Total Cholesterol	180 mg/dL	< 200 mg/dL
Prothrombin Time / INR	16.4 sec / 1.4	11–13.5 sec / < 1.2
ESR	30 mm/hr	< 20 mm/hr
Carotid Doppler	48% and 56% ECA stenosis	Not applicable

Final Diagnosis

The patient was diagnosed with eight-and-a-half syndrome secondary to a subacute infarct in the right hemipons. The left-sided cerebellar ataxia was attributed to involvement of pontocerebellar fibers. The infarct was likely due to underlying uncontrolled diabetes mellitus, hypertension, and dyslipidemia.

Treatment and Clinical Course

The patient was admitted to the neurology ward and managed conservatively. He was started on Aspirin 150 mg daily and Atorvastatin 40 mg daily. Strict glycemic control was initiated with insulin therapy. Antihypertensive therapy was optimized using Amlodipine and Telmisartan. A structured physiotherapy program was implemented, focusing on balance training, coordination, and facial muscle rehabilitation.

During the hospital stay, the patient remained hemodynamically stable and did not develop any new neurological deficits. He showed gradual improvement in gait and balance. He was discharged with instructions for continued physiotherapy, optimization of vascular risk factors, and regular follow-up.

Discussion

Eight-and-a-half syndrome is a rare brainstem disorder resulting from a lesion affecting the horizontal gaze center, internuclear connections, and ipsilateral facial nerve fascicle [1,4,5]. The lesion usually localizes to the dorsal pontine tegmentum, as observed in this case [5]. The presence of contralateral ataxia likely signifies additional involvement of the right inferior cerebellar peduncle or cerebellothalamic fibers at the pontomedullary level [5].

While Cho et al. reported eight-and-a-half syndrome due to pontine infarction in a hypertensive patient without ataxia[6], Salazar et al. described brainstem infarcts with overlapping oculomotor and cerebellar findings, but not a classic eight-and-a-half presentation [3].

Our case presents a comprehensive example of this rare syndrome with cerebellar involvement, implying a broader lesion. Elevated ESR and HbA1c along with extracranial stenosis indicate a small vessel

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vasculopathy, consistent with TOAST type 3 etiology [4,5]. The patient was managed per standard ischemic stroke protocols using antiplatelets, statins, glycemic control, and physiotherapy. Although the thrombolytic window had lapsed, early neurorehabilitation improved function and prevented complications.

Conclusion

This case emphasizes the value of clinical localization in diagnosing complex brainstem syndromes. The co-occurrence of eight-and-a-half syndrome with contralateral cerebellar ataxia pointed to an extensive lesion in the right pontine and pontocerebellar regions. Prompt MRI aided in confirming the clinical suspicion. Effective control of modifiable vascular risk factors and timely rehabilitation significantly contributed to the patient's recovery. A high index of suspicion in similar presentations can lead to accurate localization and better outcomes.

Declarations

Informed consent was obtained from the patient. There is no conflict of interest to declare. No funding was received for this study. Ethics approval was not required for a single case report.

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