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Case Report

Nine Syndrome: Unravelling A Rare Mystery of Neurology

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Abstract

Nine syndrome is a rare neurological condition characterized by ipsilateral conjugate horizontal gaze palsy (the "one"), ipsilateral internuclear ophthalmoplegia (INO) (the "half"), and ipsilateral lower motor neuron (LMN) facial cranial nerve palsy (the "seven"), which comprises "Eight-and-a-half" in total. Here, the only possible horizontal movement is contralateral abduction. If "Eight-and-a-half" is associated with contralateral hemiplegia, it is called "Nine Syndrome". Reported causes include pontine infarction, demyelination, neoplasm, infection and vascular malformation.

Here, we report the case of a 48-year-old diabetic man with no prior hospitalization who presented to the medicine department with the sudden onset of diplopia, vertigo, and facial asymmetry for 9 hours. On detailed neurological examination, he was found to have left-sided lower motor neuron facial palsy, impaired horizontal ocular movement involving the left eye, horizontal nystagmus on the abducting right eye, and right-sided extensor plantar response. The clinical constellation was consistent with Nine syndrome. An MRI performed later established an ischemic lesion in the left pons. Thrombolysis was not performed because the patient presented outside the recommended therapeutic time window. The patient showed early neurological improvement and was discharged with advice to have follow-up.

***Keywords**: Brainstem Stroke, Pontine Infarction, Neurology, Case Report.*

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Introduction

Nine syndrome is a rare but highly localizing brainstem syndrome caused by a lesion in the dorsal pontine tegmentum. It usually occurs due to ischemic stroke, but may also result from demyelinating diseases, tumors, infections, or any vascular malformations affecting the pons. The syndrome represents a combination of three neurological deficits occurring in close anatomical proximity within the pons: One-and-a-half syndrome, Ipsilateral lower motor neuron facial palsy and contralateral weakness. Commonly involved structures include the paramedian pontine reticular formation (PPRF) and the abducens nucleus, which are responsible for horizontal gaze, and the corticospinal tract. The medial longitudinal fasciculus (MLF) plays a great role in coordinating conjugate eye movements, and the facial nerve nucleus or fascicle (CN VII), which controls the ipsilateral facial muscles. Damage to these structures produces a characteristic pattern of ocular motor, facial features and contralateral weakness which allows precise neuroanatomical localization of a pontine lesion[1-3].

Patients typically present with horizontal gaze palsy toward the side of the lesion, impaired adduction of the ipsilateral eye during contralateral gaze, ipsilateral lower-motor-neuron facial weakness and contralateral hemiplegia. This constellation of findings produces the characteristic clinical pattern known as nine syndrome. Recognition of this syndrome is therefore an important diagnostic clue to underlying pontine pathology. Although uncommon, early identification is crucial, particularly in cases caused by ischemic stroke, as timely intervention such as thrombolytic therapy may significantly improve neurological outcomes[4]

Case Presentation

A 48-year-old male patient presented to the medicine department of DMCH in January 2026 with diplopia, vertigo, facial asymmetry, and weakness in one side of the body, which began suddenly, nine hours prior to arrival. The patient denied any history of fever, weight loss, prior head injury, or difficulty in vision, difficulty in hearing, prior headache, or convulsion. His medical history included diabetes mellitus

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and hypertension. His medications were comprised of Linagliptin 5 mg and Amlodipine 5mg irregularly. He was an ex-smoker and had reported no known drug allergies. On admission, the patient had elevated blood pressure (140/90 mmHg) and a regular pulse rate of 82 beats per minute. The neurological examination revealed left-sided LMN facial nerve palsy with a flattened nasolabial fold with deviation of the mouth on the right side, inability to wrinkle the forehead, and incomplete eyelid closure on the left side (Figure 1). Ocular movement testing revealed impaired adduction of the left eye and horizontal nystagmus of the right eye on attempt to see the right side (Figure 2); restricted adduction of the right eye and impaired abduction of the left eye on leftward gaze (Figure 3); Power was 3/5 in right upper and lower limb & all the reflexes on the right side were exaggerated and extensor plantar on the right side. We have calculated the National Institutes of Health Stroke Scale (NIHSS) for the patient, which scored 12.

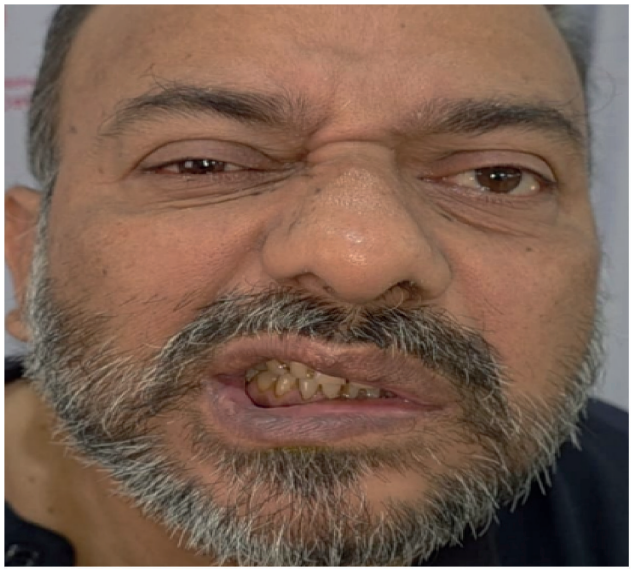

Figure 1: Left-sided Lower Motor Facial Nerve Palsy
(Photos are taken with permission of patient)

Figure 2: Impaired adduction of the left eye and horizontal nystagmus of the right eye on right gaze

(Photos are taken with the permission of patient)

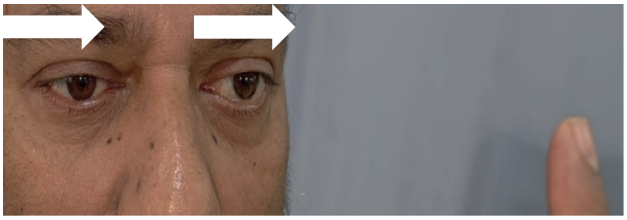

Figure 3 : *Restricted adduction of the right eye and impaired abduction of the left eye in left gaze*
(Photos are taken with the permission of patient)

On investigation, his HbA1c was 7.4% and fasting lipid profile showed slightly elevated LDL. Apart from the baselines, we performed a CT scan of the brain, which excluded hemorrhage. Diffusion-weighted MRI (DWI) of the brain revealed an acute left paramedian pontine tegmental infarct that also involved the left facial colliculus and corticospinal tract, which explained the patient's pattern of involvement (Figure 4). ECG was normal. Echo-cardiography showed non-obstructive hypertrophic cardiomyopathy (Asymmetric Septal Hypertrophy) (Figure 5). Carotid Doppler and other tests incuding CTA of brain were advised for the patient.

The patient was managed with Aspirin and Atorvastatin with strict control of diabetes. He was subsequently discharged with advice to undergo further follow-up at the Stroke Clinic, DMCH, for a proper rehabilitation program, including physiotherapy and occupational therapy, and with hypertension and diabetic control. On subsequent follow-up, the patient's facial weakness, gaze palsy, and walking difficulty were showing signs of recovery.

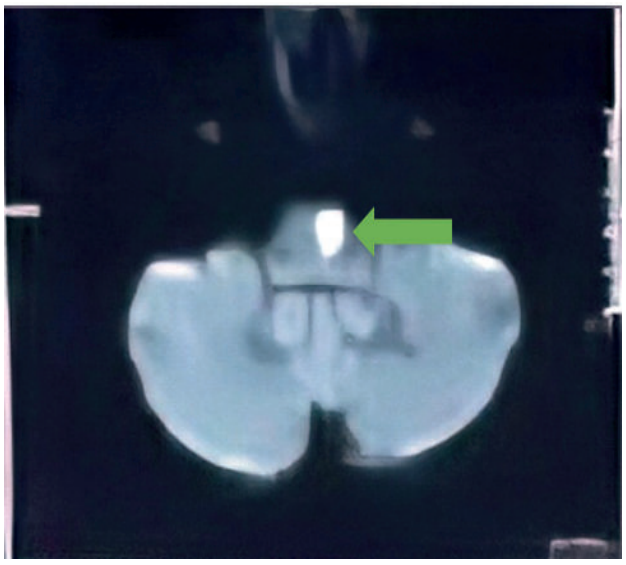

Figure 4: *DW MRI showing left pontine infarct (Green arrow)*

(Photos are taken with the permission of patient)

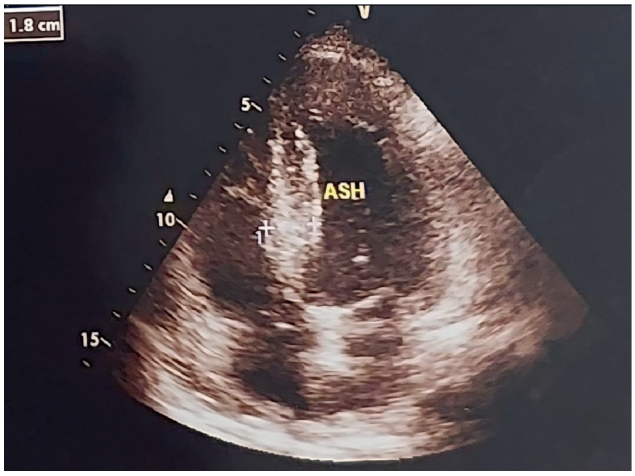

Figure 5: *Echocardiography showing Asymmetric Septal Hypertrophy*

Discussion

Nine syndrome is an uncommon neuro-ophthalmological entity resulting from a focal lesion involving the dorsal tegmentum of the caudal pons. The condition most frequently arises secondary to ischemic stroke but may also occur in association with demyelinating disorders or intrinsic brainstem neoplasms. Pathophysiologically, the syndrome reflects simultaneous involvement of several very closely situated pontine structures, including the abducens nucleus, the paramedian pontine reticular formation (PPRF), the medial longitudinal fasciculus (MLF), the intrapontine fascicle of the facial nerve and pyramidal tract. This characteristic combination of structural involvement accounts for the distinctive clinical manifestations observed in affected patients[5-6]. The dorsal pontine tegmentum contains several closely related structures responsible for horizontal eye movements and facial motor function. The abducens nucleus or the paramedian pontine reticular formation (PPRF) regulates horizontal gaze; therefore, a lesion in this region produces ipsilateral horizontal gaze palsy. The medial longitudinal fasciculus (MLF) is a highly myelinated tract that coordinates conjugate eye movements by linking the abducens nucleus with the contralateral oculomotor nucleus. Injury to the MLF results in internuclear ophthalmoplegia, characterized by impaired adduction of the ipsilateral eye with nystagmus of the contralateral abducting eye. In addition, the facial nerve (cranial nerve VII) fascicle courses around the abducens nucleus; involvement of this structure leads to ipsilateral lower motor neuron facial palsy affecting both the upper and lower facial muscles & corticospinal tract involvement leads to contralateral hemiplegia[7-8]. Concurrent involvement of these pontine structures produces the typical clinical features of nine syndrome. Patients develop ipsilateral horizontal gaze palsy, preventing both eyes from moving toward the lesioned side. This is accompanied by internuclear ophthalmoplegia, characterized by impaired adduction of the ipsilateral eye and nystagmus of the contralateral abducting eye. In addition, damage to the facial nerve fascicle results in ipsilateral lower motor neuron facial weakness involving the forehead, eyelid closure, and perioral muscles. Consequently, the only preserved horizontal eye movement is abduction of the eye opposite to the side of the lesion. The syndrome is named 'nine' because it is essentially the combination of one-and-a-half syndrome (horizontal gaze palsy + INO), ipsilateral LMN facial palsy, and contralateral hemiplegia, which can be caused by stroke, demyelination, infection, infiltrative disorders, tumours, etc [9]. If Eight-and-a-Half Syndrome has other-sided facial nerve palsy too, it is called Fifteen-and-a-Half Syndrome[11]. Neuroimaging, particularly magnetic resonance imaging (MRI), plays a crucial role in confirming the diagnosis and determining the underlying etiology, such as ischemic infarction, demyelinating disease, or space-occupying lesions. MRI also helps define the location and extent of the pontine lesion and the involvement of adjacent neural structures[10].

The management of nine syndrome depends primarily on its underlying cause. In cases resulting from ischemic stroke, patients may achieve partial or complete neurological recovery, particularly when early medical treatment is provided. Secondary prevention typically includes control of vascular risk factors such as hypertension and diabetes, along with antiplatelet or anticoagulant therapy when appropriate. If the syndrome is associated with demyelinating diseases, immunomodulatory treatment may lead to clinical improvement, although some neurological deficits can remain. When a structural lesion such as a tumor or cavernoma is responsible, surgical management may be required, and the prognosis varies according to the type and location of the lesion[8].

Rehabilitation in nine syndrome is multidisciplinary and focuses mainly on recovery of ocular motor and facial nerve function. The goal is to improve independence by promoting neuroplasticity through targeted exercises. Management of eye-movement deficits may include prism glasses, ocular-motor exercises, and, occasionally, botulinum toxin for persistent strabismus or nystagmus. Facial nerve rehabilitation involves facial muscle strengthening, visual feedback training, and lip–tongue exercises to improve facial movement and speech. Electrical stimulation and botulinum toxin may also be used in selected cases to enhance neuromuscular function and reduce abnormal muscle activity[12-13].

Conclusion

Nine syndrome is a rare neurological disorder that allows for precise localization of lesions in the pontine tegmentum. Prompt recognition, based on a precise neurological examination and appropriate neuroimaging, is critical for diagnosing serious underlying causes such as brainstem infarcts and masses. A clear comprehension of the underlying anatomy of nine syndrome aids in prompt diagnosis and management. While the prognosis for nine syndrome is generally favorable for vascular and demyelinating causes, it may require urgent treatment for space-occupying lesions. Prompt rehabilitation, including ocular and facial motor function, is an important aspect in the management of nine syndrome.

Ethical approval

Our institution does not require ethical approval for reporting individual cases or case series.

Informed consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

Declaration of conflicting interests

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