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Case Report

A rare neuro-ophthalmological presentation of Nine syndrome: clinical-radiological-anatomical correlation

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ABSTRACT

Nine syndrome is a very rare neuro-ophthalmological entity resulting from combined involvement of the abducens nucleus or paramedian pontine reticular formation (PPRF), the medial longitudinal fasciculus (MLF), the facial colliculus, and the corticospinal tract. This constellation produces the combination of horizontal gaze palsy, internuclear ophthalmoplegia, ipsilateral facial palsy, and contralateral hemiparesis/ hemianesthesia/ hemiataxia together constitute Nine syndrome (7th nerve+1 and 1/2 syndrome+1/2 hemiparesis/ hemianesthesia/ hemiataxia). Our objective was to present a case of nine syndrome providing a comprehensive clinical examination, detailed radiological evaluation and anatomical correlation to explain the neurological findings. This case highlights the importance of recognizing characteristic neuro-anatomical correlations in pontine stroke syndromes. We report a case of a 57-year-old male who presented with sudden-onset left hemiplegia, dysarthria, and binocular diplopia. Neurological examination revealed right horizontal conjugate gaze palsy, right internuclear ophthalmoplegia, right lower motor neuron facial palsy, and left hemiplegia-clinically consistent with nine syndrome. MRI brain revealed an acute right paramedian pontine infarct with basilar artery aneurysm. The patient was managed with antiplatelet therapy, high-intensity statin, glycemic control, and supportive care. Clinical improvement was noted over the following days. This case highlights the importance of recognizing combined cranial nerve and long-tract signs in localizing pontine lesions. Nine syndrome, though rare, should be considered in patients presenting with horizontal gaze palsies and contralateral hemiparesis and aggressive vascular risk-factor management is critical for preventing recurrence.

Keywords: Nine syndrome, One-and-a-half syndrome, Pontine infarct, Internuclear ophthalmoplegia, Facial palsy, Basilar artery aneurysm, Brainstem stroke

INTRODUCTION

Brainstem infarcts present with diverse neuro-ophthalmologic manifestations due to the compact organization of cranial nerve nuclei and long tracts. Pontine lesions usually present with gaze palsies in combination with other clinical signs. Combined involvement of the PPRF or abducens nucleus and the MLF resulting in horizontal conjugate gaze palsy and INO together called one-and-a-half syndrome, first described by Freeman et al in 1943.[1] Later, Fisher coined the term in 1967.[2] Eight-and-a-half syndrome represents one-and-a-half syndrome plus ipsilateral LMN facial weakness (cranial nerve VII). This was described by Eggenberger in 1998.[3] The even rarer Nine Syndrome includes eight-and-

a-half syndrome plus contralateral hemiparesis or hemisensory loss or hemiataxia due to involvement of the corticospinal and/or medial lemniscus tracts and/or inferior cerebellar peduncular fibres/red nucleus. Nine syndrome was first reported by Rosini et al.[4] Only a few cases have been reported in literature.[5] Here, we describe a patient presenting with classical features of this rare entity secondary to an acute right pontine infarction. This case underscores the importance of early diagnosis and aggressive management of vascular risk factors.

CASE REPORT

A 57-year-old male who is a known smoker and alcoholic with no other comorbidities presented with sudden-onset left upper and lower limb weakness associated with dysarthria and binocular diplopia. There was no history of headache, vomiting, seizures, loss of consciousness, trauma, or prior cerebrovascular events. On examination, the patient was conscious, alert, and oriented to time, place, and person. Higher mental functions were normal. Speech was dysarthric. Cranial nerve assessment on primary position revealed fixation of his right eye in the midposition and abduction of his left eye slightly to the left (paralytic pontine exotropia). Horizontal right-gaze showed both eyes unable to move past the midline of the primary position axis. While, left-gaze showed still fixated right eye on the primary position, and left eye abducting to the left with nystagmus. These conditions are hallmarks of one and half syndrome. A right lower motor neuron facial palsy was present, evidenced by incomplete eye closure and loss of the right nasolabial fold. Pupils were equal and reactive to light. Other cranial nerves examination was normal. Motor system examination showed significant left hemiplegia (MRC scale of power 0/5). Deep tendon reflexes were brisk on the left with an extensor plantar response. Sensory examination was normal. The constellation of findings-one-and-a-half syndrome, ipsilateral facial palsy, and contralateral hemiplegia-clinically established the diagnosis of Nine syndrome.

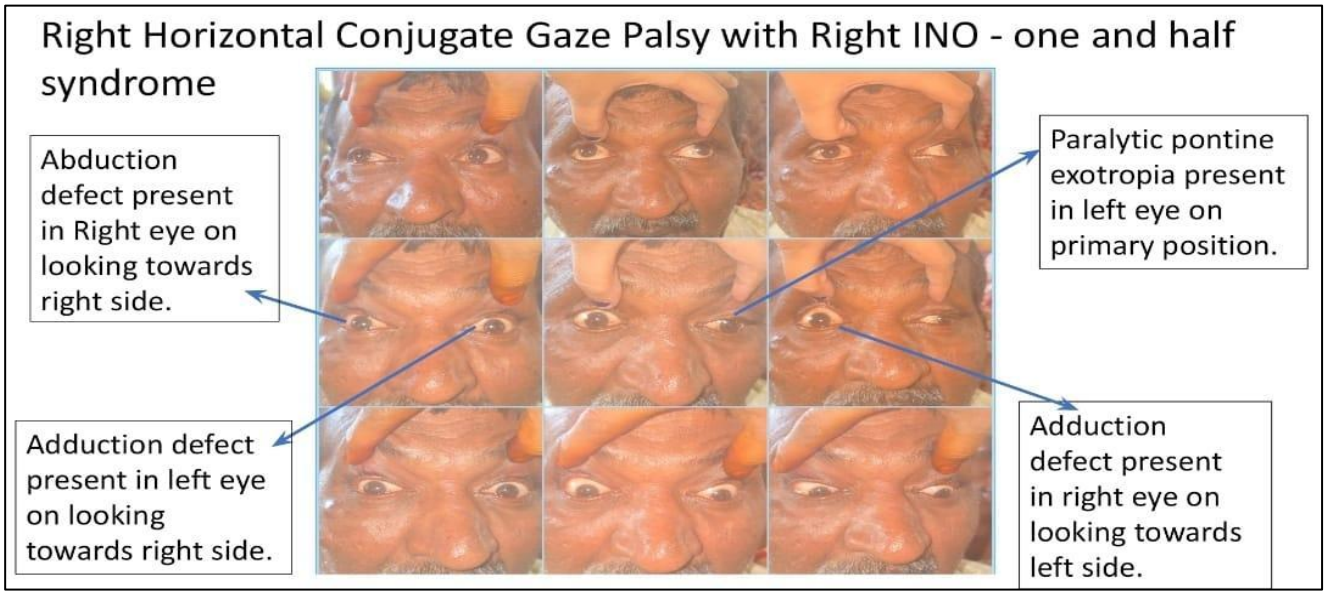

Figure 1: One-and-a-half syndrome.

Investigations and management

Magnetic resonance imaging (MRI) of the brain revealed an acute infarct involving the right paramedian region of the pons, correlating with the patient's ocular motor deficits and contralateral hemiplegia. In addition, small aneurysmal outpouchings were seen arising from the distal basilar artery and the right internal carotid artery, suggestive of underlying vascular pathology. Carotid Doppler ultrasound showed mild bilateral atheromatous plaques with no significant luminal stenosis. 2D Echo was normal. Routine hematological investigations were within normal limits except Hba1c 7.4 and dyslipidemia with elevated triglycerides (219 mg/dl). The patient was managed conservatively with antiplatelet therapy and high intensity statins, subcutaneous insulin regimen, and with supportive care. Over the course of hospitalization, the patient demonstrated gradual improvement in ocular motility and motor strength on the left side.

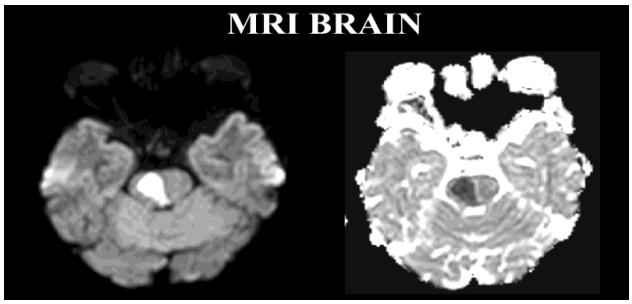

Figure 2: MRI brain.

*An acute infarct in right paramedian pontine region as evidenced by diffusion restriction on DWI with corresponding ADC hypointensity.

DISCUSSION

Nine syndrome represents a rare extension of eight-and-a-half syndrome, characterized by the addition of contralateral hemiparesis or hemisensory loss or hemiataxia due to corticospinal or medial lemniscus or inferior cerebellar peduncle/red nucleus involvement.[5] The syndrome arises from a lesion in the dorsal paramedian caudal pontine tegmentum where the abducens nucleus or PPRF, the MLF, facial colliculus, medial lemniscus, red nucleus, cerebellar peduncular fibres are arranged in close proximity extending slightly ventrally to involve corticospinal tracts. Damage to these structures' accounts for ipsilateral gaze palsy, internuclear ophthalmoplegia, lower motor neuron facial palsy, and contralateral hemisensory loss/hemiataxia.[6] This region is supplied by paramedian perforator branches of basilar artery.[7] The most common causes of INO are ischemic strokes and multiple sclerosis but rarely may be caused by trauma, infection, hemorrhage, tumors.[8] In our patient, the clinical findings were consistent with nine syndrome, and MRI confirmed an infarct localized to the exact anatomical structures responsible for this syndrome. Early recognition is important because a precise constellation of signs enables accurate brainstem localization even before imaging. Prognosis is generally favorable in unilateral, upper pontine lesions, with significant recovery following appropriate medical management and risk-factor control, as observed in this case.

CONCLUSION

Nine syndrome is an exceptionally rare pontine stroke syndrome with characteristic clinical features resulting from involvement of specific dorsal and ventral pontine structures. Since first description of Nine syndrome 13 yrs ago, there are other reported variants of the syndrome due to extension of brainstem stroke lesion to involve other parts resulting in various manifestations of this syndrome. Recognizing this combination of ocular motor deficits, facial weakness, and contralateral motor deficit is essential for precise brainstem localization. This case report contributes to the limited literature on nine syndrome and illustrates the importance of a meticulous neurological examination in stroke, as careful assessment enables accurate bedside localization of the lesion and guides timely, effective management.

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