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ONE AND A HALF SYNDROME DUE TO PONTINE HEMORRHAGE: A RARE CASE PRESENTATION

Neurology

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ABSTRACT

One and a Half Syndrome (OHS) is an infrequent ocular movement disorder characterized by combination of an ipsilateral conjugate horizontal gaze palsy (referring to the 'one' horizontal gaze palsy) and an ipsilateral internuclear ophthalmoplegia (INO) (referring to the 'half' of a horizontal gaze palsy) . Although most commonly due to ischemic stroke or demyelination, its occurrence with pontine hemorrhage is extremely infrequent. We present the case of a 45-year-old male with right hemisensory loss, diplopia, vomiting, and limited horizontal eye movements. Imaging showed a left pontine tegmentum hemorrhage. With supportive management, the patient recovered fully in six months. This case illustrates the diagnostic utility of early neuroimaging and the potential for positive outcomes in OHS secondary to pontine hemorrhage, despite its rarity.

KEYWORDS

One-and-a-half syndrome; brainstem stroke; internuclear ophthalmoplegia; ICH

INTRODUCTION

One and a Half Syndrome is a rare and distinct neurological disorder characterized by a specific pattern of horizontal eye movement weakness—complete conjugate gaze palsy in one direction ("one") and internuclear ophthalmoplegia (INO) in the other ("half"), often with abducting nystagmus while vertical gaze and convergence remain intact (1). It was first described by C. Miller Fisher in 1967, who noted that the syndrome involves a complete failure of lateral eye movement on the side of the lesion ("one") and impaired adduction of the contralateral eye ("half"), often accompanied by abducting nystagmus (2).

This syndrome is caused by a unilateral lesion of the dorsal pontine tegmentum, which affects the paramedian pontine reticular formation (PPRF) or abducens nucleus, as well as the medial longitudinal fasciculus (MLF) (1). Common causes include vascular events (particularly brainstem infarction), demyelinating illnesses including multiple sclerosis, tumors, infections, and trauma (3). Although vascular sources predominate, hemorrhagic lesions in the pontine tegmentum, particularly intracerebral hemorrhage, are extremely rare. Identifying this subtype is crucial for patient prognosis and care.

Case Presentation

A 45-year-old man arrived at the emergency department complaining of abrupt numbness and tingling on the right side of his body, including his face. These symptoms started the night before at around 10:00 p.m. There was no evidence of limb weakening linked with the sensory abnormality. He also complained of slurring his speech, diplopia, and limited horizontal eye movements. He also had four to five episodes of projectile vomiting, which contained consumed food. There was no prior history of vertigo, giddiness, dysphagia, gait instability, bowel or bladder problems, trauma, seizures, or loss of consciousness. The patient had no known hypertension, diabetes, or coronary artery disease.

On clinical evaluation, he was aware, attentive, and oriented, with intact higher mental functioning. A cranial nerve investigation revealed a horizontal gaze palsy with no conjugate eye movement to the left. (Figure 1) During an attempted rightward gaze, the right eye failed to adduct, whereas the left eye abducted with nystagmus. Vertical gaze and convergence were maintained. Motor assessment demonstrated normal muscle strength (Medical Research Council grade 5/5) in all four limbs. Sensory evaluation revealed impaired pinprick and light-touch sensations across the right side of body, including the face. There were no cerebellar symptoms.

Magnetic resonance imaging (MRI) of the brain revealed T1 weighted hyperintensity and FLAIR hyperintensity with hypointensity (hemosiderin) rim in the same location, indicating a pontine tegmental hemorrhage (Figures 2 and 3). Routine laboratory testing, such as a complete blood count, serum electrolytes, renal and liver function tests, were all within normal limits.

DISCUSSION

OHS is a rare but anatomically localized neuro-ophthalmologic syndrome caused by unilateral lesions in the dorsal pontine tegmentum. The condition presents as a horizontal gaze palsy ("one") due to PPRF or abducens nucleus involvement, combined with INO ("half") from damage to the ipsilateral MLF. These structures coordinate horizontal gaze; their disruption results in a distinctive pattern: loss of horizontal movement in the ipsilateral eye and abduction with nystagmus in the contralateral eye (4).

Our patient presented with left horizontal gaze palsy and right INO, along with right-sided hemisensory loss, consistent with a lesion in the left dorsal pontine tegmentum. MRI confirmed a focal hemorrhage in this region. The absence of cranial nerve VII palsy or motor tract involvement indicated that deeper and lateral pontine structures were spared.

Although ischemic stroke and demyelination are the leading causes of OHS, hemorrhagic etiologies are significantly rarer. Intracerebral hemorrhages make up 10–15% of all strokes, with pontine hemorrhages accounting for 5–10% of these (5). Focal hemorrhages confined to the dorsal pontine tegmentum that result in OHS are exceedingly uncommon (2). Timely recognition of the syndrome can aid in early localization and appropriate imaging.

Several case reports have discussed OHS due to pontine hemorrhage. Kofahi et al. (2020) reported a 48-year-old male with right pontine hemorrhage and OHS, showing substantial recovery with conservative management (6). Azevedo Júnior (1995) described a pontine tegmental hematoma leading to OHS and facial palsy with partial recovery (1). Nathan et al. (2024) detailed a hypertensive patient with OHS and hemiparesis secondary to a left pontine bleed, emphasizing early neuro-ophthalmologic recognition (7). Dionísio et al. (2007) also reported cases of pontine hemorrhage presenting with fever and dysarthria, highlighting the syndrome's varied presentations.

Non-vascular causes of OHS include neurocysticercosis, brainstem tuberculomas, Wernicke encephalopathy, metastases, and autoimmune diseases like systemic lupus erythematosus. Pseudo-OHS has been seen in ocular myasthenia gravis, where impaired adduction mimics INO (8). A vertical variant of the syndrome can occur with bilateral mesodiencephalic lesions. Deleu et al. (1989) described such a case with supranuclear downgaze palsy and monocular elevation deficit (9).

Our case aligns with these reports, with a localized dorsal pontine hemorrhage causing classic OHS signs. Imaging confirmed the diagnosis, and the patient recovered completely with conservative treatment, further supporting favorable outcomes with early identification and management.

CONCLUSION

This case highlights a rare presentation of One and a Half Syndrome due to a focal pontine hemorrhage. Despite its uncommon etiology,

early recognition of the characteristic ocular motor findings and prompt neuroimaging facilitated accurate localization and diagnosis. The patient's full recovery with conservative management underscores the importance of clinical vigilance and supportive care in such cases. Awareness of this rare syndrome can aid in timely diagnosis and favorable outcomes, even in hemorrhagic brainstem lesions.

Figure 1 - On right gaze, impaired adduction of the left eye with nystagmus of the abducting right eye

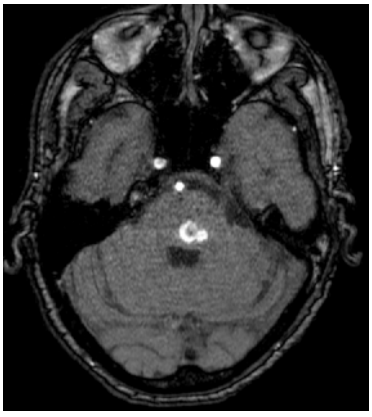

Figure -2 Magnetic resonance imaging (MRI) of the brain revealed T1 weighted hyperintensity more on the left side pontine tegmental region

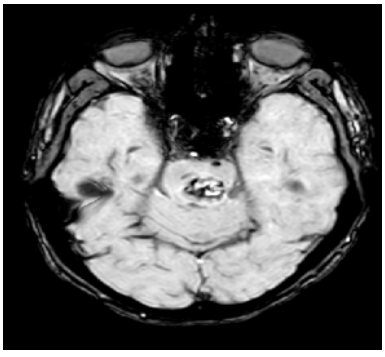

Figure -3 FLAIR hyperintensity with hypointensity (hemosiderin) rim in the left pontine tegmental region

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