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Case Report

Hypertrophic olivary degeneration without palatal tremor

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ABSTRACT

Brain atrophy and gliosis represent the typical long-term, permanent structural responses of the central nervous system following injuries such as trauma, stroke, or inflammation. Hypertrophy of a brain region following a damage in another part of brain is an unexpected finding and is unusual. Guillain Mollaret triangle is formed by ipsilateral red nucleus, ipsilateral inferior olivary nucleus and contralateral dentate nucleus. Damage to Guillain Mollaret triangle can lead to delayed development of hypertrophy of inferior olivary nucleus which is seen as hypertrophy and T2/FLAIR hyperintensity in magnetic resonance imaging. The classical clinical accompaniment of hypertrophic olivary degeneration (HOD) is palatal tremor which is present in around 50% cases. With time inferior olivary nucleus undergo atrophy but palatal tremor persists. Here we describe a case of unilateral HOD following traumatic brain injury (TBI) involving midbrain. Familiarity with the classic imaging findings of this rare phenomenon is necessary in order to avoid misdiagnosis and prevent unnecessary intervention.

Keywords: Hypertrophic olivary degeneration, Inferior olivary hypertrophy, Guillain Mollaret triangle, Traumatic brain injury

INTRODUCTION

The Guillain–Mollaret triangle is a neural circuit connecting the red nucleus of the midbrain with the inferior olivary nucleus (ION) of the medulla and the contralateral dentate nucleus of the cerebellum. This network constitutes the dentato-rubro-olivary pathway (DROP). Disruption of this circuit can lead to the loss of inhibitory regulation of the inferior olivary nucleus, resulting in neuronal hyperactivity and subsequent hypertrophic changes.[1] The abnormal rhythmic activity generated by the olivary neurons may produce oculopalatal tremor. The mechanisms underlying the development of oculopalatal tremor in only a subset of patients remain incompletely understood. On MRI, Hypertrophic olivary degeneration (HOD) may mimic a demyelinating lesion, highlighting the importance of correlating imaging findings with the clinical presentation. Furthermore, oculopalatal tremor may persist even after the hypertrophied inferior olivary nucleus undergoes subsequent atrophy. Pharmacological treatment of oculopalatal tremor remains largely ineffective.[2] Clinically, HOD presents classically as palatal tremor and can include dentato-rubral tremor and/or ocular myoclonus.[3]

CASE REPORT

A 19-year-old boy had a road traffic accident in October 2023 and was comatose and was on ventilator for few days. When he regained consciousness, he was noted to have titubation, cerebellar dysarthria, left finger nose incoordination and swaying to left while walking. His motor power was normal. There was left hemi hypoalgesia. There was no palatal tremor or ocular myoclonus. Computerized tomogram done at that time

showed right ponto mesencephalic hemorrhage. MRI brain taken 14 months after the incident showed right inferior olivary hypertrophy (Figure 1). He was treated with clonazepam, propranolol, lamotrigine and gabapentin. Last follow up, three years after the TBI, he is able to walk with minimal support and has left central deafferentation pain.

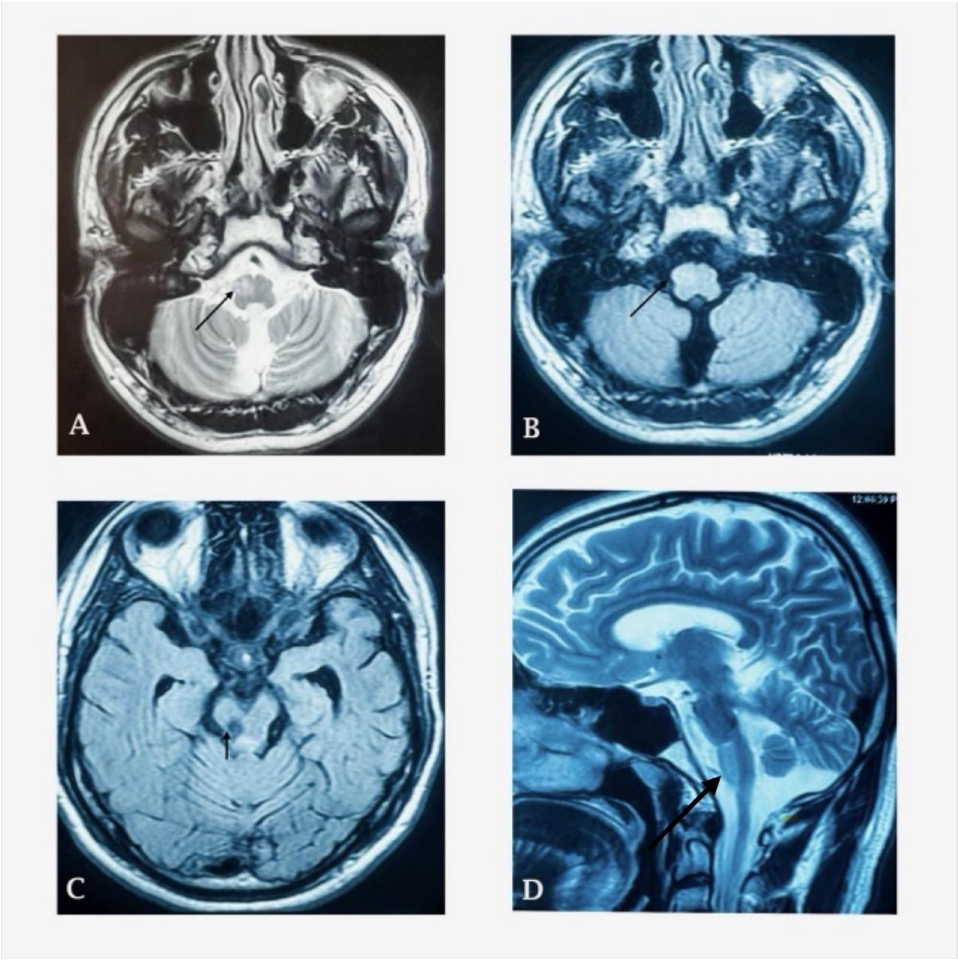


Figure 1: (A) T2 axial MRI brain image showing hyperintensity and enlargement of right inferior olivary nucleus; (B) axial FLAIR image showing hyperintensity of right ION; (C) axial FLAIR showing sequela of old haemorrhage in midbrain tegmentum on right side with gliosis extending to opposite side; (D) sagittal T2 weighted image showing vertical extent of HOD in medulla. Sequela of old haemorrhage can be seen extending to pons.

DISCUSSION

HOD or inferior olivary hypertrophy typically arises from a lesion within the Guillain-Mollaret triangle (myoclonic triangle). Climbing fibers originating from the ION travel through the contralateral inferior cerebellar peduncle to synapse with Purkinje cells in the cerebellar cortex, which then project back to the dentate nucleus. Efferent signals from the dentate nucleus cross the midline via the superior cerebellar peduncle to reach the contralateral red nucleus. The red nucleus, in turn, sends fibers via the central tegmental tract (CTT) to the ipsilateral ION (Figure 2) Red nucleus inhibits ION via neurotransmitter gamma-aminobutyric acid (GABA).

It was first described by Oppenheim in 1887 during autopsy. In 1931, Guillain and Mollaret proposed that the onset of HOD was associated with damage to the dentato-rubro-olivary pathway and detailed its anatomical relationship. Typically, patients exhibit symptoms such as palatal tremor, nystagmus, and cerebellar ataxia, which paradoxically appear as their initial neurological deficits improve. HOD is not an uncommon sequela in patients with cerebellar or brainstem haemorrhages. Patients with cerebellar haemorrhages tended to develop HOD more rapidly compared to those with brainstem haemorrhages and patients who develop secondary HOD generally face a less favourable prognosis.[4]

Disruption of dentato-rubro-olivary pathway can lead to HOD, often presenting clinically with palatal tremor or oculo-palatal tremor. These tremors result from loss of inhibitory control over the ION, causing abnormal neuronal activity and rhythmic movements. Palatal tremor has been subdivided into essential (EPT) and symptomatic palatal tremor (SPT). The syndrome of progressive ataxia and palatal tremor (PAPT) is a subgroup of SPT and is characterized by bilateral HOD.[5] The levator veli palatini

muscle is predominantly affected in symptomatic palatal tremor, leading to visible rhythmic contractions of the soft palate.[6]

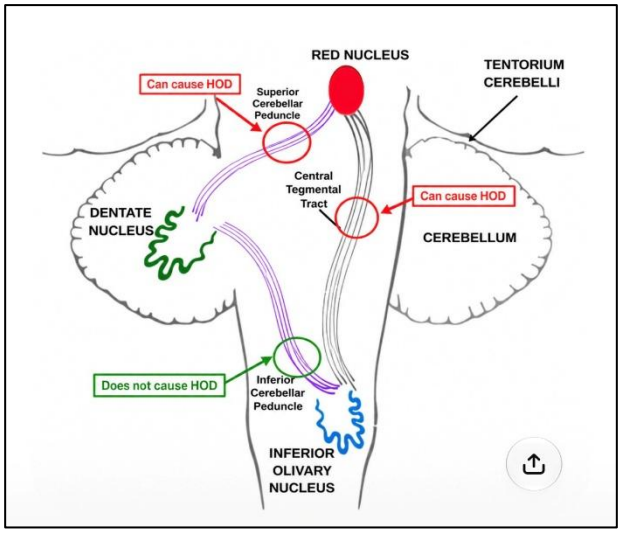


Figure 2: Guillain Mollaret triangle is formed by ipsilateral red nucleus, ipsilateral inferior olivary nucleus and contralateral dentate nucleus. Fibers connecting these nuclei are superior cerebellar peduncle (dentate nucleus to contralateral red nucleus), central tegmental tract (red nucleus to inferior olivary nucleus) and olivocerebellar tract (through inferior cerebellar peduncle). Lesions along dentato rubral and rubro olivary tracts cause HOD, while olivocerebellar lesions do not cause HOD.

The laterality of HOD depends on the lesion's location: involvement of CTT results in olivary hypertrophy on same side, whereas lesions affecting the dentate nucleus or superior cerebellar peduncle produce olivary hypertrophy on opposite side. Midline lesions in the brachium conjunctivum that affect decussation of the dentato rubral pathway may lead to bilateral HOD.

Unlike typical neurodegeneration, which involves neuronal loss and glial replacement, HOD is an example of trans-synaptic degeneration secondary to loss of afferent input to the ION that results in hypertrophy of the ION. Pathological features include neuronal enlargement, cytoplasmic vacuolation, astrocytic proliferation and hyperplasia, demyelination, and fibrillary gliosis.[7]

Common aetiologies of HOD include stroke (most often haemorrhagic), traumatic brain injury, demyelinating diseases, brainstem tumors, and interventions such as surgery or gamma knife therapy for brainstem cavernomas.[8] In addition, abscess, metronidazole intoxication, gluten sensitivity, and multifocal leukoencephalopathy have been reported as aetiologies.[9]

The MRI changes in HOD evolve over time: T2 hyperintensity emerges around 1 month after the initial insult and can persist for 3–4 years. Olivary hypertrophy typically develops by 6 months post-lesion, eventually resolving over the next 3 to 4 years.[10] Palatal tremor usually persists lifelong even after subsequent inferior olivary atrophy.

Forty-four percent of patients with HOD did not demonstrate an MRI-identifiable lesion in the DROP, and 45% of those patients did not have identifiable brain lesions outside of the DROP. Thus, nearly a fifth of HOD cases may be truly idiopathic. A majority of patients without a DROP lesion had bilateral HOD and two-thirds were male.[11] In a neurosurgical series of sixteen patients with HOD, seven patients (43.7%) had posterior fossa tumors, 6 patients (37.5%) had cavernoma, 2 patients (12.5%) sustained traumatic brain injury, and only 1 patient (6.2%) had cerebellar infarction.[12]

CONCLUSION

Hypertrophy of inferior olivary nucleus after traumatic brain injury is an uncommon event. As it is a delayed consequence of injury to Guillain Mollaret triangle, it is absent in initial MRI and it should be looked for in all cases of ponto-mesencephalic or cerebellar hemorrhage in follow up MRI. Familiarity with the classic imaging findings of this rare phenomenon is necessary in order to avoid misdiagnosis and prevent unnecessary investigations.

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REFERENCES

1. Wang H, Wang Y, Wang R, Li Y, Wang P, Li J, et al. Hypertrophic olivary degeneration: a comprehensive review focusing on etiology. Brain Res. 2019;1718:53-63.
2. Murdoch S, Shah P, Jampana R. The Guillain-Mollaret triangle in action. Pract Neurol. 2016;16(3):243-6.
3. Carvalho CH, Kimmig H, Lopez WO, Lange M, Oeckler R. Hypertrophic olivary degeneration: a neurosurgical point of view. J Neurol Surg A Cent Eur Neurosurg. 2016;77(1):59-62.
4. Lin L, Ye X, Zheng J. Clinical characteristics of hypertrophic olivary degeneration following brainstem or cerebellar hemorrhage. Sci Rep. 2025;15:31262.
5. Samuel M, Torun N, Tuite PJ, Sharpe JA, Lang AE. Progressive ataxia and palatal tremor (PAPT): clinical and MRI assessment with review of palatal tremors. Brain. 2004;127(Pt 6):1252-68.
6. Bhattacharjee S. Palatal tremor: pathophysiology, clinical features, investigations, management and future challenges. Tremor Other Hyperkinet Mov (N Y). 2020;10:40.

7. Gerace C, Fele MR, Luna R, Piazza G. Neurological picture: bilateral hypertrophic olivary degeneration. J Neurol Neurosurg Psychiatry. 2006;77(1):73.
8. Vyas S, Prabhakar A, Kumar A, Khandelwal N. Bilateral hypertrophic olivary degeneration. Ann Indian Acad Neurol. 2013;16(3):404-5.
9. Dogan SN. Hypertrophic olivary degeneration and Holmes tremor: case report and review of the literature. World Neurosurg. 2020;137:286-90.
10. Goyal M, Versnick E, Tuite P, Cyr JS, Kucharczyk W, Montanera W, et al. Hypertrophic olivary degeneration: meta-analysis of the temporal evolution of MR findings. AJNR Am J Neuroradiol. 2000;21(6):1073-7.
11. Gu CN, Carr CM, Kaufmann TJ, Kotsenas AL, Hunt CH, Wood CP. MRI findings in nonlesional hypertrophic olivary degeneration. J Neuroimaging. 2015;25(5):813-7.
12. Onen MR, Moore K, Cikla U, Ucer M, Schmidt B, Field AS, et al. Hypertrophic olivary degeneration: neurosurgical perspective and literature review. World Neurosurg. 2018;112.

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