

Refractory Trigeminal Neuralgia in MOGAD: Recognizing and Managing an Atypical Pain Phenotype



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Introduction

- Myelin oligodendrocyte glycoprotein antibody–associated disease (MOGAD) is an inflammatory demyelinating disorder distinct from multiple sclerosis and neuromyelitis optica.
- While optic neuritis and myelitis are common, cranial neuropathic pain syndromes such as trigeminal neuralgia (TN) are rarely reported.
- Recognition of TN as a manifestation of MOGAD is essential for appropriate treatment, as standard TN therapies may be ineffective.

Case Description

- 41-year-old female with known MOGAD presented with severe, refractory left-sided facial pain described as sharp, electric shocks.
- Pain localized to V2 and V3 distributions, unresponsive to carbamazepine, gabapentin, and corticosteroids.
- MRI revealed T2 hyperintensities along the left trigeminal nerve root entry zone; CSF/serum positive for MOG-IgG.
- Diagnosed with MOGAD-related trigeminal neuralgia; initiated on rituximab with significant improvement over 3 months.

Figures

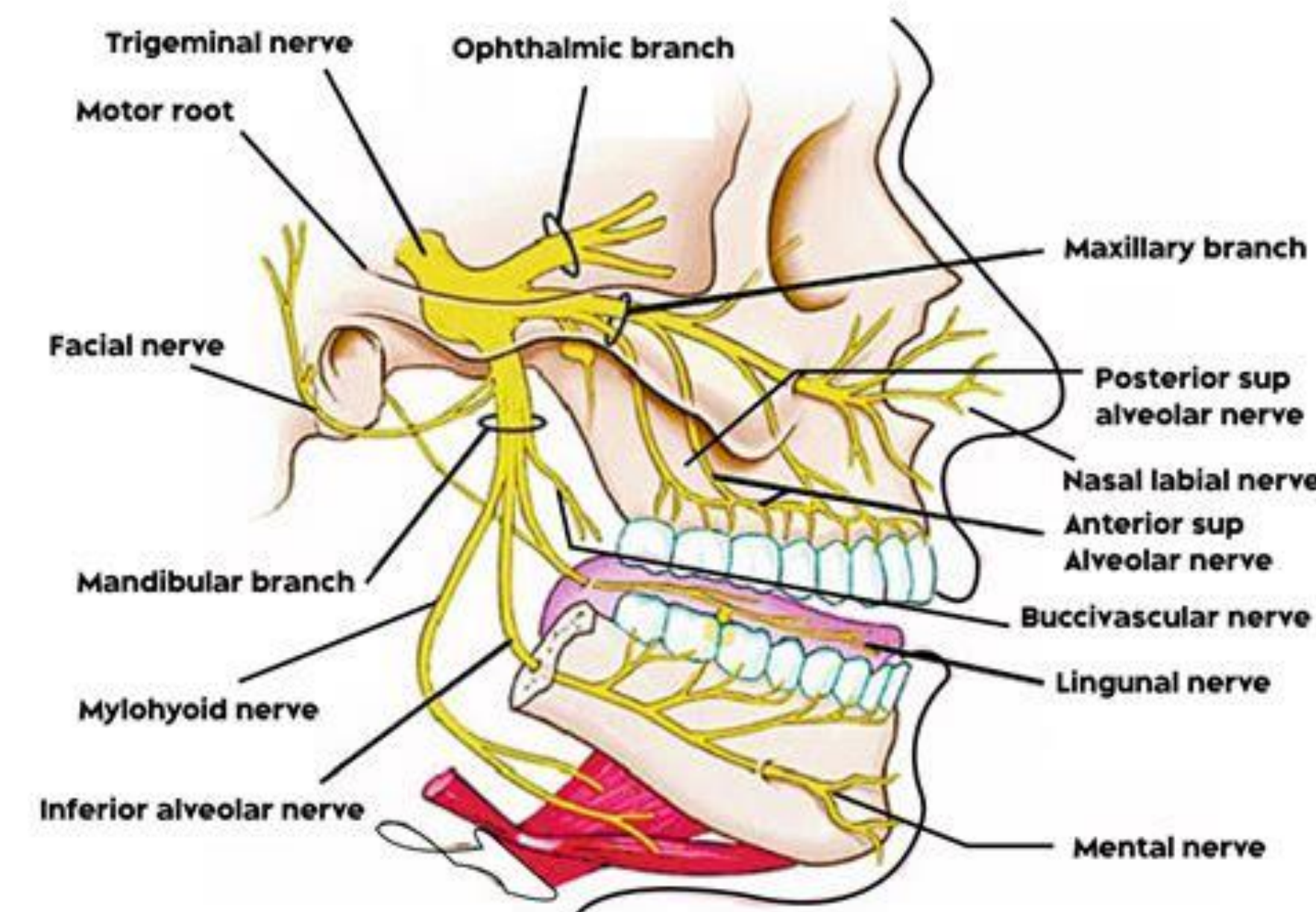
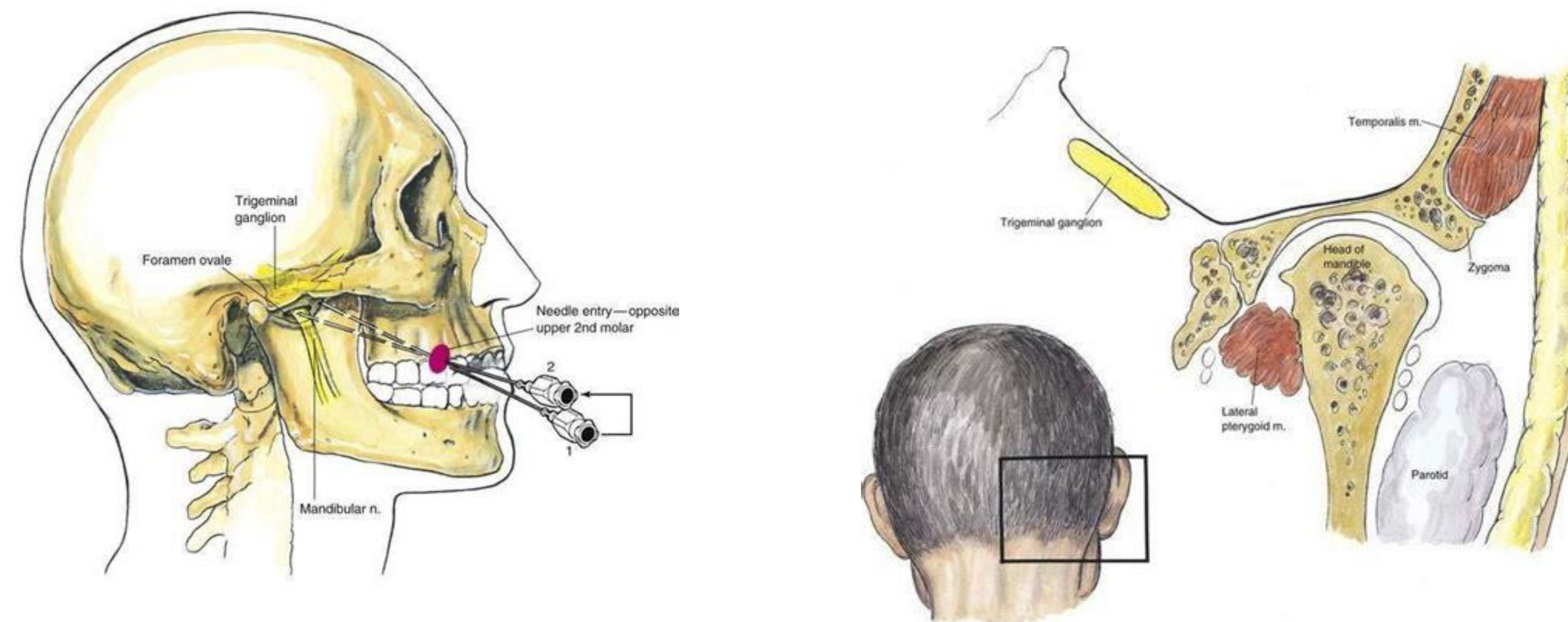


Figure 1: Trigeminal Divisions (V1–V3)



Figures 2 and 3: Gasserian Ganglion Anatomy

Discussion

- MOGAD can present atypically with cranial neuropathic pain due to inflammatory demyelination of trigeminal pathways.
- Conventional neuropathic pain medications may fail due to the immune-mediated nature of the condition.
- Early immunotherapy (e.g., rituximab, IVIG, corticosteroids) can relieve symptoms and prevent progression.
- Consider autoimmune demyelinating disease in refractory or atypical TN and coordinate with neurology.

References

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