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TRIGEMINAL NEURALGIA: PATHOPHYSIOLOGY, DIAGNOSIS AND MODERN MANAGEMENT APPROACHES

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Abstract

Trigeminal neuralgia (TN) is a chronic facial pain syndrome characterized by brief, recurrent episodes of intense, unilateral, shock-like pain within the sensory distribution of the trigeminal nerve. Despite its relatively low prevalence, the disorder causes marked functional disability and deterioration in quality of life. The underlying mechanism in classical TN is most commonly neurovascular compression that induces focal demyelination and abnormal excitability of trigeminal fibers. Secondary forms may develop in association with demyelinating diseases, tumors, or structural intracranial pathology. Diagnosis relies primarily on clinical evaluation, supported by neuroimaging to exclude secondary etiologies. Pharmacological therapy with sodium channel–modulating anticonvulsants remains first-line treatment, while surgical interventions are reserved for refractory cases. This article presents a synthesized overview of current concepts regarding the mechanisms, diagnosis, and management of trigeminal neuralgia.

Keywords

trigeminal neuralgia, neuropathic facial pain, neurovascular compression, carbamazepine, neurosurgical treatment

Introduction

Trigeminal neuralgia represents one of the most severe manifestations of neuropathic pain encountered in clinical neurology. The condition involves the fifth cranial nerve, which mediates facial sensation and contributes to mastication. Epidemiological data indicate that TN predominantly affects middle-aged and elderly individuals, with a slightly higher incidence among women. Although uncommon, its dramatic symptomatology makes early recognition essential for appropriate management.

Clinically, trigeminal neuralgia is categorized into classical, secondary, and idiopathic variants. Classical cases are usually associated with vascular compression near the nerve root entry zone. Secondary TN may arise due to multiple sclerosis plaques, neoplastic processes, or structural abnormalities. Differentiating between these forms is important for selecting optimal therapeutic strategies and predicting prognosis.

Materials and Methods

This narrative review was developed through analysis of peer-reviewed publications indexed in major biomedical databases, including PubMed and Scopus. Priority was given to recent reviews, clinical guidelines, and high-quality observational studies addressing epidemiology, pathogenesis, diagnostic criteria, and treatment outcomes. The aim was to provide an updated and clinically relevant synthesis rather than a systematic meta-analysis.

Anatomical Considerations

The trigeminal nerve comprises three principal divisions: ophthalmic (V1), maxillary (V2), and mandibular (V3). While all branches may be involved, pain most frequently affects the

maxillary and mandibular divisions. Understanding this anatomical distribution is essential for accurate clinical localization and planning of interventional procedures.

Pathophysiology

The dominant pathogenetic hypothesis involves chronic pulsatile compression of the trigeminal root by adjacent vascular structures. This mechanical irritation leads to focal demyelination and ectopic impulse generation. As a result, abnormal ephaptic transmission between fibers contributes to paroxysmal pain attacks. In secondary TN, inflammatory or demyelinating lesions may disrupt trigeminal pathways and produce similar clinical manifestations.

Clinical Presentation

Patients typically report sudden, lancinating facial pain lasting from seconds to minutes. Attacks are commonly triggered by innocuous stimuli such as speaking, chewing, washing the face, or exposure to cold air. Many individuals identify specific trigger zones, and symptom-free intervals are characteristic in early disease stages. With disease progression, some patients develop persistent background discomfort between paroxysms.

Diagnosis

Diagnosis is primarily clinical and guided by internationally accepted criteria. Neuroimaging, particularly magnetic resonance imaging, plays a crucial role in excluding secondary causes and identifying neurovascular conflict. Careful differential diagnosis is necessary to distinguish TN from dental disorders, temporomandibular joint dysfunction, postherpetic neuralgia, and other craniofacial pain syndromes.

Management

Pharmacotherapy remains the cornerstone of initial treatment. Sodium channel blockers such as carbamazepine and oxcarbazepine are considered first-line agents due to their efficacy in reducing paroxysmal discharges. Alternative medications include gabapentinoids and muscle relaxants, especially in patients with intolerance to first-line therapy.

Surgical treatment is indicated for medically refractory cases. Microvascular decompression directly addresses vascular compression and offers durable relief in appropriately selected patients. Less invasive options, including percutaneous ablative procedures and stereotactic radiosurgery, may be preferable for elderly individuals or those with significant comorbidities.

Prognosis

Clinical outcomes vary depending on etiology and treatment modality. Although many patients initially achieve symptom control with medication, relapse over time is common. Surgical approaches, particularly microvascular decompression, provide the longest duration of remission in classical TN. Persistent pain can significantly affect psychological well-being, emphasizing the need for holistic management.

Conclusion

Trigeminal neuralgia is a debilitating neuropathic disorder that demands timely recognition and individualized management. Advances in neuroimaging and neurosurgical techniques have

improved diagnostic precision and therapeutic outcomes. Future research focused on targeted neuromodulation and molecular mechanisms may further refine treatment strategies and enhance patient quality of life.

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