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TRIGEMINAL NEURALGIA: CLINICAL PRESENTATION, DIFFERENTIAL DIAGNOSIS, AND COMPREHENSIVE MANAGEMENT STRATEGIES

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Abstract

Trigeminal neuralgia (TN) is a severe neurological condition characterized by excruciating paroxysmal facial pain. This article provides a comprehensive analysis of the disease, covering its prevalence, etiology, and classification into central and peripheral forms [1, 2]. Special attention is given to the differential diagnosis of TN from other prosopalgias and dental conditions, such as pulpitis and periodontitis [3, 4]. The study reviews contemporary treatment modalities, including pharmacological interventions (carbamazepine, phenytoin), physiotherapy, and various surgical techniques, such as microvascular decompression and neurectomy [5, 7]. The goal is to provide a synthesized clinical guide for the diagnosis and management of this complex pathology [9].

Keywords

Trigeminal neuralgia, facial pain, differential diagnosis, carbamazepine, microvascular decompression

Introduction

Prosopalgia, defined as facial pain of various origins, represents a significant challenge in clinical practice [5]. Trigeminal neuralgia (TN) is among the most frequent causes of such pain, with a prevalence of 30 to 50 cases per 100,000 population and an annual incidence of 2 to 4 people per 100,000 according to the World Health Organization [1, 2]. The condition most commonly affects women aged 50–69 and often exhibits right-sided localization [1]. Factors contributing to its development include vascular disorders, endocrine-metabolic changes, allergic reactions, and psychogenic factors [1, 5]. Stomatoneurology occupies a critical place in clinical dentistry because patients suffering from facial pain often consult dentists before being referred to other specialists [6, 9].

Classification and Etiology

Trigeminal neuralgia is classified into two primary forms based on its genesis: central and peripheral [1, 2]. It is crucial not to conflate these forms, as they possess distinct clinical progressions and etiologies [4].

* Central Genesis: Often occurs in individuals aged 40–60 (68% female). Etiological factors include vascular diseases (hypertension), trauma, infections (influenza, ARVI), CNS lesions (arachnoiditis, encephalitis), and endogenous intoxications [1, 5].

* Peripheral Genesis: Primarily caused by tumors or tumor-like formations in facial soft tissues or bones, trauma from dental prostheses, ossification of the mandibular canal, or viral infections like herpes zoster [6, 7].

Clinical Presentation

The hallmark of TN is a sudden, sharp, "electric shock-like" pain that lasts from a few seconds to several minutes [1]. These paroxysms can occur spontaneously or be triggered by facial movements such as talking, eating, washing, or shaving [5].

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Trigger Zones (Trigger Points): For central TN (occurring in 84% of cases), the presence of trigger zones is characteristic [1, 9]. These are specific areas on the skin (nasolabial region) or oral mucosa (palate, alveolar process) where even slight irritation provokes an attack.

Associated Symptoms: Pain may be accompanied by vegetative manifestations, including facial hyperemia, lacrimation, rhinorrhea, and occasionally hyperselevation [4]. Patients often exhibit facial muscle hyperkinesis (twitching) during an attack and may freeze in a specific posture to avoid exacerbating the pain [1]. Long-term sufferers may experience skin dryness, loss of eyelashes, or even atrophy of the masticatory muscles on the affected side [4, 6].

Differential Diagnosis

Accurate diagnosis is vital, as TN symptoms often overlap with other conditions.

* Dental Diseases: Unlike acute pulpitis, TN pain is rarely spontaneous at night and is triggered by touching "trigger zones" rather than thermal stimuli [3]. In acute periodontitis, the pain is constant, localized to a specific tooth, and increases with percussion, whereas TN pain is paroxysmal and radiates along the nerve branches [4, 6].

* Maxillary Sinusitis: Odontogenic sinusitis presents with constant pain, nasal discharge, and sinus opacification on X-rays, none of which are present in typical TN [6, 7].

* Costen's Syndrome (Myofascial Pain): This involves dull aching in the temporomandibular joint (TMJ), clicking sounds, and muscle tenderness, often linked to bite abnormalities [8].

* Other Prosopalgias:

* Sluder's Syndrome: Sharp pain in the periorbital and nasal region with significant vegetative symptoms, relieved by cocainization of the nasal cavity [4].

* Eagle's Syndrome: Pain caused by an elongated styloid process (over 25 mm), triggered by swallowing or turning the head [8, 9].

* Glossopharyngeal Neuralgia: Pain starts in the root of the tongue or tonsils and is triggered by swallowing [1].

Treatment

1. Conservative Pharmacotherapy

The primary pharmacological agent for TN is Carbamazepine (Finlepsin) [5]. It stabilizes the membranes of overexcited neurons by blocking sodium channels. The starting dose is usually 0.1 g 2–3 times a day, gradually increasing until the pain is relieved [1, 9].

Other medications include:

* Phenytoin (Diphenin): Used if Carbamazepine is ineffective [5].

* Gabapentin: Effective in cases related to multiple sclerosis [9].

* Baclofen: Relieves muscle spasms and provides analgesic effects [1].

* B-Vitamins (Milgamma, Neurobion): These stimulate metabolism in neurons, restore nerve tissue, and enhance the effect of pain relievers [6, 9].

2. Physiotherapy

During acute periods, therapies such as UV radiation, UHF, and electrophoresis with novocaine or diphenhydramine are prescribed [5]. Diadynamic currents (Bernard currents) are particularly effective for their analgesic action, as they inhibit pain impulses and promote the release of endorphins [6].

3. Therapeutic Blockades

Perineural blockades involve injecting local anesthetics (novocaine, lidocaine) or mixtures (hydrocortisone, B-vitamins) into the exit points of the trigeminal branches [7, 8]. While these provide temporary relief, repeated use (15–20 injections) can have a therapeutic effect. It is

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noted that "alcoholization" (ethanol injection) is now viewed negatively due to the risk of neuritis, scarring, and recurrence [7].

4. Surgical Interventions

When conservative methods fail, surgical options are considered:

* Microvascular Decompression: Used when an abnormally placed artery compresses the nerve. A cushion is placed between the vessel and the nerve under general anesthesia [1, 2].

* Gamma Knife: Destroys part of the nerve using ionizing radiation without incisions [9].

* Neurectomy and Decompression: Peripheral forms may be treated by removing the nerve from bone canals (e.g., Korolenko method for the III branch) [7, 8].

* Laser Destruction: Modern techniques use high-frequency electromagnetic waves or lasers to destroy the nerve root through a puncture needle, which is suitable for elderly patients [9].

Discussion

The management of trigeminal neuralgia requires a personalized approach [8]. Conservative treatment with Carbamazepine remains the gold standard, but its long-term use can be limited by side effects or decreasing efficacy [5]. Physiotherapeutic methods like diadynamic currents significantly improve patient outcomes by reducing edema and normalizing trophic processes [6].

Regarding surgical interventions, decompression operations show high success rates (nearly 90%), particularly when the nerve is freed from narrowed bone canals [7]. However, patients must be warned that surgery often results in permanent sensitivity loss (numbness) in the affected area [7, 8]. While this numbness is unpleasant, most patients find it far more bearable than the preceding neuralgic pain. The use of alcoholization should be discouraged as it leads to scarring and makes future surgeries more difficult [7].

Conclusion

Trigeminal neuralgia is one of the most painful conditions known to medicine, significantly impacting the quality of life [1]. Successful management depends on a precise differential diagnosis to exclude dental pathologies and other facial pain syndromes [4, 9]. A combination of anticonvulsants, vitamins, and physiotherapy often provides relief in early stages [5, 6]. For resistant cases, modern surgical techniques like microvascular decompression or laser destruction offer high efficacy and safety, even for elderly patients [2, 9].

REFERENCES

1. Karlov, V.A. Facial Neuralgia. Moscow: Meditsina, 1991.
2. Megdatov, R.S. Trigeminal Neuralgia. Moscow: Meditsina, 1999.
3. Mukovozov, I.N. Differential Diagnosis of Surgical Diseases. Leningrad: Meditsina, 1982.
4. Puzin, M.N. Neurostomatological Diseases. Moscow: Meditsina, 1997.
5. Erokhina, L.G. Facial Pain. Moscow: Meditsina, 1973.
6. Grechko, V.E., Puzin, M.N., Stepanchenko, A.V. Odontogenic Lesions of the Trigeminal Nerve System. Moscow, 1988.
7. Robustova, T.G. Surgical Stomatology. Moscow: Meditsina, 2003.
8. Timofeev, A.A. Manual of Maxillofacial Surgery and Surgical Stomatology. Kiev, 2002.
9. Gaivoronskaya, T.V., Sveshnikov, G.G., et al. Trigeminal Neuralgia: A Textbook. Krasnodar: KubGMU, 2012.