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Morphofunctional Changes in the Trigeminal Nerve in Inflammatory Diseases of the Maxillofacial Region

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Abstract. Trigeminal neuralgia (TN) is the most common form of prosopalgia and is characterised by brief, intense paroxysmal pain in the territory of one or more branches of the trigeminal nerve. Inflammatory and odontogenic processes of the maxillofacial region are considered among its triggering factors. The aim of this article is to analyse and summarise current data on the anatomical prerequisites, etiological factors and morphofunctional changes of the trigeminal nerve underlying TN, with particular attention to the role of inflammatory diseases of the maxillofacial region. An analytical review of foreign and Russian-language publications from 2000–2010 was carried out using comparative analysis, generalisation and systematisation. The analysis showed that the narrow bony canals through which the branches of the nerve exit, inflammatory processes leading to arachnoid adhesions, and neurovascular compression of the nerve root result in demyelination of type A fibres, formation of ectopic excitation foci and paroxysmal pain. Treatment options range from anticonvulsants to botulinum toxin and radiosurgery, but each has limitations, which justifies further research on pathogenetically oriented therapy.

Keywords: Trigeminal neuralgia; trigeminal nerve; prosopalgia; demyelination; neurovascular compression; inflammatory diseases; maxillofacial region.

1. Introduction

Trigeminal neuralgia is the most common and best-known form of prosopalgia. Its high prevalence, the severity and intensity of the pain syndrome, diagnostic difficulties and problems of therapy determine both the scientific and practical importance of this pathology. The prevalence of TN is estimated at 30–50 patients per 100,000 population. The leading clinical feature is excruciating pain in the territory of one of the branches of the trigeminal nerve, lasting from a few seconds to 1–2 minutes. With a prolonged course, these paroxysms alter the physical and psycho-emotional state of the patient, contribute to depressive and emotional-volitional disorders, and reduce quality of life.

TN was described as a separate nosological entity more than 200 years ago. The first description in Western literature belongs to the Venetian physician Massa (1550); in 1756 André described its paroxysmal manifestations as a "painful tic" provoked by chewing, talking and coughing, and the British physician John Fothergill described it as a separate disease, which is why TN is also called Fothergill's disease. Despite this long history, many questions of its etiology, pathogenesis and treatment remain unresolved or debatable, and resistance to standard therapy is frequently observed. This makes it necessary to clarify the pathogenetic features of TN, including the role of inflammatory diseases of the maxillofacial region.

The aim of this article is to analyse and summarise current data on the anatomical prerequisites, etiological factors and morphofunctional changes of the trigeminal nerve underlying

TN, with particular attention to the role of inflammatory diseases of the maxillofacial region.

2. Materials and Methods

The study was conducted as an analytical review of foreign and Russian-language publications from 2000–2010 devoted to the anatomy of the trigeminal nerve, the etiology and pathogenesis of TN, the morphological changes of its myelinated fibres, neuroimaging of neurovascular compression, and methods of conservative and surgical treatment. The methods of comparative analysis, generalisation and systematisation of scientific data were used. The findings were grouped into four areas: anatomical prerequisites, etiological factors, morphofunctional changes of the nerve, and the mechanism of pain formation.

3. Results

Anatomical prerequisites

The trigeminal nerve is a mixed nerve containing sensory fibres that innervate the face and motor fibres that innervate the masticatory muscles. The bodies of the first sensory neurons are located in the trigeminal (Gasserian) ganglion in Meckel's cave on the anterior surface of the petrous part of the temporal bone. Their peripheral processes form three branches: the ophthalmic nerve, which exits through the superior orbital fissure; the maxillary nerve, through the foramen rotundum; and the mandibular nerve, the largest and mixed branch, through the foramen ovale. All exit points are limited in diameter, which creates conditions for compression of the nerve by surrounding tissues.

The central processes enter the brain stem as the sensory root and terminate in the principal (pontine) nucleus, which provides tactile sensitivity, the mesencephalic nucleus, which provides proprioceptive sensitivity, and the spinal nucleus, which provides pain and temperature sensitivity. The trigeminal nerve provides sensitivity to the skin of the face, the eyeball, the mucous membranes of the nasal and oral cavities, the teeth and the anterior two-thirds of the tongue. Close anatomical contact of the nerve root and nuclei with the large vessels of the posterior cranial fossa underlies the vascular theory of TN.

Etiological factors

TN occurs more often in women over 40 years of age, with predominant involvement of the maxillary and mandibular branches; the pain is usually unilateral, more often right-sided, and is provoked by irritation of trigger zones during washing, brushing teeth or chewing. Among the proposed causes are narrowing of the bony canals through which the branches pass, increased sensitivity of the nerve to hypothermia of the face, a high position of the petrous apex on the affected side, and stenosis of the mandibular canal; however, recurrences after surgical widening of the canal indicate that canal stenosis is not the only cause. TN has also been associated with intoxication, infection, facial and head injuries, arterial hypertension, atherosclerosis, metabolic disorders and allergic processes.

Of particular importance for maxillofacial practice are odontogenic and inflammatory factors: traumatic tooth extraction with damage to the alveolar nerves, retained root fragments or bone splinters in the socket, pulpitis, osteomyelitis and inflammatory diseases of the paranasal sinuses. The pain syndrome in odontogenic TN is moderate and prolonged, without trigger zones. Inflammatory processes such as otitis media, sinusitis and dental caries also lead to the formation of coarse arachnoid adhesions, which contribute to the development of neurovascular conflict.

Morphofunctional changes of the nerve

The generally accepted mechanism of classical TN is compression of the trigeminal nerve root by an adjacent vessel, most often the superior cerebellar artery and less often the anterior or posterior inferior cerebellar arteries; veins can also cause compression and recurrence of TN after microvascular decompression [1]. Sites of neurovascular compression can be visualised by magnetic resonance cisternography [2], but vascular contact with the nerve is also frequently found in asymptomatic individuals [3], so its presence alone does not explain the disease. Pulsating compression of the root leads to demyelination, and ultrastructural and immunohistochemical

changes of the peripheral myelinated axons have been demonstrated in patients with TN [4]. With age, atherosclerosis coarsens the arterial walls and arachnoid adhesions, intensifying these processes.

Symptomatic TN is caused by tumours of the cerebellopontine angle, including acoustic neuroma, which makes neuroimaging necessary [5], as well as by arachnoid cysts, aneurysms and multiple sclerosis [6]. Insufficient blood supply to the trigeminal ganglion and diabetes mellitus, in which hyperglycaemia promotes nerve demyelination, are considered additional predisposing factors, and new hypotheses of pathogenesis continue to be proposed [7].

Mechanism of pain formation

According to the gate control theory of pain developed by R. Melzack and P. Wall, fast-conducting myelinated type A fibres (antinociceptive) and unmyelinated type C fibres (nociceptive) compete in the transmission of impulses. When the trigeminal root is compressed at its entry into the brain stem, demyelination of type A fibres occurs, numerous voltage-gated sodium channels appear in the demyelinated areas, and contacts form between these areas and C fibres. As a result, prolonged high-amplitude activity of the altered A fibres arises, which is clinically manifested as paroxysmal pain.

4. Discussion

The analysed data show that TN is a multifactorial disease in which local anatomical prerequisites, inflammatory and odontogenic factors and neurovascular compression interact, while the final common pathway is demyelination of the nerve root and ectopic generation of impulses. The frequent finding of vascular contact in asymptomatic individuals [3] indicates that compression alone is not sufficient, and supports the view that additional factors, including inflammation in the maxillofacial region and the formation of arachnoid adhesions, contribute to the transition of contact into a pathological conflict. This is of practical importance for dentists and maxillofacial surgeons, since careful tooth extraction, timely treatment of pulpitis, osteomyelitis and sinusitis, and removal of root remnants may reduce the risk of TN.

The therapeutic options described in the literature reflect the variety of pathogenetic mechanisms. Anticonvulsants remain the basis of conservative treatment, but carbamazepine carries a risk of serious allergic cutaneous reactions [8], which has stimulated the study of alternatives such as levetiracetam [9] and sumatriptan [10]. Injection of botulinum toxin type A into trigger zones has been used to control pain [11], and in drug-resistant cases gamma knife radiosurgery provides pain relief [12, 13]. Homeopathic treatment [14] and acupoint injection of vitamin B12 [15] have also been described, but the evidence for these methods is limited to observational studies.

This article is a literature review and has the corresponding limitations: the analysed sources differ in design and sample size, and most of them were published before 2010, so the conclusions require confirmation by more recent clinical and morphological studies.

5. Conclusion

Trigeminal neuralgia develops as a result of the interaction of anatomical prerequisites (narrow bony canals and close contact of the nerve root with vessels), inflammatory and odontogenic processes of the maxillofacial region, and neurovascular compression, which lead to demyelination of type A fibres and the formation of ectopic foci of excitation manifested by paroxysmal pain. Prevention and timely treatment of inflammatory diseases of the maxillofacial region should therefore be considered part of the prevention of TN, and the limited effectiveness and side effects of existing treatment justify further research on pathogenetically oriented therapy.

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