

Features of the Trigeminal Nerve in Inflammatory Diseases of the Maxillofacial Region

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Abstract: Classical trigeminal neuralgia (TN) is a severe paroxysmal pain syndrome that exhausts patients mentally and physically and significantly limits their working and personal lives. The aim of this article is to analyse current concepts of the pathogenesis, clinical features, diagnosis and treatment of classical TN, with emphasis on the neurovascular conflict and the morphofunctional changes of the trigeminal nerve root. 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 key pathogenetic mechanism is a neurovascular conflict in which an arterial loop crossing the nerve root causes prolonged intermittent compression and focal demyelination, whereas simple contact of a vessel with the root, frequently found in asymptomatic individuals, does not. The anatomical variant of the conflict influences the clinical picture, and neuroimaging is necessary in all patients to exclude symptomatic forms. Conservative treatment should target the trigger zones, remyelination and central sensitisation, while microvascular decompression is indicated when drug therapy fails.

Keywords: trigeminal neuralgia, trigeminal nerve root, neurovascular conflict, demyelination, microvascular decompression, maxillofacial region.

Introduction

Trigeminal neuralgia is one of the most severe pain syndromes. Its painful paroxysms exhaust patients mentally and physically, significantly limiting their working and personal lives. According to the World Health Organization, the incidence of TN ranges from 3 to 5 new cases per 100,000 population per year. The International Classification of Diseases (ICD-10) distinguishes classical and symptomatic TN.

Neurosurgical observations revealed compression of the trigeminal nerve root by tumours, aneurysms, angiomas, arteries and veins in almost 60% of patients with TN, and compression of the root entry zone near the pons by the cerebellar arteries became the basis of the theory of neurovascular conflict as the cause of paroxysmal facial pain. This concept was later developed through microvascular decompression of the root, and the number of its supporters has grown. Nevertheless, several fundamental questions remain unanswered: why vascular contact with the root is found on MRI and at autopsy in people who have never had TN [1]; why pain is localised mainly in the territories of the second and third branches; why TN is more common on the right; and why it predominantly affects middle-aged and elderly people. The features of clinical manifestations depending on the anatomical variant of the neurovascular conflict have also not been sufficiently described.

The aim of this article is to analyse current concepts of the pathogenesis, clinical features, diagnosis and treatment of classical TN, with emphasis on the neurovascular conflict and the morphofunctional changes of the trigeminal nerve root.

Materials and Methods

The study was conducted as an analytical review of foreign and Russian-language publications from 2000–2010 devoted to the pathogenesis and morphology of TN, neuroimaging of neurovascular compression, symptomatic forms of the disease, 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 five areas: neurovascular conflict and contact, anatomical variants of the conflict, morphological changes of the root, symptomatic forms, and diagnostic criteria.

Results

Neurovascular conflict and neurovascular contact

As a result of neurovascular compression, mechanical damage occurs to the trigeminal afferents at the level of the axons of the first neuron, which form the sensory portion of the root. The literature proposes a distinction between neurovascular conflict and neurovascular contact: a conflict arises when the apex of a loop of the superior or anterior inferior cerebellar artery crosses the root in the vertical plane and causes prolonged intermittent compression with demyelination, whereas contact of the artery with the root without such crossing does not cause a conflict. MRI with volumetric reconstruction is widely used to detect compression and to evaluate its sites [2]; however, contact between the root and adjacent vessels is often found in volunteers without TN and is regarded as a variant of the norm.

Anatomical variants of the conflict and their clinical significance

Several explanations have been proposed for the unresolved clinical questions. The predominant involvement of the second and third branches is explained by the shorter length of the axons forming these branches compared with those of the first branch, under the same traumatic force of the arterial loop. The predominance of right-sided pain is attributed to the higher position of the apex of the right petrous pyramid, which brings the root closer to the pontomesencephalic segment of the superior cerebellar artery, especially in its dolichoectasia. The predominance of TN in middle-aged and elderly people is linked to the reduced elasticity of the arterial wall at the site of the conflict.

The anatomical variant of the conflict also influences the clinical picture. When one artery compresses the root from the ventral side and another from the dorsal side (an “arterio-arterial sandwich”), a neuralgic status often develops. When the root is compressed by an artery ventrally and its dorsal displacement is limited by an adjacent vein (an “arterio-venous sandwich”), the frequency and intensity of paroxysms increase in the horizontal position, and attacks appear at night. Conflict with a dolichoectatic basilar artery may add signs of trigeminal neuropathy to the clinical picture.

Morphological changes of the nerve root

Morphological examination of the root in patients with TN reveals foci of densely packed axial cylinders devoid of myelin, with axolemmal contact over a considerable length, which may underlie the ephaptic transmission of impulses from myelinated sensory fibres to unmyelinated nociceptive C fibres. Ultrastructural and immunohistochemical changes of the peripheral myelinated axons have also been demonstrated in patients with neuralgia [3]. Demyelination at the site of conflict, and its absence

in simple contact, is therefore considered the morphological basis of the disease.

Symptomatic forms

In symptomatic TN, the neurovascular conflict also plays a role, but additional mechanisms are involved: in tumours of the posterior cranial fossa, including acoustic neuroma, the tumour itself acts mechanically on the root [4], and in multiple sclerosis, focal demyelination results from the underlying disease [5]. New hypotheses of the pathogenesis of TN continue to be proposed [6].

Diagnostic criteria

The diagnosis of classical TN is based on the following criteria: the paroxysmal nature of the pain, an attack duration of no more than 3 minutes, the presence of trigger zones, an absolute refractory period, and a response to carbamazepine in the initial period of the disease. Since the clinical picture of symptomatic TN may not differ from that of classical TN, neuroimaging is necessary in all patients. To identify the neurovascular conflict and clarify its anatomical features, spiral computed tomographic angiography with three-dimensional reconstruction is recommended.

Discussion

The analysed data show that the presence of a vessel near the trigeminal root is not in itself pathological, and that only a conflict with specific anatomical characteristics leads to demyelination and the formation of an ectopic focus of excitation. This resolves the contradiction between the high frequency of vascular contact in asymptomatic people [1] and the vascular theory of TN. For maxillofacial practice, this means that patients with facial pain following dental and inflammatory diseases require careful differential diagnosis and neuroimaging, since odontogenic and inflammatory factors may coexist with or mask a neurovascular conflict.

Treatment of TN includes conservative and surgical methods. The pathogenetic basis of conservative therapy is influence on increased excitability of peripheral nociceptors, demyelination, ephaptic spread of impulses and dysfunction of the suprasegmental parts of the trigeminal system. Such therapy shortens the time to remission, although it remains palliative. Carbamazepine is the drug of first choice but carries a risk of serious allergic cutaneous reactions [7], which has stimulated the study of levetiracetam [8] and sumatriptan [9]. Botulinum toxin type A injected into trigger zones has been used to control pain [10], while homeopathic treatment [11] and acupoint injection of vitamin B12 [12] are supported only by observational studies.

Surgical methods are divided into destructive interventions, including gamma knife radiosurgery [13, 14], and the physiological method of microvascular decompression of the root. Recurrence after decompression may be caused by veins [15], which supports the importance of assessing all vessels in contact with the root before surgery. Microvascular decompression is also indicated in patients with recurrence after destructive interventions. This review is limited by the predominance of sources published before 2010 and by the different designs of the analysed studies.

Conclusion

The pathogenetic basis of classical trigeminal neuralgia is a neurovascular conflict in which an arterial loop crossing the nerve root causes prolonged intermittent compression and focal demyelination, and the anatomical variant of the conflict determines the clinical features of the disease. Conservative therapy should target three main components of pathogenesis: inhibition of the trigger zones,

stimulation of remyelination at the site of the conflict, and reduction of synaptic transmission in the trigeminal nucleus. If complete remission is not achieved within 3–4 months of drug treatment and the conflict is confirmed by spiral computed tomographic angiography, microvascular decompression of the trigeminal root is recommended.

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