



**ASSESSMENT OF THE ROLE OF ENT PATHOLOGY IN THE DEVELOPMENT OF
FACIAL PAIN**

Boymamatova Parvina Furkatjonovna
Samarkand State, Medical Institute, Assistant

Samieva Gulnoza Ukurova
Samarkand State, Medical Institute, Associate Professor
E-mail: samg83@rambler.ru

Annotation

This article describes the pathology of facial pain, as well as a comparative study of domestic and European scientists. Important points in the development of facial pain, a number of authors consider chronic inflammatory changes in the peripheral branches of the trigeminal nerve and development as a result of inflammatory processes in the paranasal sinuses. Against the background of inflammation of the mucous membrane of the sinuses, the nature of the pain syndrome is determined by a complex system of sensitive innervation of the paranasal sinuses: both peripheral receptors and fibers of the trigeminal nerve in the nasal mucosa, and receptors and fibers of the trigeminal nerve in the walls of the vessels supplying these sinuses, as well as sensitive fibers of the dura and pia mater.

Keywords: prosopalgia, headaches, maxillary sinus pain, paroxysmal trigeminal pain, Charlain syndrome, sinusitis, algogenic system, tonzilloectomy, cuneiform sinus mucocele.

Introduction

The problems of headache, facial pain are discussed among medical scientists around the world and, despite significant progress, the issue of studying pain and combating it remains largely unresolved.

Currently, the literature has accumulated a huge number of publications on various issues of facial pain. A review of most publications on this problem is presented in the works of many authors. They emphasize the diversity of forms of prosopalgia, the polyetiology of pain phenomena and the complexity of pathogenesis [8, 11, 12, 14, 19, 22, 32].

Unfortunately, the main issues of etiology, pathogenesis, clinics of facial pain still cause controversy among scientists and practitioners.

There are different opinions about the causes of facial pain in the literature: some authors assign the main role in the development of prosopalgia to general diseases, others to local factors that affect various parts of the nervous system, and others are supporters of polyetiological genesis.

Among the authors who believe that the main role in the development of facial pain belongs to local factors, there are disagreements regarding the location of the lesion of the trigeminal nerve system. So, some express the opinion that diseases of the dentoalveolar system are leading in the genesis of neuralgia. A number of authors [2, 6, 11] consider various odontogenic inflammatory diseases to be



specific causes of neuralgia. Others [27, 30, 31] do not agree with the odontogenic theory of the origin of prosopalgia.

There is less controversy about the role of chronic inflammation of the paranasal sinuses and other ENT organs in the development of facial pain.

The issues of the relationship between diseases of the ENT organs and various diseases of the nervous system have been given much attention by domestic and foreign researchers [19, 25, 28, 30]. However, they were limited to mentioning or briefly describing individual prosopalgias associated with ENT pathology.

At the end of the 90s, there was again an increase in the interest of individual researchers in the issue of the relationship between ENT pathology and facial pain. According to the literature, pain in the head and face can occur in clinical practice as a result of diseases of the nose and paranasal sinuses, ear, throat, and therefore this issue deserves separate consideration [5, 12, 16, 17, 21, 23, 27, 29, 31]. Some works cover this topic in more detail [11, 14, 20 and 29].

However, the problem of the relationship between diseases of the upper respiratory tract and facial pain has so far remained little studied, and the frequency of their occurrence remains unclear. Neither in the otorhinolaryngological nor in the neurological literature have we found works in which an attempt would be made to resolve this issue on the basis of a large, strictly selected material.

Facial pain of various localization can be caused by diseases of the nose, ear, throat of an inflammatory nature, tumors, features of the anatomical structure of the ENT organs, their traumatic injuries or surgical interventions on the ENT organs, and also be psychogenic in nature.

An indication of acute and chronic inflammation of the ENT organs in patients with facial pain gives grounds to discuss the possible role of ENT pathology in the origin of prosopalgia.

Important points in the development of facial pain, a number of authors consider chronic inflammatory changes in the peripheral branches of the trigeminal nerve [10, 17]. These changes, in their opinion, can develop as a result of inflammatory processes in the paranasal sinuses. Against the background of inflammation of the mucous membrane of the sinuses, the nature of the pain syndrome [27] is determined by a complex system of sensitive innervation of the paranasal sinuses: both peripheral receptors and fibers of the trigeminal nerve in the nasal mucosa and receptors and fibers of the trigeminal nerve in the walls of the vessels supplying these sinuses are irritated, as well as sensitive fibers of the dura and pia mater.

Some authors [1,4] observed swelling of the nasal mucosa in 22 patients with essential neuralgia of the Vth nerve; with an increase in attacks of neuralgia in the nasal mucosa, signs of secondary catarrh were detected, which were sometimes regarded as phenomena of catarrhal sinusitis, especially since with X-ray examination in some cases revealed blackout of one or another sinus. In some cases, the pain syndrome turned out to be longer than the sinusitis that caused it. The authors [5,7,9] believed that the maxillary nerve is often affected in sinusitis, and the mandibular nerve is often affected in various dental diseases (caries, alveolar pyorrhea, cyst, etc.) [10], after conducting a clinical and radiological analysis of 145 patients with trigeminal neuralgia, revealed in 34 patients the defeat of the trigeminal nerve on the side of the affected maxillary sinus.



M. A. Vishnyakova in X-ray examination of 104 patients with trigeminal neuralgia in 53 found pathological changes in the maxillary sinus in the form of parietal thickening or its homogeneous darkening, which she regards as confirmation of the value of sinusitis as an etiological factor of trigeminal neuralgia. Similar data are given by other authors [15,12] – X-ray examination of 18 patients in 5 of them revealed changes of varying degrees of damage to the maxillary sinuses.

Some authors [14, 20] note a large proportion of local inflammatory diseases (primarily sinusitis and dental disease) in patients under the age of 50 years; and maxillary sinuses. Characteristically, the localization of this pathology corresponded to the side and branch of the affected trigeminal nerve. This gave grounds to establish a causal relationship between them.

There are indications of the possibility of developing attacks of typical trigeminal neuralgia in sinusitis [21,23,24]. Cases [25, 31] of persistent headaches, proceeding according to the type of true trigeminal neuralgia, are described in patients with identified retention cysts of the maxillary sinuses (especially its upper orbital wall).

In turn, long-term trigeminal neuralgia can adversely affect the state of the ENT organs. In the literature, there are data [18] on the violation of the trophic processes of the nasal cavity as a result of prolonged trigeminal neuralgia, leading to malnutrition and secretion of nasal tissues. In these cases, the patients found x-ray changes in the maxillary sinus in the absence of anamnestic data and corresponding clinical manifestations.

Particular attention is paid to the role of the pathology of the ENT organs in the occurrence of neuralgia of the nasociliary nerve and neuralgia of the pterygopalatine ganglion. Thus, the causes of neuralgia of the nasociliary nerve (Charlen's syndrome) can be hypertrophied of the turbinates, deviated nasal septum, sinusitis, odontogenic diseases, nerve compression due to swelling of the nasal mucosa, and facial injuries [8, 11, 20, 27].

A syndrome of damage to the anterior ethmoid nerve has been described [10,19], which in most cases is caused by an inflammatory process in the cells of the ethmoid bone and in the region of the inferior turbinate of the nasal cavity.

Charlene's syndrome is considered similar to neuralgia of the pterygopalatine ganglion and these pain syndromes are referred to as facial sympathalgia. A. M. Vein [4] emphasizes the diagnostic value of relieving pain when lubricating with a 5% solution of cocaine the exit area of this nerve above the superior nasal concha.

L. B. Litvak [23] describes sphenoiditis with neuralgia of the pterygopalatine node, pain in the temple, eyes, and sinusitis with secondary trigeminal pain in the head.

At the same time, there are observations [17] concerning patients who had a history of sinusitis, but during sinus surgery, microscopic examination of the sinus node and mucous membrane did not reveal signs of inflammation.

According to foreign authors [26], any local pathological changes in the ENT organs or the dentition are more likely to play the role of a provoking factor, rather than being the cause of the disease.

Co-authors [1], analyzing cases of a combination of pathology of ENT organs and pterygopalatine ganglionitis, note that not just summation occurs, but a qualitatively new pathological condition arises



with severe symptoms due to the aggravation of one disease by another. A vicious circle appears the inflammatory process exacerbates the course of ganglionitis, which, by changing the trophism of tissues, contributes to the transition of inflammation into a protracted, latent form.

As an etiological factor in the development of neuralgic syndromes [11], inflammatory and sclerotic processes in the palatine tonsils are indicated.

Angina, chronic tonsillitis, tonsillectomy can be the cause, in addition to neuralgia of the pterygopalatine ganglion and neuralgia of the glossopharyngeal nerve (Sicard's syndrome), neuralgia of the lingual, upper laryngeal - branch of the vagus nerve, neuralgia of the auricular nerve [9, 11, 24, 27].

It is suggested [21] that the etiological factors of trigeminal neuralgia can also be considered etiological factors of the Sicard syndrome. The causes of neuralgia of the glossopharyngeal nerve are chronic tonsillitis, tonsillectomy, hypertrophy of the styloid process, tumors of the cerebellopontine angle, aneurysm of the carotid artery, and cancer of the larynx [25].

In clinical practice, craniofacial neuropathies (neuritis or symptomatic neuropathies) account for a large proportion. The cause of trigeminal neuritis, according to other authors [11, 14], is nerve compression due to inflammation of the tissues surrounding the nerve, ischemia, intoxication, according to other authors [20, 85], extracranial processes (diseases of the teeth, jaws, nose and paranasal sinuses), intracranial processes (injuries to the base of the skull, tumors and aneurysms, multiple sclerosis).

A significant role in the etiology of trigeminal neuritis is given to chronic inflammatory diseases of the paranasal sinuses, as well as surgical interventions on them [8, 11, 14].

To date, there have been discussions in the literature about the pathogenesis of facial pain. The most studied in this regard is trigeminal neuralgia.

In recent years, with the help of special techniques, it was possible to show the presence of various stages of degeneration of axial cylinders [2,9,16]. These changes are found in all cases of the disease already in the early stages and increase with its duration.

The data obtained allow us to consider that the primary link in pathogenesis is, as a rule, the defeat of its peripheral segment. This position is confirmed by the well-known fact - switching off the affected branch leads to the cessation of pain attacks until the nerve regenerates.

Noteworthy are studies [18, 23], which indicate an irritative process that occurs at any level of the trigeminal nerve and is a source of constant subthreshold impulses. Against their background, according to the law of summation of irritation, a dominant focus can form, periodically leading to explosions of excitation. Persistent foci of excitation are formed in the cerebral cortex and thalamus under the influence of a long-term influx of pain impulses [17, 23].

However, the most logical is the point of view of researchers who explain the paroxysmal nature of trigeminal pain by a multineuronal reflex involving several levels of the nervous system [3, 11,]. Simulation of mental nerve compression in animals is accompanied by the appearance of synchronization in the subcortical structures and the surface electroencephalogram, followed by the appearance of spike activity.



These factors ultimately made it possible to substantiate the concept that under the influence of prolonged subthreshold stimulation from the periphery, an algogenic system is created in the brain, which is stable, highly excitable, and responds to any afferent messages with paroxysmal-type excitation [15, 24]. Factors that cause prolonged subthreshold impulses from the periphery are narrowing of the infraorbital canal, cysts or tumors of the maxillary sinus, frontal sinus osteomas, as well as local chronic inflammatory processes that, irritating the peripheral formations of the trigeminal nerve for a long time, contribute to the development of trigeminal neuralgia.

These changes in the periphery are long-acting factors, and trigeminal neuralgia is manifested by short-term paroxysms.

The central link of the algogenic system is the oral part of the spinal nucleus of the trigeminal nerve with its numerous connections, including the reticular formation of the brain stem, cerebellar nuclei, and other structures [15].

Complex etiological, pathogenetic and symptomatic treatment of facial pain in most cases is a difficult problem.

A number of etiological factors of prosopalgia - infectious, toxic, metabolic, compression or traumatic nature - are difficult to eliminate or require long-term conservative, and in some cases surgical treatment (tunnel and compression syndromes of the trigeminal and glossopharyngeal nerves).

Complex treatment should be pathogenetic. It is necessary to carry out etiotropic therapy aimed at preventing further progression of neuropathy, restorative treatment that favors the regression of structural changes in the nerve trunks, and the treatment of the pain syndrome itself. At the same time, many components of this therapy that block nerves and humoral pain factors are essentially etiotropic, as they contribute to the relief of inflammation, edema, and the release of flotation substances [12, 15]. For the treatment of prosopalgia, especially trigeminal neuralgia, the specific action of the drug carbamazepine (tegreol, finlepsin, stazepin) is used; other antiepileptic drugs (trimethine, suxilep, clonazepam, dipropyl acetate) are used to reduce the action of carbamazepine, but they are, to varying degrees, less effective or more toxic [7,15,28,31].

Narcotic analgesics (acetylsalicylic acid, antipyrine, phenacetin, indomethacin) are effective due to their analgesic, anti-inflammatory, kininoblocking, antiprostaglandin action, due to the fact that they improve microcirculation, especially with prolonged prosopalgia. For the treatment of prosopalgia, accompanied by vegetative-vascular disorders, ganglioblocking, antispasmodic or vascular tonic agents are used (anaprilin, phentolamine, tropafen, caffetamine, dihydroergotamine).

In the complex treatment of prosopalgia, a certain place is occupied by blockades of reflexogenic zones, local use of distracting and analgesic drugs in the form of ointments, lubrication of mucous membranes, aerosol irrigation or injections.

Of particular note is the method that is most widely used in the diagnosis and treatment of patients with prosopalgia: lubrication with a 5% solution of cocaine or 2% dicaine solution of the mucous membrane of the posterior sections of the middle nasal passage in Slader's syndrome, the anterior sections of the upper nasal passage in Charlene's syndrome; lubrication of the root of the tongue, the palatine curtain, the lateral wall of the pharynx with Sicard's syndrome. These procedures stop the onset



of an attack of pain or significantly alleviate it. Repeated daily lubrication according to this technique is included in the treatment complex for these types of prosopalgia, along with the use of neuroleptics and antidepressants.

To date, the question of the legality of the use of the method of alcoholization has not been resolved. With the failure of conservative treatment, the question of the possibility of surgical intervention is raised.

The use of intracranial neurosurgical methods of treatment is justified in patients who are not helped by all other methods of treatment [9, 22]. However, they are more technically complex and fraught with complications [56]. Therefore, in the treatment of trigeminal neuralgia, it is first necessary to perform interventions on the peripheral branches of the trigeminal nerve [5, 22], however, the existing methods of such intervention [5, 28] do not prevent relapses, the treatment of which is also an unresolved issue. Summarizing the literature data, it should be noted that a number of authors have reliable data on the role of ENT pathology in the etiology of facial pain [8, 11, 14, 17,27]. However, the question of the relationship between ENT pathology and prosopalgia in both otorhinolaryngology and neurology has not yet been sufficiently studied, clinical observations and experience have not been systematized, and the necessary conclusions have not been drawn.

There are almost no works in the literature in which attempts would be made to resolve the question: in which cases the pathological process of the ENT organs causes pain, and in which cases it does not. Until now, clinicians do not have a method for an objective assessment of the pain syndrome, which makes it difficult to monitor the course of the disease.

Thus, the problem of studying facial pain and identifying the significance of ENT pathology in the genesis of prosopalgia is relevant, and the question of the participation of an otorhinolaryngologist in the diagnosis and treatment of patients with prosopalgia also needs to be addressed.

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