

Modern Perceptions on the Diagnosis and Treatment of Face Pain Syndromes

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Abstract: Pain in the facial area (prosopalgia) is a complex, polyetiological, and clinically diverse group of pathological conditions, including nociceptive, neuropathic, and psychogenic forms. Among them, atypical facial pain occupies a special place, the pathogenesis of which is associated with the disruption of the central mechanisms of pain modulation and psycho-emotional disorders. The review presents modern data on the pathogenesis, classification, diagnosis, and treatment of various forms of facial pain, including odontogenic, myogenic, neuropathic, and psychogenic syndromes. Special attention was paid to the diagnostic difficulties in persistent idiopathic facial pain (PIFP) and the need for an interdisciplinary approach in therapy. Despite significant progress in the field of neurophysiology of pain, the pathogenetic mechanisms of this condition remain insufficiently studied, which necessitates further research aimed at identifying biomarker central sensitization and developing personalized treatment strategies.

Keywords: facial pain, atypical prosopalgia, neuropathic pain, psychogenic pain, diagnostics, cognitive behavioral therapy.

Introduction. Facial pain syndromes represent a clinically and pathophysiologically heterogeneous group of conditions encompassing nociceptive, neuropathic, myogenic, and psychogenic components. Despite significant progress in understanding pain mechanisms over recent decades, diagnostic and therapeutic complexities persist, particularly regarding persistent idiopathic and mixed forms of facial pain (PIFP - persistent idiopathic facial pain, formerly termed "atypical facial pain") [1,6]. Misinterpretation of facial pain causes frequently leads to unwarranted dental or surgical interventions, prolonged patient suffering, and increased economic burden on healthcare systems [11,18]. The purpose of this review is to systematize current data on classification, pathogenesis, clinical presentation, and multidisciplinary approaches to diagnosis and treatment of facial pain syndromes, as well as to highlight promising research directions and clinical practice developments.

Epidemiology and Clinical Significance

The prevalence of facial pain in the population varies depending on research criteria and methodology; according to epidemiological studies, the frequency of chronic facial pain syndromes is estimated at approximately 10-26% in the general population when considering various causes, while the proportion of persistent idiopathic facial pain (PIFP) among all facial pain cases constitutes a smaller but clinically significant fraction [1,6,11]. PIFP is more commonly detected in middle-aged women; chronic progression is frequently accompanied by deterioration in quality of life, depression, and somatoform disorders [10,16].

Classification

For diagnostic uniformity, it is recommended to use the international ICHD-3 classification (International Classification of Headache Disorders, 3rd ed.) and specialized classifications in the field of orofacial pain. In practice, four major nosological groups are distinguished:

Nociceptive (odontogenic, somatic) pain - inflammatory and structural lesions of teeth, periodontium, TMJ, oral mucosa.

Neuropathic pain - trigeminal neuralgia (classical/secondary), post-traumatic and postoperative neuropathies, persistent dental-alveolar pain syndromes (PIDAP/PDAP).

Myogenic/myofascial pain - masticatory muscle dysfunction, trigger points, bruxism, etc.

Psychogenic/somatoform and idiopathic pain (PIFP) - pain without detectable structural causes, often with prominent psychoemotional components [6,12,14].

Pathogenesis: Key Mechanisms and Interactions

Pathogenetically, facial pain syndromes result from the interaction of peripheral and central mechanisms:

Peripheral sensitization develops with odontogenic inflammation, nerve trauma, or prolonged nociceptor activation; excitability of primary afferent fibers increases, ectopic impulse generators arise [22,12].

- Central sensitization - enhancement of neural excitability in trigeminal nuclei and central structures due to prolonged nociceptive input; formation of pathological pain transmission and altered pain signal modulation [6,15].
- Neuroimmune mechanisms: microglial and astrocytic activation, proinflammatory mediator release contributes to pain chronification and reduced analgesic effectiveness.
- Psychoneurological factors: depression, anxiety, and chronic stress alter antinociceptive system tone (serotonergic and noradrenergic), intensifying subjective pain perception and maintaining chronic status [1,10].

This complex of processes explains why the same peripheral trauma may lead to rapid recovery in some patients while resulting in prolonged disabling pain in others.

Clinical Presentation: Main Syndromes and Characteristic Features

Odontogenic and Somatic Pain

Odontogenic pain typically has clear topography and irradiation corresponding to the affected tooth/periodontium; however, referred pains and incorrect localization are possible, requiring careful differentiation [22,17]. Often patients with referred odontogenic pain undergo multiple dental consultations and receive unnecessary interventions due to symptom misinterpretation [11].

Trigeminal neuralgia (TN) is characterized by brief, paroxysmal, "electric" pain attacks, usually provoked by tactile stimulation of the trigeminal zone; these paroxysms are accompanied by refractory periods and characteristic muscular reactions [15]. Secondary TN forms are associated with vascular compression, tumor processes, or demyelinating diseases (e.g., multiple sclerosis). Diagnostic indicators include clear trigger presence and response to carbamazepine/oxcarbazepine.

Post-traumatic neuropathies and persistent dental-alveolar pains (PDAP/PIDAP) are characterized by constant pressing or burning pain, paresthesias, and absence of structural pathology on imaging; pathogenetically similar to neuropathic pain and often poorly responsive to conventional analgesics [12,18].

The myofascial component manifests as trigger points in masticatory muscles, persistent tone elevation, and referred irradiation to ear, temporal area, and neck. These conditions are closely related to occlusal dysfunction, bruxism, and stress [14,2].

PIFP is a diagnosis of exclusion, characterized by prolonged, poorly localized, often migrating facial pain without objective neurological or dental findings. Often accompanied by facial "numbness," "swelling" sensations, anxious and depressive symptoms; treatment is challenging and requires a multidisciplinary approach [1,6,10].

Diagnosis: Principles and Algorithm

The diagnostic process should be systematic and staged:

Thorough history - pain characteristics (type, intensity, dynamics, trigger factors), relationship to dental interventions or trauma, accompanying symptoms (paresthesias, sensory reduction).

Physical examination - oral cavity inspection, TMJ assessment, muscle tone evaluation, neurological examination of facial area sensitivity and reflexes.

Strategic application of instrumental methods:

Orthopantomogram (OPG), targeted radiography, CT - excluding odontogenic or bone pathology;

Brain/craniofacial MRI - when suspecting trigeminal nerve vascular compression, tumors, orbital or cavernous sinus diseases;

Electroneurography, quantitative sensory testing (QST) - when suspecting neuropathic pathology;

Psychiatric/psychological assessment - depression and anxiety scales, coping strategy analysis; early intervention when disorders are identified.

Differential diagnosis - excluding migraine and other primary headaches, temporomandibular disorders, sinusitis, neurological and dental pathologies [6,11,22].

The recommended algorithm is "organic pathology exclusion - neural structure assessment - psychoemotional status evaluation" with appropriate specialist involvement.

Therapy: Contemporary Approaches and Treatment Tactics

General Principles

Treatment should be multicomponent, effective, and minimally invasive. Individual therapy selection, early multidisciplinary coordination, and avoidance of unwarranted surgical or dental interventions without indications are important [11,23].

Pharmacotherapy

- Antidepressants (tricyclics: amitriptyline; selective or serotonin-noradrenergic: duloxetine) - effective for neuropathic and psychogenic pains through central analgesia enhancement and mood normalization [1,6].
- Anticonvulsants (carbamazepine, oxcarbazepine for classical TN; gabapentin, pregabalin for neuropathic and PIFP-like conditions) - reduce neuronal hyperexcitability [15,12].
- Local methods: topical lidocaine preparations, capsaicin (under expert supervision), local anesthetic injections for diagnostic blocks.
- Botulinum therapy (botulinum toxin type A) - several clinical studies demonstrated effectiveness for trigeminal neuralgia and certain PIFP/myofascial pain forms, providing temporary intensity and frequency reduction of pain paroxysms; however, the method requires dose standardization and precise injection techniques [7,1,24].
- Novel biological approaches - research on neuroproteins and neuroimmune inflammation targets (e.g., S100-antibody studies and other targets) is in early clinical investigation stages, with no definitive recommendations yet [5].

Non-pharmacological and Non-medical Methods

- Physiotherapy: manual therapy, myofascial release, therapeutic exercise - indicated for myogenic pain and as adjunctive methods for mixed syndromes [14,25].
- Transcranial magnetic stimulation (TMS) - applied as non-pharmacological option for resistant chronic pains, with evidence of temporary improvement in selected patients [7].

- Biofeedback, relaxation methods, cognitive-behavioral therapy (CBT) - mandatory component of PIFP and psychogenic form treatment; CBT improves coping strategies and reduces pain's emotional component [1,10].
- Invasive neurosurgical interventions: microvascular decompression for trigeminal nerve vascular compression - indicated for classical TN; radiofrequency ablation and gamma knife - palliative local intervention options for refractory pain, requiring careful patient selection and risk discussion [15].

Combined and Staged Approach

Preference is given to staged, combined regimens: dental pathology correction (if present), pharmacotherapy (considering psychotropic drug potential), non-pharmacological methods, and psychotherapy. This integrated approach increases long-term remission chances and reduces ineffective manipulation frequency [11,14].

Problems and Research Perspectives

Current main problems: absence of universal PIFP and PDAP biomarkers, deficit of high-quality randomized studies for many therapeutic interventions (including botulinum toxin and neuromodulation), insufficient diagnostic criteria standardization in dental and general medical clinical practice [12,7]. Promising directions include:

- Development of patient stratification criteria by pain mechanisms (peripheral/central/psychogenic),
- Search for neuroimaging and neurochemical biomarkers of central sensitization,
- Multidisciplinary clinical studies of combined interventions (pharmacotherapy + CBT + physiotherapy),
- Botulinum therapy and transcranial stimulation protocol standardization for facial pains.

Practical Recommendations for Clinicians

Always begin with thorough history and odontogenic pathology exclusion - only then consider PIFP diagnosis.

Involve neurologist and, when necessary, psychiatrist/psychotherapist early, especially with prolonged or unusual pain progression.

Avoid unwarranted tooth extractions or other radical interventions without clinical and instrumental evidence of organic pathology.

Apply multimodal therapy: pharmacotherapy + non-pharmacological methods + psychotherapy.

For refractory forms, discuss neuromodulation and neurosurgery options in interdisciplinary consultation.

Conclusion

Facial pain syndromes require comprehensive, interdisciplinary approaches combining thorough differential diagnosis, rational pharmacotherapy, and non-pharmacological interventions. Persistent idiopathic facial pain remains a clinically complex nosology where the combination of neurophysiological and psychoemotional factors determines clinical strategy. Further research should focus on elucidating central sensitization mechanisms, developing biomarkers, and evidence-based personalized treatment regimens.

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