

Review Article

Oral fire, mental storm: the profound interplay between trigeminal neuralgia, burning mouth syndrome and mental health disorders

Komal Rewatkar¹, Astha Jathar^{2*}, Noopur Kokane¹, Hrishita Biswas¹

¹Department of Public Health Dentistry, Government Dental College and Hospital, Maharashtra, India

²Department of Oral and Maxillofacial Surgery, Government Dental College and Hospital, Maharashtra, India

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*Correspondence:

Dr. Astha Jathar,

E-mail: jatharastha@gmail.com

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ABSTRACT

Chronic orofacial pain conditions such as trigeminal neuralgia (TN) and burning mouth syndrome (BMS) extend well beyond physical discomfort and are increasingly recognized as disorders that affect psychological and emotional well-being. Both are frequently overlooked in dental and general medical practice, yet both produce functional limitation, social withdrawal and measurable psychiatric morbidity. This narrative review examines the links between chronic orofacial pain and mental health, with emphasis on the neurobiological and psychosocial mechanisms that connect them. It also considers the role of modifiable behavioral risk factors, particularly tobacco and nicotine exposure, which sensitize transient receptor potential channels on trigeminal and oral mucosal afferents and may aggravate oral burning and neuropathic facial pain. Peripheral sensitization drives central sensitization within trigeminal pathways and alters affective-motivational circuits, contributing to the depression, anxiety, sleep disruption and cognitive impairment reported in these patients, and creating a self-sustaining pain-distress loop. The review argues for interdisciplinary management that combines pharmacological neuromodulation, adapted dental care, psychological therapy, tobacco cessation support, nutritional correction and patient education, delivered within a preventive public health framework. Dental practitioners are well placed to detect these conditions early, to screen for tobacco use and emotional distress, and to coordinate referral. Treating the mouth and the mind together, rather than sequentially, offers the best prospect of restoring function and quality of life.

Keywords: Burning mouth syndrome, Chronic pain, Depression, Mental health, Tobacco use, Trigeminal neuralgia

INTRODUCTION

Oral health reflects general health and is also a determinant of emotional and mental well-being. Chronic orofacial pain alters the ability to eat, speak and smile, activities that are central to social identity, and converts them into distressing experiences. TN and BMS are two of the most painful and most frequently misunderstood conditions encountered at the interface of dentistry and neurology.^{1,2}

TN produces intermittent, electric shock-like pain, whereas BMS produces a persistent burning sensation in the absence of visible mucosal change.

Both conditions are now accommodated within the International Classification of Diseases (ICD-11) framework for chronic pain, which distinguishes pain as a disease in its own right from pain as a symptom of another condition.³ That distinction matters clinically, because it reframes persistent orofacial pain as a condition requiring management in its own right rather than as a signal to be traced to a dental cause.

The overlap between oral symptoms and psychiatric morbidity creates a feedback loop that erodes quality of life, and this loop is frequently missed when the two domains are assessed separately.

Objectives

The objective of this narrative review is to synthesize current evidence on the relationship between chronic orofacial pain and mental health, to examine the mechanisms that link them, to appraise the contribution of modifiable behavioral risk factors such as tobacco use, and to set out the implications for interdisciplinary clinical practice.

DISCUSSION

The literature considered here converges on a single interpretation: TN and BMS are not confined to the oral cavity but engage the same central circuits that regulate mood, sleep and cognition, and the resulting psychiatric morbidity feeds back to intensify the pain. The sections that follow examine the clinical presentation of each condition, the evidence that tobacco acts as a modifiable contributor, the mental health sequelae reported in these populations, the neurobiological basis of the association, and the management implications that follow from it.

TN: oral pain that paralyzes

TN affects the fifth cranial nerve and presents as intense, shooting pain along the face and jaw. Dental practitioners frequently encounter patients who report pain on brushing, chewing or exposure to cold air, and misdiagnosis as dentine hypersensitivity or temporomandibular disorder is common.¹ TN is classified among the most severe forms of neuropathic pain, and current European guidance recommends magnetic resonance imaging to identify neurovascular compression and to exclude secondary causes such as multiple sclerosis. The condition is more prevalent in women and in patients above 50 years of age.⁴

The unpredictability and severity of attacks provoke marked anxiety and avoidance behavior. Patients may avoid eating or brushing in order not to trigger pain, which in turn compromises oral hygiene and nutrition. The dentist therefore has a pivotal role in recognizing the symptom pattern early and in referring appropriately.

BMS: when the mouth burns without a flame

BMS is defined as a chronic intraoral burning or dysesthetic sensation occurring in the absence of visible clinical change and of an identifiable local or systemic cause.^{2,5} Patients describe burning, tingling or numbness, most often affecting the tongue, lips and palate, and xerostomia and dysgeusia frequently accompany the burning sensation. Diagnostic criteria have varied considerably across published trials, which complicates comparison between studies and may contribute to under-recognition in practice.⁵

Oral health professionals are usually the first point of contact for these patients. In the absence of observable

lesions, complaints are sometimes dismissed, which compounds the psychological burden. BMS shows a strong association with peri- and postmenopausal women, implicating hormonal, neurological and psychological factors.⁶

The role of tobacco in oral burning and neuropathic facial pain

Tobacco and nicotine exert complex, bidirectional effects on oral nociception that are relevant to both BMS and neuropathic facial pain. Nicotine acts centrally through nicotinic acetylcholine receptors and peripherally on sensory nerve endings, producing short-term antinociceptive effects alongside longer-term pronociceptive adaptation. Nicotine can sensitize transient receptor potential (TRP) channels, notably TRPV1, on trigeminal and oral mucosal afferents, increasing responsiveness to thermal and chemical stimuli and promoting release of neuropeptides such as substance P and calcitonin gene-related peptide, which amplify nociceptive signaling and local neurogenic inflammation.^{7,8} Experimental work indicates that the sensory effects of cigarette smoke are mediated through nicotinic and TRPA1 mechanisms as well, so the irritant profile of tobacco cannot be attributed to a single channel.⁹

Clinically, adverse oral sensations related to nicotine exposure are reported with smoked and smokeless products and with some oral nicotine replacement therapies (NRT). Mucosal irritation, burning and mouth ulceration occur in a substantial minority of quitters during the early withdrawal period, indicating that both ongoing nicotine exposure and abrupt withdrawal can alter oral sensory homeostasis and transiently worsen burning.¹⁰

At the circuit level, persistent peripheral sensitization can drive central sensitization within trigeminal pathways, altering thalamocortical processing and engaging emotional pain circuits including the insula, anterior cingulate cortex and amygdala. These changes contribute to the comorbidity between chronic orofacial pain and mood disorders, and create a behavioral feedback loop: nicotine transiently reduces distress through dopaminergic reward pathways, while chronic use and withdrawal destabilize dopaminergic, serotonergic and noradrenergic signaling and sustain both pain and negative affect, reinforcing tobacco use as maladaptive self-medication.^{8,11}

Taken together, the evidence supports tobacco and nicotine as modifiable contributors to BMS-like symptoms and to the worsening of orofacial neuropathic pain, through TRP channel sensitization, neurogenic inflammation and affective-motivational circuit change. Clinicians should therefore assess tobacco exposure in every patient with unexplained oral burning or trigeminal pain, counsel on possible transient worsening during

cessation, and arrange coordinated cessation support, including selection of NRT formulations that minimize oral irritation.

Mental health sequelae: oral pain, emotional storm

Chronic orofacial pain extends beyond physical suffering and affects emotional and cognitive function. Pain and depression share biological pathways and neurotransmitter systems, and their co-occurrence worsens recognition, treatment response and outcome for both conditions.¹¹ Neuropsychiatric and neuroimaging studies indicate that sustained oral pain disrupts the circuits that regulate mood, sleep and executive control, which helps to explain the high prevalence of psychiatric comorbidity in this population.

Depression

Persistent oral pain fosters helplessness and hopelessness. A systematic review and meta-analysis of psychological factors in BMS found significantly increased odds of depression among patients compared with controls (pooled odds ratio 3.18, 95% confidence interval 1.75-5.76), with anxiety similarly more prevalent.¹² Depressive symptoms are also common in TN and are associated with greater disease impact.^{13,14} Depression worsens pain perception through descending serotonergic dysfunction and reduces motivation for oral hygiene and treatment adherence.

Anxiety and anticipatory fear

The electric shock-like attacks of TN are unpredictable and generate intense anticipatory anxiety and hypervigilance.¹³ Patients modify eating, speaking and brushing habits to avoid triggers, reinforcing avoidance behavior. Cohort and Mendelian randomization data suggest that the relationship is bidirectional: longer disease duration and wider trigeminal involvement are associated with more severe depressive, anxiety and insomnia symptoms, while genetic liability to these disorders is associated with increased risk of TN.¹⁵ BMS, by contrast, produces chronic low-grade anxiety arising from a persistent, unexplained burning sensation that no visible lesion validates.¹²

Sleep disruption and fatigue

Pain often intensifies in the evening, contributing to sleep fragmentation and daytime fatigue. Sleep loss in turn heightens pain sensitivity and emotional reactivity.¹⁶

Cognitive impairment

Prolonged pain is associated with reduced working memory, attention and executive function.¹⁶ Neuroimaging in chronic orofacial pain shows structural and functional alteration in the prefrontal cortex, anterior

cingulate cortex and hippocampus, regions that are central to cognitive-emotional integration.¹⁷

Social withdrawal and isolation

Embarrassment about altered speech, halitosis or visible discomfort leads to avoidance of social interaction and to loneliness, which aggravates depressive symptoms and intensifies pain perception, completing a self-sustaining pain-depression-anxiety triad.^{13,16}

Neurobiological links between oral pain and mood disorders

The trigeminal system communicates directly with brain regions that process emotion, including the amygdala and anterior cingulate cortex, and functional imaging in trigeminal neuropathic pain and temporomandibular disorders demonstrates consistent alteration within these networks.¹⁷ Neurotransmitters involved in both pain and mood regulation, namely serotonin, dopamine and norepinephrine, are implicated in chronic pain states, and tobacco use interferes with the same systems: nicotine initially augments dopamine release, but sustained exposure alters receptor sensitivity and contributes to mood instability and dependence.^{8,11} Persistent peripheral input from oral tissues can modify central nervous system plasticity and thalamocortical relay, so that pain is perceived as more intense and more persistent even in the absence of new peripheral stimuli. This argues for early intervention in oral pain disorders, before central changes consolidate.

Oral health implications: from function to inflammation

Both conditions have direct consequences for oral hygiene and dental care. Patients with TN may avoid brushing or attending appointments for fear of triggering an attack, with resulting plaque accumulation, gingival inflammation and caries. Patients with BMS who have xerostomia and altered taste perception may neglect hygiene or consume irritant foods, worsening mucosal discomfort. Poor oral hygiene contributes to systemic inflammatory burden and to comorbidity, so maintaining hygiene matters for general health as well as for pain control.

Clinicians should assess oral hygiene status at every visit and offer adaptive strategies, including gentle cleaning methods, saliva substitutes and fluoride supplementation. Routine dental review is also an opportunity for early detection of pain-related disorders and for emotional support.

Cultural and behavioral dimensions

Oral complaints are underreported in many communities because of stigma, cultural norms or fatalism. Where tobacco chewing is culturally accepted, its contribution to

oral pain syndromes is frequently overlooked. Public health programs should address these gaps through culturally appropriate education on the links between oral habits, oral disease and mental health. Dental practitioners should be trained in brief tobacco intervention and in recognizing emotional distress, and integrating behavioral health services within dental clinics could substantially improve patient outcomes. Health education should promote oral self-care, reduce harmful practices and encourage routine review, particularly in populations at high risk of TN and BMS.

Interdisciplinary and preventive approaches

Effective management of chronic orofacial pain and its psychological sequelae requires a collaborative strategy that addresses biological, behavioral and environmental dimensions together. TN and BMS seldom respond to single-modality care, and the best outcomes arise from a coordinated model that integrates medical, dental, psychological and behavioral interventions with preventive public health measures.

Medical management

For TN, sodium channel-blocking anticonvulsants remain the cornerstone of treatment. Carbamazepine and oxcarbazepine are first-line agents, with lamotrigine, baclofen or gabapentin used as add-on options in resistant cases, and surgical approaches such as microvascular decompression, percutaneous rhizotomy or stereotactic radiosurgery considered when medical therapy fails or is not tolerated.⁴ In BMS, pharmacological neuromodulation using low-dose tricyclic antidepressants, serotonin-norepinephrine reuptake inhibitors, topical/systemic clonazepam, or alpha-lipoic acid has been used to modulate central sensitization and oxidative stress, although the evidence base remains limited and diagnostic heterogeneity across trials constrains interpretation.⁵

Dental and oral care

Dentists prevent secondary complications through gentle, non-irritating oral hygiene regimens, salivary substitutes and management of xerostomia. Soft toothbrushes, fluoride varnishes and avoidance of abrasive or strongly flavored dentifrices help to maintain oral health without triggering pain. Regular review allows early detection of mucosal change and provides opportunities for reassurance and behavioral reinforcement.

Psychological and behavioral interventions

Given the bidirectional link between chronic pain and emotional distress, psychological therapy is integral rather than adjunctive. Cognitive behavioral therapy and acceptance and commitment therapy reduce pain catastrophizing, improve coping and enhance quality of life, and coping strategies differ measurably between

patients with TN and those with temporomandibular disorders, which supports formal psychological evaluation as part of assessment.¹⁸ Group-based interventions and peer support can reduce perceived isolation and normalize patient experience.

Tobacco cessation and behavioral risk modification

Tobacco and nicotine aggravate oral mucosal irritation, alter neurochemical signaling and worsen both pain and anxiety.⁷ Brief cessation counseling, NRT and motivational interviewing can be delivered within dental settings. Patients should be counseled that oral symptoms may fluctuate transiently during withdrawal, and supportive follow-up should be arranged.¹⁰

Nutritional and lifestyle support

Deficiencies of vitamin B complex, folate, iron and zinc have been associated with oral burning and should be identified and corrected where present.⁶ Adequate hydration, avoidance of irritant foods and balanced nutrition support mucosal integrity and neuronal function. Where xerostomia is severe, saliva substitutes or sialogogues may be considered.

Patient education and empowerment

Clear, empathetic communication about the diagnosis, the expected course and self-care techniques reduces fear and uncertainty. Educational material on pain mechanisms and on stress-pain interaction enhances self-efficacy, and oral-mental health literacy modules can be incorporated into clinical and community settings.

Public health and preventive dimensions

Community oral health programs should include screening for chronic oral pain, tobacco habits and psychological distress. Intersectoral collaboration between dental, medical and mental health services can improve early diagnosis and reduce disease burden at population level.

Limitations

This is a narrative rather than a systematic review, and the included literature was selected for relevance rather than through a predefined search protocol, so selection bias cannot be excluded. Much of the evidence linking psychological morbidity to TN and BMS is cross-sectional, which limits causal inference, and the heterogeneity of diagnostic criteria for BMS across studies restricts direct comparison of prevalence estimates.

The evidence on tobacco as a contributor to oral burning is drawn largely from experimental and observational work rather than from interventional studies in BMS populations.

CONCLUSION

Oral health cannot be considered in isolation from mental health. TN and BMS illustrate how neuropathic oral pain can escalate into sustained emotional distress, and how that distress in turn intensifies the pain. When compounded by modifiable factors such as tobacco use, poor nutrition and social isolation, these conditions warrant early and holistic intervention. Dental professionals occupy a frontline position from which to recognize the symptom pattern, screen for tobacco use and psychological distress, support behavioral change and initiate referral. This review advances understanding in the field by drawing the neurobiological, behavioral and psychosocial strands of the evidence into a single clinical framework, and by identifying tobacco exposure as a modifiable target that is relevant to both the pain and the mood components of these disorders. Bridging the gap between oral and mental health does more than relieve pain; it restores function and dignity to patients' daily lives.

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