Burning mouth syndrome: review of etiopathogenetic factors and update on clinical management strategies

Fabiola Rinaldi[1]
Davide Gerardi[2]
Pierangelo Burdo[1]
Francesca Angiolani[1]
Gustavo Duarte Mendes[3]
Maurizio Piattelli[1]
Giuseppe Varvara[1]

[1] Department of Innovative Technologies in Medicine & Dentistry, Dental School, "G. D'Annunzio" University of Chieti-Pescara, Chieti, Italy
[2] Department of Life, Health and Environmental Sciences, University of L'Aquila, L'Aquila, Italy
[3] Faculdade de Odontologia da Universidade Metropolitana de Santos, Santos, Brazil

Corresponding author: Giuseppe Varvara
e-mail: varvara@unich.it

All authors contributed equally to this work.

Abstract

This study focuses on the possible etiopathogenetic mechanisms underlying burning mouth syndrome and the most appropriate clinical management for its treatment. Burning Mouth Syndrome (BMS) is characterized by an intraoral burning sensation or dysesthesia without any detectable alterations in the oral mucosa, causing significant discomfort in affected patients. The idiopathic nature of that type of chronic orofacial pain makes it challenging to identify specific etiopathogenetic factors and mechanisms. Therefore, the therapeutic strategy for BMS is challenging to choose among, including pharmacological approaches involving synthetic drugs or compounds derived from natural substances. The most appropriate clinical management for its treatment may involve different therapeutic approaches, although a definitive protocol for managing BMS has yet to be established.

Keywords Burning Mouth Syndrome, oral pain, neuropathic pain.

Introduction

Burning Mouth Syndrome (BMS) is a chronic condition characterized by a burning sensation in the oral cavity or dysesthesia without any detectable alteration of the oral mucosa. It is often associated with dysgeusia and xerostomia (1). Because a recognizable cause is impossible to identify, it is also known as chronic idiopathic orofacial pain (COFP) (2).
Chronic idiopathic orofacial pain represents a heterogeneous group of disorders characterized by typical symptoms, such as spontaneous, continuous, or intermittent pain, stabbing, and/or burning, with various types of sensory alterations (3). They can lead to dysfunctions in multiple systems: musculoskeletal, vascular, neurovascular, neuropathic, idiopathic, and psychogenic (4).
Assessing and managing patients with BMS poses significant challenges for clinicians because its underlying mechanisms are poorly understood, and patients often exhibit inconsistent responses to various treatments. Furthermore, the rate of spontaneous remission is remarkably low, at only 3-4% after 5-6 years from the diagnosis (5). There are currently no worldwide guidelines for the treatment of BMS, and existing review articles have included clinical studies with limited follow-up periods (<2 months) (6).

How to Cite

F. Rinaldi, D. Gerardi, P. Burdo, F. Angiolani, G. Duarte Mendes, M. Piattelli, G. Varvara.
Burning mouth syndrome: review of etiopathogenetic factors and update on clinical management strategies.
Oral and Implantology
Vol. 17 No. 1 (2025), 35-43.
DOI: 10.11138/oi.v17i1.118

Burning Mouth Syndrome is one of the less common neuropathic/idiopathic COFP disorders. It affects a small percentage of the general population, with a higher prevalence in women, especially in postmenopausal age (7). Its significant impact on patients' quality of life and stomatognathic functions requires investigating the possible underlying causes of BMS to expedite its diagnosis and, above all, provide new treatment perspectives.

This narrative and comprehensive review includes a structured search strategy and protocol to select the most relevant items from the literature, ensuring the information provided is reliable and trustworthy.

This review aimed to explore the potential etiopathogenetic mechanisms underlying BMS and all possible treatment options for its management.

The search strategy used the Pubmed, Web of Science, and Scopus databases. The search strategy was conducted using the following search terms: "Burning Mouth Syndrome," "BMS," "Stomatodynia," "Oral Pain," "Neuropathic Pain," "Oral Cavity," "BMS," "Diagnosis," and "Treatment." Studies available in the English language were considered. Clinical studies, reviews and systematic reviews, case series, and case reports were included in the present review. Conference proceedings, abstracts in journals, and articles published in a language other than English were excluded.

The chosen articles were arranged in a structured document that included the authors' names, publication year, type of study, and key information and conclusions presented. Therefore, the collected data were organized into "General Pathogenesis of BMS," "Diagnosis," and "Management of Patients Affected by BMS."

General Pathogenesis of BMS

Despite numerous studies, BMS remains a condition with many unresolved aspects. Initially, BMS was classified as psychogenic pain, but the current evidence shows its etiology is multifactorial, and current evidence increasingly supports the neuropathic nature of the disease (8).

Burning Mouth Syndrome could represent a convergent clinical presentation of dysfunctions in the peripheral, central, or both nervous systems. Most cases of BMS may be attributed to a small fiber peripheral neuropathy in the intraoral mucosal epithelium. Immunostaining of tongue biopsies showed increased expression of TRPV1 (Transient Receptor Potential cation channel subfamily V member 1) channels, which are involved in temperature perception and nociception, suggesting that a higher number of TRPV1 receptors could lead to an increased sensation of pain. Transient Receptor Potential channels are essential in sensitization and nociception (9).

Another subset of BMS could constitute a subclinical trigeminal neuropathy, a theory based on abnormalities of the masseter reflexes and the blink reflex, commonly evaluated in examining trigeminal nerve function, observed in about 20% of patients with BMS. The rest of the patients might experience BMS as centrally mediated pain, possibly related to the hypofunction of dopaminergic neurons in the basal ganglia, which are involved in the inhibitory modulation of pain. The alterations in this system are similar to those observed in Parkinson's disease, and there is some evidence of an increased incidence of BMS in patients with Parkinson's disease (9).

Neurophysiological and imaging studies suggest a role of nigrostriatal dopaminergic system dysfunction in the pathophysiology of BMS: similar neurotransmitter alterations are found in psychiatric disorders such as major depression; dopamine, involved in the nigrostriatal and mesolimbic pathways, can be modulated by a positive psychological state (10). BMS's psychological vs. organic pathogenesis has been debated, but molecular and imaging techniques reveal abnormalities in the central or peripheral nervous system in patients with chronic symptoms. Although the cause remains unknown, psychological and physical manifestations are believed to derive from a single central nervous system abnormality (11).

The systematic review of Galli et al. (12) seems to support the role of anxiety and depression in the etiology of BMS. At the same time, further studies are needed to clarify the possible role of other psychological factors. Although some studies have not reported evidence of their involvement in BMS, using the State-Trait Anxiety Inventory (STAI) (13), many others show us the close correlation between anxiety, depression, and BMS. Using the same scale, Schiavone et al. (14) highlighted a higher prevalence of anxiety and depression in patients with burning mouth syndrome compared to healthy controls.

Neuroendocrine and endocrine system disorders are also described in patients with BMS. Plasma adrenaline levels are significantly lower among individuals with BMS (15).

Cytokine levels in patients with burning mouth syndrome compared to healthy controls can be evaluated to determine their role in the pathogenesis of this condition. IL-6 and TNF-α increase in patients with BMS compared to healthy subjects, supporting a neuroinflammatory etiology (16-17). However, some studies do not confirm significant differences in the levels of these cytokines between patients and controls (18). The study of Ji et al. (19) reported elevated levels of IL-18 as the most likely contributor to the etiopathogenesis of BMS.

Diagnosis of BMS

Patients affected by BMS report a persistent burning sensation in the oral cavity, predominantly localized in the anterior two-thirds of the tongue, extending towards the hard palate, gums, and oral mucosa. In 10% of cases, involvement of the lower lip is observed. Symptoms such as allodynia, sharp pain, tingling, sensations of electric shocks, numbness, and itching may occur. The pain is generally bilateral and symmetric, but it can intensify unilaterally throughout the day; it is usually spontaneous, although it may be exacerbated by spicy or acidic foods (20).

Different classifications of BMS based on its

etiology are reported in the literature: Scala et al. (1) have differentiated BMS into primary (idiopathic) and secondary (resulting from local or systemic conditions). Lamey (21) has identified three types: Type I, characterized by a burning sensation increasing throughout the day without psychiatric disorders; Type II, where there is constant pain associated with chronic anxiety; Type III, where the pain is intermittent and localized in atypical areas, connected to stomatitis, adverse food reactions, and nonspecific psychiatric disorders. Coculescu et al. (22) classified the etiological factors into local, systemic, psychological, neurological, and idiopathic categories.

The diagnosis of BMS is mainly based on the exclusion of secondary factors. A detailed medical and dental history is essential, including an evaluation of pain, identification of aggravating or alleviating factors, and the patient's psychosocial history. The physical examination must include examining the head, neck, temporomandibular joint, masticatory muscles, and cranial nerves and assessing oral health and dental prostheses (20).

The recommended laboratory tests include complete blood count, blood glucose, HbA1c, thyroid tests, assessments of iron and ferritin, IgE levels, and vitamins B6, B12, and D, as well as ANA, SSA/SSB, ESR, antibodies for H. pylori and Candida, and swabs for viral and bacterial infections. Patch tests and psychometric evaluations (such as SCL-90R, MPI, HADS, and Beck) can help identify psychological influences. It is also appropriate to consider the study of gastroesophageal reflux (20).

A second type of BMS is a secondary syndrome characterized by local or systemic alterations that can explain the symptoms.

The systemic factors underlying secondary BMS include deficiencies in iron, zinc, vitamin B12, and folates; hormonal disorders (diabetes, thyroid disorders, anemia); and the use of certain medications (benzodiazepines, neuroleptics, antihypertensives).

Zinc is essential in growth and development, immune response, neurological function, and reproduction. In a study of 276 patients with BMS, 74 (26.8%) had low serum zinc levels, suggesting its possible role in the onset of BMS symptoms. Zinc supplementation therapy reduced the average pain score on the numeric scale in these patients from 8.1 to 4.1, compared to an average decrease from 7.7 to 6.7 in a control group (23). The Numeric Rating Scale (NRS) is an 11-point scale where the patient indicates the intensity of their pain verbally or by circling the number that best describes it. It is a convenient and simple tool to administer, requiring no visuomotor coordination (24).

Thyroid hormone imbalances also appear relevant in secondary burning mouth syndrome onset. It seems to be particularly associated with hypothyroidism (25). Additionally, the levels of TSH (thyroid-stimulating hormone), anti-TPO (anti-thyroid peroxidase antibodies), anti-TG (anti-thyroglobulin antibodies), and FT3 (free triiodothyronine) in patients with Hashimoto's thyroiditis were correlated with the presence and severity of BMS (26). Many patients with thyroid abnormalities, in addition to the burning sensation, also experience taste alterations. Thyroid hormones correlate with taste buds' maturation and specialization (27).

Local factors include alterations such as poor denture adaptability, parafunctional habits, galvanism, allergic reactions, or xerostomia (28-29).

The secondary burning mouth syndrome diagnosis is made after thoroughly analyzing the patient's clinical and pharmacological history, physical examination, and blood tests.

Treatment focuses on addressing the underlying causes and symptom remission. It is odd to affirm that a multidisciplinary approach involving dentists, physicians, nutritionists, and other specialists is often required.

Management of patients affected by BMS

The initial approach to managing patients affected by BMS focuses on ruling out any underlying systemic or local conditions that may be causing the symptoms.

There are no established treatment guidelines for BMS due to unclear etiological factors.

The main objective for the clinician is to alleviate symptoms and enhance the patient's quality of life.

Therapeutic strategies include traditional pharmacological approaches, such as anticonvulsants or antidepressants, medicinal plants, and natural supplements. Additionally, given the link between BMS and psychological factors, a multidisciplinary approach, combining medical treatment with psychological therapy, is often recommended.

Pharmacological therapy

Antidepressants

Selective Serotonin Re-uptake Inhibitors (SSRIs) inhibit the re-uptake of serotonin, prolonging its availability at the synaptic cleft.

While some studies on drugs such as Sertraline and Paroxetine have shown favorable efficacy results, confidence in these findings is limited by the lack of placebo-controlled and blinded studies (30).

Serotonin-norepinephrine reuptake Inhibitors (SNRIs) act on both noradrenergic and serotonergic neurons in the nervous system. Serotonin and norepinephrine mediate the endogenous mechanisms of pain inhibition (31).

Trazodone is a second-generation antidepressant that acts as a serotonin reuptake inhibitor and has been used in the treatment of anxiety and pain symptoms.

The administration of 100 mg of Trazodone per day for the first four days, followed by 200 mg for eight weeks, did not show benefits compared to placebo in pain reduction (5). Additionally, the reported adverse effects, such as dizziness, drowsiness, abdominal pain, headaches, palpitations, tremors, xerostomia, and urinary incontinence, limit its use (32).

Amitriptyline is a tricyclic antidepressant (TCA) with analgesic properties. A recent retrospective study compared the efficacy of Amitriptyline and Clonazepam in reducing oral pain. Patients were evaluated at six weeks and three months, with both groups reporting reduced pain. The main issue with Amitriptyline is its frequent side effect of xerostomia, which can worsen

the pre-existing dry mouth associated with BMS (33).
Milnacipran treatment has significantly improved chronic pain in the orofacial region. (34). Kato et al. also demonstrated effective results with this drug. The improvement rate increases with the higher daily doses of Milnacipran (30 mg/ 60 mg/ 90 mg) (35).

Anticonvulsants

Gabapentin and Pregabalin reduce the production of excitatory neurotransmitters such as glutamate, noradrenaline, and substance P by binding to the α2δ subunit of voltage-gated calcium channels. Due to their favorable hepatic safety profile, these drugs are widely used in controlling pain associated with diabetic neuropathy and post-herpetic neuralgia (36).
In a crossover study, patients with BMS were treated with Gabapentin, Alpha-Lipoic Acid (ALA), a combination of both, or a placebo. Fifty percent of the subjects who took Gabapentin reported improvements in pain scores, compared to only 15% in the placebo group. The anticonvulsant combined with ALA produced superior effects, with a 70% reduction in patient pain (6). Similar results had already been highlighted in a previous study: administering 300 mg of Gabapentin combined with ALA led to a significant improvement in pain and, in some cases, complete recovery after two months of therapy (37).
Similar benefits were also achieved with the administration of 150 mg of Pregabalin, reducing VAS scores after four months (42). Additionally, systemic use of Pregabalin (150 mg), compared to Clonazepam (2 mg), has shown significant efficacy in reducing pain scores, although it comes with the risk of more severe potential side effects (38).
Clonazepam topical administration seems to be a good option for the initial management of BMS. It provides rapid analgesia, though short duration, and reduces the side effects of oral administration.
Clonazepam (0.5-2.0 mg) significantly improved pain intensity in the test group compared to the control group, which was treated with a placebo after one month. At six months, the majority of patients treated with this benzodiazepine showed a 50% reduction in symptoms, with five experiencing total remission (39). Furthermore, a meta-analysis by Cui et al. (40) confirmed that topical Clonazepam is an effective treatment modality for both short-term (less than ten weeks) and long-term (more than ten weeks) use. Although this therapy appears to be effective in managing pain, preliminary data indicate that mood, taste dysfunction, and xerostomia do not improve. Additionally, side effects such as dizziness, transient diarrhea, and myalgia have been reported (41).

H2 Receptor Antagonists

Lafutidine is an H2 receptor antagonist that inhibits gastric acid secretion. A randomized controlled study on 74 patients with BMS demonstrated the benefits of administering 10 mg of Lafutidine twice daily. After four, eight, and twelve weeks of treatment, VAS scores improved significantly compared to baseline in the group treated with the drug. Minor side effects, including mild nausea and abdominal distension, did not require treatment discontinuation (42).

Saliva substitutes

Saliva substitutes, such as Biotene and urea, often relieve xerostomia in patients with burning mouth syndrome, which can further affect taste function. Mouth rinses with lysozyme lactoperoxidase (Biotene) did not show a reduction in pain compared to placebo in the long term (four months) (43). Topical application of urea also did not show a statistically significant difference compared to placebo after three months (44).

Phytomedicine

Capsaicin, derived from *Capsicum frutescens*, is a potent agonist of the TRPV1 (transient receptor potential vanilloid) receptor, a non-selective cation channel predominantly expressed in sensory neurons. Initially, its application causes a transient burning sensation due to capsaicin binding to TRPV1, which opens the channel, allowing the influx of Na+ and Ca++ ions and subsequent membrane depolarization (45). For this reason, a pre-treatment with anesthetic cream has been proposed to reduce the temporary increase in intraoral burning sensation induced by capsaicin patches (46). This initial burning sensation is followed by a prolonged phase of analgesia, during which pain fibers become insensitive to nociceptive stimuli due to functional and structural changes in the nerve fibers themselves; this process contributes to the long-lasting pain relief provided by capsaicin, making it a promising option for managing chronic pain conditions such as BMS (47).
Previous studies have shown that these receptors significantly increase in the lingual mucosa of patients with BMS. Activation of TRPV1 in the peripheral nerve endings leads to the release of neuropeptides such as substance P, neurokinin A (NKA), and calcitonin gene-related peptide (CGRP), which contribute to the onset of pain through hyperalgesia and inflammation (48).
Short-term studies (two months) on the efficacy of capsaicin have shown that 76% of participants improved their symptoms, with a significant reduction in VAS scores; in long-term studies (four months), capsaicin, when used topically, also maintained a substantial decrease in VAS scores, demonstrating its sustained efficacy in managing symptoms over time (43).
The oral administration of capsaicin also statistically improved VAS scores in patients with BMS. However, 32% of participants reported severe gastric pain by the end of the four-week treatment period (49).
The active extracts derived from *Hypericum perforatum* have a strong affinity for gamma-aminobutyric acid (GABA), adenosine, serotonin 5HT1 receptors, and benzodiazepine receptors. Additionally, they act as a monoamine oxidase inhibitor (MAOI), contributing to its antidepressant and anxiolytic effects (50). By inhibiting the re-uptake of norepinephrine, serotonin, and dopamine, *Hypericum perforatum* provides beneficial antidepressant effects. As a GABA agonist, it induces temporary hyperpolarization of the neuronal membrane, leading to desensitization and inhibition of neurotransmission, resulting in anxiolytic and analgesic effects (51). Short-term use (twelve

weeks) of *Hypericum perforatum* in treating burning mouth syndrome has shown favorable outcomes but not significantly better than those observed with a placebo (52). Moreover, *Hypericum perforatum* rarely causes adverse reactions, except for dizziness, and it is generally well-tolerated. However, when used in combination with other medications, it may lead to severe interactions. By activating cytochrome P450 enzymes, which are involved in drug metabolism, it reduces the plasma concentration and potency of certain drugs, such as warfarin, cyclosporine, oral contraceptives, anticonvulsants, digoxin, theophylline, and HIV protease inhibitors. Additionally, it can enhance the serotonergic action of serotonin receptor agonists (triptans), SSRIs, SNRIs, TCAs, and MAOIs (53).

Alpha lipoic acid (ALA) is a natural substance in the body and vegetables such as tomatoes, potatoes, and broccoli. It acts as an enzymatic cofactor for the pyruvate dehydrogenase and α-ketoglutarate dehydrogenase complexes in glucose and lipid metabolism. Alpha-lipoic acid is a powerful universal antioxidant in treating diabetic neuropathies (54). As a result, ALA has been studied as a treatment for BMS with underlying peripheral neuropathy. The evidence regarding the efficacy of ALA in treating BMS is mixed: several randomized, double-blind, and controlled studies have demonstrated a positive effect of ALA in improving pain after two months (55). Other studies, however, have shown no significant benefit compared to placebo (56).

Catuama is an herbal product composed of four medicinal plant extracts: *Paullinia cupana* (guarana), *Trichilia catigua* (catuaba), *Zingiber officinale* (ginger), and *Ptychopetalum olacoides* (muirapuama). It acts on dopaminergic, serotonergic, and opioid pathways, demonstrating antidepressant, antinociceptive, and vasorelaxant effects in animal models (57). After three months, Catuama significantly reduced pain scores in patients with BMS compared to the placebo group. Mild side effects reported include drowsiness, weight gain, and insomnia (58).

Lycopene is a carotenoid found in fruits and vegetables, particularly tomatoes. It has antioxidant, anti-inflammatory, and anti-apoptotic properties (59). In a double-blind study on 60 patients with BMS, the effect of administering olive oil enriched with lycopene (300 ppm) over 12 weeks was compared to a placebo (water and coloring). Both groups showed improvement in VAS scores but without statistically significant differences (60).

Crocin is a carotenoid found in flowers of the Crocus genus; it has neuroprotective effects by reducing oxidative stress and cell death by inhibiting microglial activation and suppressing inflammatory cytokine production (61). It was demonstrated that Crocin is a source of antioxidant activity against reactive oxygen species (ROS) (62). Palmitoylethanolamide (PEA) is an endogenous compound (fatty acids and ethanolamide) with anti-inflammatory and analgesic properties belonging to the N-acyl ethanolamines. Its micronized (mPEA) or ultra-micronized (umPEA) forms have shown a progressive reduction in pain intensity, significantly more significant than the control, in patients suffering from chronic and/or neuropathic pain (63). Its efficacy has also been studied in patients with BMS. After administering 1200 mg/day of umPEA, a significant short-term benefit (60 days) was observed, although this effect diminished after four months (64).

Melatonin is a neurohormone that regulates circadian biological rhythms and possesses antioxidant, anti-inflammatory, anticancer, anxiolytic, and antinociceptive activities (65).

A crossover clinical study of Varoni et al. using high doses of melatonin, thus 12 mg/day, did not provide pain relief or improvement in sleep scores compared to placebo in the short term (two months). Four patients discontinued the treatment due to side effects such as severe tremors, sexual dysfunction, blurred vision, and a severe sensation of heaviness (66).

Castillo-Felipe et al. (67), instead, demonstrated that the administration of significantly lower doses of melatonin (1 mg/day) reduces the burning sensation, depression, and anxiety without the occurrence of serious side effects.

Photobiomodulation (PBM)

Photobiomodulation is a therapy that uses various light sources to benefit cells and tissues. It has analgesic, anti-inflammatory, and biological stimulation effects, improving pain relief and tissue healing (68).

The effectiveness of low-level laser therapy (LLLT) has been shown in cases of chronic low back pain, chronic myofascial pain syndrome in the neck, and chronic neck pain (69). Furthermore, it reduces the burning sensation by increasing the release of serotonin and β-endorphins while decreasing bradykinin secretion. It blocks the depolarization of C fibers, which transmit heat and pain stimuli (70). The reduction of intraoral burning may also occur due to the decrease in vasodilation caused by the narrowing of the capillary diameter. Morphological changes in the capillary bed have been observed through video capillaroscopy (71).

Spanemberg et al.'s study (72) demonstrated a significant reduction in pain scores at 11 weeks in the group treated with infrared laser therapy, while the group treated with red laser therapy showed no significant differences compared to the controls.

A recent long-term study suggested the advantage of PBM in treating orofacial neuropathic pain, including BMS, with a 4.5-fold likelihood of pain reduction compared to placebo. However, no improvement was observed in the patient's psychological well-being or quality of life (73).

Other studies have also highlighted the efficacy of PBM, such as the one conducted by Arbabi-Kalati et al. (74), who evaluated 20 patients with BMS divided into two groups (laser and placebo). The laser group was irradiated (gallium-arsenide-iodine laser, 630 nm, 30 mW, 1 J/cm²) twice a week for two weeks, targeting 10 areas of the oral mucosa. The placebo group received the same treatment with the laser turned off. Results were collected at the end of the two-week treatment, and the laser group showed a statistically

significant improvement in VAS scores compared to the placebo group.
Dos Santos et al. (75) treated 10 patients with BMS using a diode laser at 660 nm, 40 mW power, 20 J/cm², with one weekly session for 10 weeks. The treatment significantly improved all patients' symptoms.
Thanks to its effectiveness in reducing pain and improving the quality of life for patients with BMS, PBM could represent an alternative therapy for BMS. PBM is superior to Clonazepam (76), providing consistent and long-lasting pain relief.
However, further evidence is still needed to ensure its efficacy and safety in treating BMS. Additionally, it is essential to optimize application parameters (wavelength, power, dose, exposure time, spot size) and the techniques used.

Neuromodulation and physiological techniques against BMS

Repetitive transcranial magnetic stimulation (rTMS) is a non-invasive neuromodulation technique designed for treating BMS following brain neuroimaging studies that demonstrated changes in the pain matrix (77). Unilateral Repetitive transcranial magnetic stimulation of the primary motor cortex (M1) and dorsolateral prefrontal cortex (DLPFC) produces a widespread analgesic effect in both experimental and clinical pain studies (78). A study by Umezaki et al. demonstrated a rapid decrease in VAS scores, which remained stable for up to two months following rTMS treatment (79). However, this approach requires expensive equipment and significantly more time and commitment in the clinic than conventional pharmacological treatments.
Cognitive Behavioral Therapy (CBT) is a psychological treatment used to manage depression, anxiety, and physical symptoms. Specific techniques include biofeedback, relaxation, exposure, and cognitive reframing. In BMS, pain-related catastrophizing, a cognitive factor, influences both the intensity of pain and the quality of life. This factor perpetuates and exacerbates chronic pain; a treatment focused on reducing pain-related catastrophizing has significantly improved symptoms in patients with BMS (80).
The efficacy of CBT was demonstrated in an early study where patients with BMS were randomized into a cognitive therapy group, which met weekly for 12-15 weeks, or an attention-control group. The treatment group showed a statistically significant decrease in pain severity, and this benefit was maintained at the six-month follow-up (81). Similar results were obtained in a more recent study where patients underwent group psychotherapy for three months. A significantly higher percentage of participants in group therapy reported symptomatic improvement compared to those who received standard care (70% versus 40%). Although the effects of treatment have been significant and results maintained for 6-12 months, completing a course of CBT requires 12-16 sessions, which can be costly. A group format may be considered as an alternative to individual therapy to address this issue. Previous research has shown no significant differences in effectiveness between these two formats (82).

Conclusions

This review explored the potential etiopathogenetic mechanisms underlying BMS and the most appropriate clinical management for its treatment.
BMS is a multifactorial condition with a complex etiopathogenesis. Current evidence supports a neuropathic origin, with psychological factors, hormonal changes, and circadian rhythms possibly contributing, but unclearly. BMS treatment strategies vary, including central neuromodulators, alpha lipoic acid, and LLLT. Photobiomodulation has emerged as a promising alternative, offering long-lasting pain relief with minimal side effects. Finally, psychological approaches, like CBT, are essential to BMS management, such as adjuvant therapy.
The multidisciplinary approach is the most effective strategy for managing BMS.
Further studies analyzing larger sample sizes are required to develop therapeutic schemes that ensure long-term symptom relief and improved patient quality of life.

Declarations

Funding
There is no funding for this article.

Conflicts of interest

The authors declare no conflicts of interest.

References

1. Scala A, Checchi L, Montevecchi M, Marini I, Giamberardino MA. Update on Burning Mouth Syndrome: Overview and Patient Management. Crit Rev Oral Biol Med. 2003;14(4):275-91. Doi: 10.1177/154411130301400405.
2. International Classification of Orofacial Pain, 1st Edition (ICOP). Cephalalgia. 2020 Feb;40(2):129-221. Doi: 10.1177/0333102419893823.
3. Raja SN, Carr DB, Cohen M, Finnerup NB, Flor H, Gibson S, Keefe FJ, Mogil JS, Ringkamp M, Sluka KA, Song XJ, Stevens B, Sullivan MD, Tutelman PR, Ushida T, Vader K. The Revised International Association for the Study of Pain Definition of Pain: Concepts, Challenges, and Compromises. Pain. 2020 Sep 1;161(9):1976-1982. Doi: 10.1097/j.Pain.0000000000001939.
4. Balasubramaniam R, Klasser GD, Delcanho R. Separating Oral Burning from Burning Mouth Syndrome: Unravelling a Diagnostic Enigma. Aust Dent J. 2009 Dec;54(4):293-9. Doi: 10.1111/j.1834-7819.2009.01153.x.
5. Tan HL, Smith JG, Hoffmann J, Renton T. A Systematic Review of Treatment for Patients with Burning Mouth Syndrome. Cephalalgia. 2022 Feb;42(2):128-161. Doi: 10.1177/03331024211036152.
6. Liu YF, Kim Y, Yoo T, Han P, Inman JC. Burning Mouth Syndrome: A Systematic Review of Treatments. Oral Dis. 2018 Apr;24(3):325-334. Doi: 10.1111/Odi.12660.
7. Kohorst JJ, Bruce AJ, Torgerson RR, Schenck LA, Davis MDP. The Prevalence of Burning Mouth Syndrome: A Population-Based Study. Br J Dermatol. 2015 Jun;172(6):1654-1656. Doi: 10.1111/Bjd.13613.
8. Eli I, Baht R, Littner MM, Kleinhauz M. Detection of Psychopathologic Trends in Glossodynia Patients. Psychosom Med. 1994 Sep-Oct;56(5):389-94. Doi: 10.1097/00006842-199409000-00002.
9. Klein B, Thoppay JR, De Rossi SS, Ciarrocca K. Burning Mouth Syndrome. Dermatol Clin. 2020 Oct;38(4):477-483. Doi: 10.1016/j.Det.2020.05.008.
10. Fedele S, Fricchione G, Porter SR, Mignogna MD.

Burning Mouth Syndrome (Stomatodynia). QJM. 2007 Aug;100(8):527-30. Doi: 10.1093/Qjmed/Hcm049.
11. Nicoletti M, Neri G, Maccauro G, Tripodi D, Varvara G, Saggini A, Potalivo G, Castellani ML, Fulcheri M, Rosati M, Toniato E, Caraffa A, Antinolfi P, Cerulli G, Pandolfi F, Galzio R, Conti P, Theoharides TC. Impact of Neuropeptide Substance P an Inflammatory Compound on Arachidonic Acid Compound Generation. Int J Immunopathol Pharmacol. 2012 Oct-Dec;25(4):849-57. Doi: 10.1177/039463201202500403.
12. Galli F, Lodi G, Sardella A, Vegni E. Role of Psychological Factors in Burning Mouth Syndrome: A Systematic Review and Meta-Analysis. Cephalalgia. 2017 Mar;37(3):265-277. Doi: 10.1177/0333102416646769.
13. Mendak-Ziółko M, Konopka T, Bogucki ZA. Evaluation of Select Neurophysiological, Clinical and Psychological Tests for Burning Mouth Syndrome. Oral Surg Oral Med Oral Pathol Oral Radiol. 2012 Sep;114(3):325-32. Doi: 10.1016/j.Oooo.2012.04.004.
14. Schiavone V, Adamo D, Ventrella G, Morlino M, De Notaris EB, Ravel MG, Kusmann F, Piantadosi M, Pollio A, Fortuna G, Mignogna MD. Anxiety, Depression, and Pain in Burning Mouth Syndrome: First Chicken or Egg? Headache. 2012 Jun;52(6):1019-25. Doi: 10.1111/j.1526-4610.2012.02171.x.
15. Koike K, Shinozaki T, Hara K, Noma N, Okada-Ogawa A, Asano M, Shinoda M, Eliav E, Gracely RH, Iwata K, Imamura Y. Immune and Endocrine Function in Patients with Burning Mouth Syndrome. Clin J Pain. 2014 Feb;30(2):168-73. Doi: 10.1097/AJP.0b013e31828c4bf1.
16. Shaik Y, Sabatino G, Maccauro G, Varvara G, Murmura G, Saggini A, Rosati M, Conti F, Cianchetti E, Caraffa A, Antinolfi P, Pandolfi F, Potalivo G, Galzio R, Conti P, Theoharides TC. IL-36 Receptor Antagonist with Special Emphasis on IL-38. Int J Immunopathol Pharmacol. 2013 Jan-Mar;26(1):27-36. Doi: 10.1177/039463201302600103.
17. Kritas SK, Saggini A, Varvara G, Murmura G, Caraffa A, Antinolfi P, Toniato E, Pantalone A, Neri G, Frydas S, Rosati M, Tei M, Speziali A, Saggini R, Pandolfi F, Theoharides TC, Conti P. Mast Cell Involvement in Rheumatoid Arthritis. J Biol Regul Homeost Agents. 2013 Jul-Sep;27(3):655-60.
18. Boras VV, Brailo V, Lukac J, Kordić D, Blazić-Potocki Z. Salivary Interleukin-6 and Tumor Necrosis Factor-Alpha in Patients with Burning Mouth Syndrome. Oral Dis. 2006 May;12(3):353-5. Doi: 10.1111/j.1601-0825.2005.01209.x.
19. Ji EH, Diep C, Liu T, Li H, Merrill R, Messadi D, Hu S. Potential Protein Biomarkers for Burning Mouth Syndrome Discovered by Quantitative Proteomics. Mol Pain. 2017 Jan;13:1744806916686796. Doi: 10.1177/1744806916686796.
20. Bender SD. Burning Mouth Syndrome. Dent Clin North Am. 2018 Oct;62(4):585-596. Doi: 10.1016/j.Cden.2018.05.006.
21. Lamey PJ. Burning Mouth Syndrome. Dermatol Clin. 1996 Apr;14(2):339-54. Doi: 10.1016/S0733-8635(05)70361-2.
22. Coculescu EC, Radu A, Coculescu BI. Burning mouth syndrome: a review on diagnosis and treatment. J Med Life. 2014 Oct-Dec;7(4):512-5.
23. Cho GS, Han MW, Lee B, Roh JL, Choi SH, Cho KJ, Nam SY, Kim SY. Zinc Deficiency May Be a Cause of Burning Mouth Syndrome as Zinc Replacement Therapy Has Therapeutic Effects. J Oral Pathol Med. 2010 Oct;39(9):722-7. Doi: 10.1111/j.1600-0714.2010.00914.x.
24. Hartrick CT. A Four-Category Verbal Rating Scale (VRS-4), an 11-Point Numeric Rating Scale (NRS-11), and a 100-Mm Visual Analog Scale (VAS) Were Compared in the Assessment of Acute Pain after Oral Surgery. Clin J Pain. 2001 Mar;17(1):104-5. Doi: 10.1097/00002508-200103000-00016.
25. Egido-Moreno S, Valls-Roca-Umbert J, Perez-Sayans M, Blanco-Carrión A, Jane-Salas E, López-López J. Role of Thyroid Hormones in Burning Mouth Syndrome. Systematic Review. Med Oral Patol Oral Cir Bucal. 2023 Jan 1;28(1):E81-E86. Doi: 10.4317/Medoral.25596.
26. Talattof Z, Dabbaghmanesh MH, Parvizi Y, Esnaashari N, Azad A. The Association between Burning Mouth Syndrome and Level of Thyroid Hormones in Hashimotos Thyroiditis in Public Hospitals in Shiraz, 2016. J Dent (Shiraz). 2019 Mar;20(1):42-47. Doi: 10.30476/DENTJODS.2019.44562.
27. Brosvic GM, Doty RL, Rowe MM, Harron A, Kolodiy N. Influences of Hypothyroidism on the Taste Detection Performance of Rats: A Signal Detection Analysis. Behav Neurosci. 1992 Dec;106(6):992-8. Doi: 10.1037//0735-7044.106.6.992.
28. Traini T, Pettinicchio M, Murmura G, Varvara G, Di Lullo N, Sinjari B, Caputi S. Esthetic Outcome of an Immediately Placed Maxillary Anterior Single-Tooth Implant Restored with a Custom-Made Zirconia-Ceramic Abutment and Crown: A Staged Treatment. Quintessence Int. 2011 Feb;42(2):103-8.
29. Perinetti G, Caputi S, Varvara G. Risk/Prevention Indicators for the Prevalence of Dental Caries in Schoolchildren: Results from the Italian OHSAR Survey. Caries Res. 2005 Jan-Feb;39(1):9-19. Doi: 10.1159/000081651.
30. Yamazaki Y, Hata H, Kitamori S, Onodera M, Kitagawa Y. An Open-Label, Noncomparative, Dose Escalation Pilot Study of the Effect of Paroxetine in Treatment of Burning Mouth Syndrome. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2009 Jan;107(1):E6-11. Doi: 10.1016/j.Tripleo.2008.08.024.
31. Welsch P, Üçeyler N, Klose P, Walitt B, Häuser W. Serotonin and Noradrenaline Reuptake Inhibitors (SNRIs) for Fibromyalgia. Cochrane Database Syst Rev. 2018 Feb 28;2(2):CD010292. Doi: 10.1002/14651858.CD010292.Pub2.
32. Tammiala-Salonen T, Forssell H. Trazodone in Burning Mouth Pain: A Placebo-Controlled, Double-Blind Study. J Orofac Pain. 1999 Spring;13(2):83-8.
33. Fenelon M, Quinque E, Arrive E, Catros S, Fricain JC. Pain-Relieving Effects of Clonazepam and Amitriptyline in Burning Mouth Syndrome: A Retrospective Study. Int J Oral Maxillofac Surg. 2017 Nov;46(11):1505-1511. Doi: 10.1016/j.Ijom.2017.03.032.
34. Ito M, Kimura H, Yoshida K, Kimura Y, Ozaki N, Kurita K. Effectiveness of Milnacipran for the Treatment of Chronic Pain in the Orofacial Region. Clin Neuropharmacol. 2010 Mar-Apr;33(2):79-83. Doi: 10.1097/WNF.0b013e3181cb5793.
35. Kato Y, Sato T, Katagiri A, Umezaki Y, Takenoshita M, Yoshikawa T, Sato Y, Toyofuku A. Milnacipran Dose-Effect Study in Patients with Burning Mouth Syndrome. Clin Neuropharmacol. 2011 Jul-Aug;34(4):166-9. Doi: 10.1097/WNF.0b013e318227f100.
36. Wiffen PJ, Derry S, Bell RF, Rice AS, Tölle TR, Phillips T, Moore RA. Gabapentin for Chronic Neuropathic Pain in Adults. Cochrane Database Syst Rev. 2017 Jun 9;6(6):CD007938. Doi: 10.1002/14651858.CD007938.Pub4.
37. López-D'alessandro E, Escovich L. Combination of Alpha Lipoic Acid and Gabapentin, Its Efficacy in the Treatment of Burning Mouth Syndrome: A Randomized, Double-Blind, Placebo Controlled Trial. Med Oral Patol Oral Cir Bucal. 2011 Aug 1;16(5):E635-40. Doi: 10.4317/Medoral.16942.
38. Çınar SL, Kartal D, Pergel T, Borlu M. Effectiveness and Safety of Clonazepam, Pregabalin, and Alpha Lipoic Acid for the Treatment of Burning Mouth Syndrome. Erciyes Med J. 2018;40(1):35-8. Doi: 10.5152/Etd.2018.17160.
39. Rodríguez de Rivera Campillo E, López-López J, Chimenos-Küstner E. Response to Topical Clonazepam in Patients with Burning Mouth Syndrome: A Clinical Study. Bull Group Int Rech Sci Stomatol Odontol. 2010 May 21;49(1):19-29.
40. Cui Y, Xu H, Chen FM, Liu JL, Jiang L, Zhou Y, Chen QM. Efficacy Evaluation of Clonazepam for Symptom Remission in Burning Mouth Syndrome: A Meta-Analysis. Oral Dis. 2016 Sep;22(6):503-11. Doi: 10.1111/Odi.12422.
41. Heckmann SM, Kirchner E, Grushka M, Wichmann MG, Hummel T. A Double-Blind Study on Clonazepam in Patients with Burning Mouth Syndrome. Laryngoscope. 2012 Apr;122(4):813-6. Doi: 10.1002/Lary.22490.
42. Toida M, Kato K, Makita H, Long NK, Takeda T, Hatakeyama D, Yamashita T, Shibata T. Palliative Effect of Lafutidine on Oral Burning Sensation. J Oral Pathol Med. 2009

Mar;38(3):262-8. Doi: 10.1111/j.1600-0714.2008.00736.x.
43. Marino R, Torretta S, Capaccio P, Pignataro L, Spadari F. Different Therapeutic Strategies for Burning Mouth Syndrome: Preliminary Data. J Oral Pathol Med. 2010 Sep;39(8):611-6. Doi: 10.1111/j.1600-0714.2010.00922.x.
44. Silva LA, Siqueira JT, Teixeira MJ, Siqueira SR. The Role of Xerostomia in Burning Mouth Syndrome: A Case-Control Study. Arq Neuropsiquiatr. 2014 Feb;72(2):91-8. Doi: 10.1590/0004-282X20130218.
45. Frydas S, Varvara G, Murmura G, Saggini A, Caraffa A, Antinolfi P, Tete' S, Tripodi D, Conti F, Cianchetti E, Toniato E, Rosati M, Speranza L, Pantalone A, Saggini R, Di Tommaso LM, Theoharides TC, Conti P, Pandolfi F. Impact of Capsaicin on Mast Cell Inflammation. Int J Immunopathol Pharmacol. 2013 Jul-Sep;26(3):597-600. Doi: 10.1177/039463201302600303.
46. Jensen TS, Høye K, Fricová J, Vanelderen P, Ernault E, Siciliano T, Marques S. Tolerability of the Capsaicin 8% Patch Following Pretreatment with Lidocaine or Tramadol in Patients with Peripheral Neuropathic Pain: A Multicentre, Randomized, Assessor-Blinded Study. Eur J Pain. 2014 Oct;18(9):1240-7. Doi: 10.1002/j.1532-2149.2014.00479.x.
47. Anand P, Bley K. Topical Capsaicin for Pain Management: Therapeutic Potential and Mechanisms of Action of the New High-Concentration Capsaicin 8% Patch. Br J Anaesth. 2011 Oct;107(4):490-502. Doi: 10.1093/Bja/Aer260.
48. Jääskeläinen SK. Pathophysiology of Primary Burning Mouth Syndrome. Clin Neurophysiol. 2012 Jan;123(1):71-7. Doi: 10.1016/j.Clinph.2011.07.054.
49. Petruzzi M, Lauritano D, De Benedittis M, Baldoni M, Serpico R. Systemic Capsaicin for Burning Mouth Syndrome: Short-Term Results of a Pilot Study. J Oral Pathol Med. 2004 Feb;33(2):111-4. Doi: 10.1111/j.1600-0714.2004.0194n.x.
50. Müller WE, Rolli M, Schäfer C, Hafner U. Effects of Hypericum Extract (LI 160) in Biochemical Models of Antidepressant Activity. Pharmacopsychiatry. 1997 Sep;30 Suppl 2:102-7. Doi: 10.1055/s-2007-979528.
51. McCarson KE, Enna SJ. GABA Pharmacology: The Search for Analgesics. Neurochem Res. 2014 Oct;39(10):1948-63. Doi: 10.1007/S11064-014-1254-x.
52. Sardella A, Lodi G, Demarosi F, Tarozzi M, Canegallo L, Carrassi A. Hypericum Perforatum Extract in Burning Mouth Syndrome: A Randomized Placebo-Controlled Study. J Oral Pathol Med. 2008 Aug;37(7):395-401. Doi: 10.1111/j.1600-0714.2008.00663.x.
53. Henderson L, Yue QY, Bergquist C, Gerden B, Arlett P. St John's Wort (Hypericum Perforatum): Drug Interactions and Clinical Outcomes. Br J Clin Pharmacol. 2002 Oct;54(4):349-56. Doi: 10.1046/j.1365-2125.2002.01683.x.
54. Ziegler D, Low PA, Litchy WJ, Boulton AJ, Vinik AI, Freeman R, Samigullin R, Tritschler H, Munzel U, Maus J, Schütte K, Dyck PJ. Efficacy and Safety of Antioxidant Treatment with α-Lipoic Acid over 4 Years in Diabetic Polyneuropathy: The NATHAN 1 Trial. Diabetes Care. 2011 Sep;34(9):2054-60. Doi: 10.2337/Dc11-0503.
55. Femiano F, Gombos F, Scully C, Busciolano M, De Luca P. Burning Mouth Syndrome (BMS): Controlled Open Trial of the Efficacy of Alpha-Lipoic Acid (Thioctic Acid) on Symptomatology. Oral Dis. 2000 Sep;6(5):274-7. Doi: 10.1111/j.1601-0825.2000.Tb00138.x.
56. Carbone M, Pentenero M, Carrozzo M, Ippolito A, Gandolfo S. Lack of Efficacy of Alpha-Lipoic Acid in Burning Mouth Syndrome: A Double-Blind, Randomized, Placebo-Controlled Study. Eur J Pain. 2009 May;13(5):492-6. Doi: 10.1016/j.Ejpain.2008.06.004.
57. Quintão NL, Ferreira J, Beirith A, Campos MM, Calixto JB. Evaluation of the Effects of the Herbal Product Catuama in Inflammatory and Neuropathic Models of Nociception in Rats. Phytomedicine. 2008 Apr;15(4):245-52. Doi: 10.1016/j.Phymed.2007.03.015.
58. Spanemberg JC, Cherubini K, de Figueiredo MA, Gomes AP, Campos MM, Salum FG. Effect of an Herbal Compound for Treatment of Burning Mouth Syndrome: Randomized, Controlled, Double-Blind Clinical Trial. Oral Surg Oral Med Oral Pathol Oral Radiol. 2012 Mar;113(3):373-7. Doi: 10.1016/j.Oooo.2011.09.005.
59. Conti P, Varvara G, Murmura G, Tete S, Sabatino G, Saggini A, Rosati M, Toniato E, Caraffa A, Antinolfi P, Pandolfi F, Potalivo G, Galzio R, Theoharides TC. Comparison of Beneficial Actions of Non-Steroidal Anti-Inflammatory Drugs to Flavonoids. J Biol Regul Homeost Agents. 2013 Jan-Mar;27(1):1-7.
60. Cano-Carrillo P, Pons-Fuster A, López-Jornet P. Efficacy of Lycopene-Enriched Virgin Olive Oil for Treating Burning Mouth Syndrome: A Double-Blind Randomised. J Oral Rehabil. 2014 Apr;41(4):296-305. Doi: 10.1111/Joor.12147.
61. Shaik-Dasthagirisaheb YB, Varvara G, Murmura G, Saggini A, Caraffa A, Antinolfi P, Tete' S, Tripodi D, Conti F, Cianchetti E, Toniato E, Rosati M, Speranza L, Pantalone A, Saggini R, Tei M, Speziali A, Conti P, Theoharides TC, Pandolfi F. Role of Vitamins D, E and C in Immunity and Inflammation. J Biol Regul Homeost Agents. 2013 Apr-Jun;27(2):291-5.
62. Zeka, K., Ruparelia, K. C., Arroo, R. R., Bernardi, S., Corradini, V., Pajewski, L. A., Vegliò, F., & Continenza, M. A. (2014). In vitro biocompatibility of a new hydrogel with Crocin, powerful antioxidant found in Crocus Sativus L. flowers. Italian Journal of Anatomy and Embryology, 118(2), 200. Retrieved from https://oajournals.fupress.net/index.php/ijae/article/view/3922.
63. Paladini A, Fusco M, Cenacchi T, Schievano C, Piroli A, Varrassi G. Palmitoylethanolamide, a Special Food for Medical Purposes, in the Treatment of Chronic Pain: A Pooled Data Meta-Analysis. Pain Physician. 2016 Feb;19(2):11-24.
64. Ottaviani G, Rupel K, Gobbo M, Poropat A, Zoi V, Faraon M, Di Lenarda R, Biasotto M. Efficacy of Ultramicronized Palmitoylethanolamide in Burning Mouth Syndrome-Affected Patients: A Preliminary Randomized Double-Blind Controlled Trial. Clin Oral Investig. 2019 Jun;23(6):2743-2750. Doi: 10.1007/S00784-018-2720-7.
65. Wilhelmsen M, Amirian I, Reiter RJ, Rosenberg J, Gögenur I. Analgesic Effects of Melatonin: A Review of Current Evidence from Experimental and Clinical Studies. J Pineal Res. 2011 Oct;51(3):270-7. Doi: 10.1111/j.1600-079X.2011.00895.x.
66. Varoni EM, Lo Faro AF, Lodi G, Carrassi A, Iriti M, Sardella A. Melatonin Treatment in Patients with Burning Mouth Syndrome: A Triple-Blind, Placebo-Controlled, Crossover Randomized Clinical Trial. J Oral Facial Pain Headache. 2018 Spring;32(2):178-188. Doi: 10.11607/Ofph.1913.
67. Castillo-Felipe C, Tvarijonaviciute A, López-Arjona M, Pardo-Marin L, Pons-Fuster E, López-Jornet P. Response to Treatment with Melatonin and Clonazepam versus Placebo in Patients with Burning Mouth Syndrome. J Clin Med. 2022 Apr 29;11(9):2516. Doi: 10.3390/Jcm11092516.
68. Bone laser biostimulation applied to implant dentistry: case report. June 2013Clinical Oral Implants Research 24(s8). doi: 10.1111/clr.1218. Conference: Premium Day 2013At: PadovaVolume: Clinical Oral Implants Research 24 (Suppl. 8), 21–22.
69. Chow RT, Heller GZ, Barnsley L. The Effect of 300 mW, 830 Nm Laser on Chronic Neck Pain: A Double-Blind, Randomized, Placebo-Controlled Study. Pain. 2006 Sep;124(1-2):201-10. Doi: 10.1016/j.Pain.2006.05.018.
70. Al-Maweri SA, Javed F, Kalakonda B, AlAizari NA, Al-Soneidar W, Al-Akwa A. Efficacy of Low Level Laser Therapy in the Treatment of Burning Mouth Syndrome: A Systematic Review. Photodiagnosis Photodyn Ther. 2017 Mar;17:188-193. Doi: 10.1016/j.Pdpdt.2016.11.017.
71. Scardina GA, Casella S, Bilello G, Messina P. Photobiomodulation Therapy in the Management of Burning Mouth Syndrome: Morphological Variations in the Capillary Bed. Dent J (Basel). 2020 Sep 1;8(3):99. Doi: 10.3390/Dj8030099.
72. Spanemberg JC, López López J, de Figueiredo MA, Cherubini K, Salum FG. Efficacy of Low-Level Laser Therapy for the Treatment of Burning Mouth Syndrome: A Randomized, Controlled Trial. J Biomed Opt. 2015 Sep;20(9):098001. Doi: 10.1117/1.JBO.20.9.098001.
73. De Pedro M, López-Pintor RM, Casañas E, Hernández G.

Effects of Photobiomodulation with Low-Level Laser Therapy in Burning Mouth Syndrome: A Randomized Clinical Trial. Oral Dis. 2020 Nov;26(8):1764-1776. Doi: 10.1111/Odi.13443.
74. Arbabi-Kalati F, Bakhshani NM, Rasti M. Evaluation of the Efficacy of Low-Level Laser in Improving the Symptoms of Burning Mouth Syndrome. J Clin Exp Dent. 2015 Oct 1;7(4):E524-7. Doi: 10.4317/Jced.52298.
75. Dos Santos Lde F, Carvalho Ade A, Leão JC, Cruz Perez DE, Castro JF. Effect of Low-Level Laser Therapy in the Treatment of Burning Mouth Syndrome: A Case Series. Photomed Laser Surg. 2011 Dec;29(12):793-6. Doi: 10.1089/Pho.2011.3016.
76. Arduino PG, Cafaro A, Garrone M, Gambino A, Cabras M, Romagnoli E, Broccoletti R. A Randomized Pilot Study to Assess the Safety and the Value of Low-Level Laser Therapy versus Clonazepam in Patients with Burning Mouth Syndrome. Lasers Med Sci. 2016 May;31(4):811-6. Doi: 10.1007/S10103-016-1897-8.
77. Tan Y, Wu X, Chen J, Kong L, Qian Z. Structural and Functional Connectivity Between the Amygdala and Orbital Frontal Cortex in Burning Mouth Syndrome: An fMRI Study. Front Psychol. 2019 Jul 25;10:1700. Doi: 10.3389/Fpsyg.2019.01700.
78. Passard A, Attal N, Benadhira R, Brasseur L, Saba G, Sichere P, Perrot S, Januel D, Bouhassira D. Effects of Unilateral Repetitive Transcranial Magnetic Stimulation of the Motor Cortex on Chronic Widespread Pain in Fibromyalgia. Brain. 2007 Oct;130(Pt 10):2661-70. Doi: 10.1093/Brain/Awm189.
79. Umezaki Y, Badran BW, DeVries WH, Moss J, Gonzales T, George MS. The Efficacy of Daily Prefrontal Repetitive Transcranial Magnetic Stimulation (rTMS) for Burning Mouth Syndrome (BMS): A Randomized Controlled Single-Blind Study. Brain Stimul. 2016 Mar-Apr;9(2):234-42. Doi: 10.1016/j.Brs.2015.10.005.
80. Matsuoka H, Chiba I, Sakano Y, Toyofuku A, Abiko Y. Cognitive Behavioral Therapy for Psychosomatic Problems in Dental Settings. Biopsychosoc Med. 2017 Jun 13;11:18. Doi: 10.1186/S13030-017-0102-z.
81. Bergdahl J, Anneroth G, Perris H. Cognitive Therapy in the Treatment of Patients with Resistant Burning Mouth Syndrome: A Controlled Study. J Oral Pathol Med. 1995 May;24(5):213-5. Doi: 10.1111/j.1600-0714.1995.Tb01169.x.
82. Miziara ID, Filho BC, Oliveira R, Rodrigues Dos Santos RM. Group Psychotherapy: An Additional Approach to Burning Mouth Syndrome. J Psychosom Res. 2009 Nov;67(5):443-8. Doi: 10.1016/j.Jpsychores.2009.01.013.