Impact Factor 2024: 7.101

Burning Mouth Syndrome-Pathogenesis, Diagnosis, and Multidisciplinary Approach

Dr. R Sumukh Bharadwaj

Consultant Endodontist, Mysuru, Karnataka, India

Abstract: <u>Background</u>: Burning mouth syndrome (BMS) is a chronic, idiopathic oral pain condition characterized by persistent burning or dysesthetic sensations of the oral mucosa in the absence of visible clinical abnormalities or laboratory findings. It disproportionately affects postmenopausal women and has profound impacts on quality of life. <u>Objective</u>: This review aims to provide an updated and comprehensive overview of the pathogenesis, diagnostic approach, and multidisciplinary management of BMS. <u>Methods</u>: A literature review was conducted using PubMed, Scopus, and Google Scholar databases focusing on studies from 1990–2024. Clinical trials, systematic reviews, and key narrative reviews on BMS were included. <u>Results</u>: The etiology of BMS is multifactorial, with contributions from peripheral neuropathy, central sensitization, hormonal imbalance, psychological disturbances, and salivary alterations. Diagnosis remains one of exclusion, requiring comprehensive clinical evaluation and exclusion of systemic, local, and iatrogenic causes. A multidisciplinary management approach incorporating dental, neurological, psychiatric, nutritional, and alternative therapeutic perspectives yields the best outcomes. <u>Conclusion</u>: BMS remains a complex, heterogeneous disorder with no universally effective treatment. Advances in neurobiology and pain science hold promise for targeted therapies. A patient-centered, multidisciplinary approach remains the cornerstone of effective care.

Keywords: Burning mouth syndrome, neuropathic pain, oral pain, diagnosis, multidisciplinary management, stomatodynia

1. Introduction

Burning mouth syndrome (BMS) is defined by the International Headache Society as a chronic intraoral burning sensation that recurs daily for more than two hours per day for over three months, in the absence of clinically evident mucosal lesions or laboratory abnormalities [1]. Typical symptoms include burning, tingling, or scalding sensations affecting the tongue, lips, and palate, often accompanied by dysgeusia and xerostomia [2].

The reported prevalence varies between 0.7% and 5% depending on diagnostic criteria and population studied [3]. BMS exhibits a marked gender and age predilection, affecting women seven times more frequently than men, particularly in the peri- and postmenopausal period [4]. This epidemiological profile suggests hormonal contributions,

though neurobiological and psychological factors also play major roles.

The clinical burden of BMS is substantial. Patients frequently report **severe impairment in quality of life**, including difficulty eating, sleeping disturbances, emotional distress, and reduced social interaction [5]. Many undergo multiple consultations across specialties without relief, leading to frustration and increased health care costs [6].

Despite decades of research, the **etiopathogenesis remains unclear**, diagnostic criteria lack uniformity, and treatment outcomes remain unpredictable. This underscores the need for a **comprehensive review of current understanding** of its pathogenesis, diagnostic work-up, and the role of multidisciplinary care in improving outcomes.

1.1 Pathogenesis of Burning Mouth Syndrome

Table 1: Proposed Etiological Factors in Burning Mouth Syndrome

Category	Proposed Mechanism	Examples/Notes
Neuropathic factors	Small fiber neuropathy, trigeminal nerve dysfunction	Peripheral/central neuropathy
Hormonal factors	Altered estrogen and progesterone levels	Postmenopausal women more affected
Psychological factors	Stress, anxiety, depression, altered pain perception	Strong correlation with symptom severity
Salivary changes	Hyposalivation, qualitative salivary changes	Xerostomia, mucosal dryness
Nutritional deficiencies	Deficiencies in iron, folic acid, vitamin B complex	Rare but must be excluded
Genetic/immune factors	Altered immune response, genetic polymorphisms	Under investigation

The etiology of BMS is multifactorial and poorly understood, likely involving an interplay between neuropathic, hormonal, psychological, and salivary factors. It is now considered a **neuropathic pain disorder** in many patients, though other mechanisms also contribute.

1) Neuropathic Mechanisms

Peripheral neuropathy has been implicated as a major mechanism. Quantitative sensory testing and biopsies have demonstrated **reduced intraepithelial nerve fiber density in the tongue** of BMS patients, consistent with small-fiber

neuropathy [7]. Neurophysiological studies also suggest dysfunction of the **chorda tympani nerve** leading to impaired taste and compensatory hyperactivity of trigeminal nociceptors [8].

Central mechanisms also play a role. Functional imaging has shown altered activation of the **dopaminergic and serotonergic systems** in the basal ganglia and limbic system, suggesting central pain processing abnormalities [9]. Such findings support the classification of BMS as a "chronic neuropathic pain disorder of the oral cavity."

Volume 14 Issue 9, September 2025
Fully Refereed | Open Access | Double Blind Peer Reviewed Journal
www.ijsr.net

Paper ID: SR25924003350 DOI: https://dx.doi.org/10.21275/SR25924003350

International Journal of Science and Research (IJSR)

ISSN: 2319-7064 Impact Factor 2024: 7.101

2) Hormonal and Endocrine Factors

The high prevalence in postmenopausal women points to an estrogen-related mechanism. Estrogen deficiency alters oral mucosal receptor sensitivity, reduces salivary secretion, and may modify taste perception [10]. However, randomized controlled trials on **hormone replacement therapy** show inconsistent benefit [11]. Thyroid dysfunction and insulin resistance have also been linked with secondary BMS [12].

3) Psychological and Psychiatric Factors

Psychological factors are consistently associated with BMS. Up to 70% of patients demonstrate psychiatric comorbidity, including anxiety, depression, and somatization [13]. Stress-related dysregulation of the hypothalamic-pituitary-adrenal axis may amplify nociceptive input, perpetuating burning sensations [14]. Cognitive-affective processes such as catastrophization further exacerbate symptoms.

4) Salivary Gland and Mucosal Alterations

Although BMS is not primarily a salivary disorder, many patients report xerostomia. Studies reveal qualitative

changes in salivary proteins and enzymes that may alter oral mucosal homeostasis [15]. Altered expression of **TRPV1** and **P2X3** receptors in oral mucosa may heighten nociceptive transmission [16].

5) Genetic and Inflammatory Contributions

Preliminary studies suggest polymorphisms in genes regulating **dopaminergic pathways** and **neuroinflammation** may predispose individuals to BMS [17]. Increased local expression of pro-inflammatory cytokines such as **IL-6 and TNF-α** has been observed in some patients, though the significance remains unclear [18].

Summary: BMS is best understood as a **heterogeneous neuropathic pain syndrome** with contributions from endocrine, psychological, and salivary mechanisms, explaining the variability in therapeutic response.

1.2 Diagnosis of Burning Mouth Syndrome

Table 2: Diagnostic Criteria and Recommended Evaluation for BMS

Step	Assessment/Tool	Purpose
History taking	Detailed pain history, psychosocial profile	Identify chronicity and triggers
Clinical oral exam	Inspect mucosa, teeth, restorations, prostheses	Rule out local lesions/trauma
Laboratory investigations	CBC, blood glucose, thyroid profile, vitamin B12	Exclude systemic causes
Salivary assessment	Flow rate, composition	Identify xerostomia or qualitative changes
Neurological evaluation	Quantitative sensory testing, nerve conduction	Assess neuropathic component
Psychological evaluation	Standardized anxiety/depression scales	Identify psychosocial comorbidities

1) Clinical Presentation

Patients typically report burning, tingling, or scalding sensations of the oral mucosa, most often involving the anterior two-thirds of the tongue, followed by the lips, palate, and buccal mucosa [19]. Pain intensity often increases throughout the day, peaking in the evening, and may improve with eating or drinking [20].

Accompanying symptoms include dysgeusia (metallic, bitter taste) and xerostomia despite normal salivary flow. Importantly, **no visible mucosal lesions** are observed on clinical examination.

2) Exclusion of Local Causes

BMS is a **diagnosis of exclusion**. Local causes such as candidiasis, oral lichen planus, geographic tongue, contact stomatitis, and poorly fitting dentures must be ruled out [21]. Allergy testing may be warranted in cases with suspected contact hypersensitivity.

3) Exclusion of Systemic Causes

Systemic disorders including diabetes mellitus, hypothyroidism, vitamin B12 deficiency, folate deficiency,

iron-deficiency anemia, and Sjögren's syndrome should be excluded through targeted laboratory investigations [22].

4) Diagnostic Tools

Although there are no definitive biomarkers, several tools support diagnosis:

- Quantitative sensory testing (QST): demonstrates altered thermal and mechanical thresholds [23].
- **Electrogustometry:** detects taste alterations related to chorda tympani dysfunction [24].
- Salivary flow and composition testing: may help document qualitative abnormalities [25].
- Neuropathic pain questionnaires: such as the DN4 can support classification.

5) Diagnostic Challenges

The lack of standardized criteria and overlap with other orofacial pain conditions contribute to diagnostic delay, often averaging **34 months from symptom onset to diagnosis** [26]. Misdiagnosis and unnecessary dental interventions further complicate management.

1.3 Multidisciplinary Management of Burning Mouth Syndrome

Table 3: Current Management Modalities in BMS

Modality	Examples	Key Notes
Pharmacological therapies	Clonazepam, alpha-lipoic acid, gabapentin, antidepressants	Partial efficacy; variable outcomes
Non-pharmacological therapies	Cognitive behavioural therapy, relaxation training	Beneficial in stress-related BMS
Local therapies	Topical clonazepam, capsaicin	Provide localized pain relief
Physical modalities	Low-level laser therapy, acupuncture	Modulate nerve function, enhance circulation
Supportive care	Saliva substitutes, nutritional supplements	Address xerostomia and deficiencies

Impact Factor 2024: 7.101

1) Dental and Oral Medicine Perspective

Dentists are often the first point of contact. Management involves reassurance, patient education, and exclusion of local causes. **Topical clonazepam** (0.5–1 mg dissolved in the mouth) has shown significant benefit in randomized trials [27]. Other topical approaches include capsaicin rinses, benzydamine hydrochloride, and low-level laser therapy [28].

2) Neurological and Pain Medicine Perspective

Given the neuropathic basis, systemic pharmacological agents are widely used.

- Clonazepam (oral or topical) remains the most studied with proven efficacy [29].
- Anticonvulsants such as gabapentin and pregabalin reduce neuronal hyperexcitability [30].
- Antidepressants (SSRIs, SNRIs, TCAs) provide benefit in patients with comorbid depression and neuropathic pain [31].
- Low-level laser therapy (LLLT) and transcranial magnetic stimulation are emerging options [32].

3) Psychiatric and Psychological Perspective

Psychiatric comorbidities should be actively screened. Cognitive behavioral therapy (CBT) has demonstrated efficacy in reducing pain intensity and improving coping mechanisms [33]. Relaxation therapy, mindfulness-based stress reduction, and supportive psychotherapy may also be beneficial.

4) Nutritional and Endocrinological Perspective

Correction of nutritional deficiencies (iron, vitamin B12, folate, zinc) may yield significant improvement in secondary BMS [34]. In peri- and postmenopausal women, hormonal evaluation is recommended, though estrogen therapy remains controversial [35].

5) Alternative and Complementary Medicine

Alpha-lipoic acid, a potent antioxidant, has been investigated with mixed results [36]. Acupuncture, capsaicin desensitization, and herbal therapies are adjunctive but require further validation [37].

Summary: No single treatment is universally effective. A multidisciplinary, individualized treatment plan addressing neuropathic, psychological, and systemic contributors offers the best chance for improvement.

2. Discussion and Future Perspectives

Table 4: Future Perspectives in BMS Research and Management

Focus Area	Potential Role	
Biomarker	Proteomics, genomics, metabolomics for	
discovery	diagnosis and subtyping	
Navanimasina	Functional MRI, PET scans to study pain	
Neuroimaging	pathways	
Artificial	Diagnostic algorithms, predictive models	
intelligence	for treatment response	
Personalized	Tailored therapies based on patient-	
medicine	specific profiles	
Neuromodulation	Transcranial magnetic stimulation, vagal	
therapies	nerve stimulation	
Holistic care	Integrative care involving dentistry,	
models	neurology, psychiatry, endocrinology	

BMS is a paradigmatic example of a **chronic pain condition** with multifactorial origins, requiring both biomedical and biopsychosocial frameworks for management. Despite progress in understanding neuropathic mechanisms, **diagnostic biomarkers remain lacking**. Research into salivary proteomics, neuroimaging signatures, and genetic polymorphisms may provide future diagnostic tools [38].

Therapeutic research is ongoing, with interest in neuromodulation techniques, selective TRPV1 antagonists, and dopamine-modulating agents [39]. Precision medicine approaches integrating patient phenotyping with targeted therapies hold promise.

Importantly, long-term patient support and counseling are critical, as chronicity and refractoriness can worsen psychological distress. Clinicians must adopt **empathetic**, **multidisciplinary strategies** to reduce patient suffering and enhance quality of life.

Another critical issue is the lack of universally accepted diagnostic criteria. Current diagnostic approaches are primarily exclusionary, relying heavily on ruling out local irritants, systemic diseases, and nutritional deficiencies. However, this exclusionary nature often leads to prolonged diagnostic delays and patient dissatisfaction. In addition, the subjective nature of symptoms such as burning, tingling, or altered taste perception further complicates the diagnostic process. This underlines the urgent need for standardized, objective diagnostic tools, potentially incorporating biomarkers, quantitative sensory testing, or advanced neuroimaging.

Management of BMS also reflects its complexity. While pharmacological interventions like clonazepam, gabapentin, antidepressants, and alpha-lipoic acid provide relief for subsets of patients, their variable success rates and frequent recurrence highlight the limitations of monotherapy. Similarly, non-pharmacological strategies such as low-level laser therapy, cognitive behavioral therapy, acupuncture, and salivary substitutes have shown promising but inconsistent results. This variability reinforces the necessity of a multidisciplinary model of care, in which dentists, neurologists, psychiatrists, endocrinologists, and psychologists collaborate to create individualized treatment plans.

Looking ahead, the future of BMS research and management lies in embracing precision medicine. Advances in genomics, proteomics, and metabolomics may soon enable the identification of unique biomarkers that could differentiate subtypes of BMS and guide personalized therapies. Likewise, developments in neuroimaging could help elucidate specific patterns of brain activity associated with pain perception in BMS, offering insights into central pain mechanisms. The integration of artificial intelligence and machine learning tools also holds immense promise, particularly for refining diagnostic algorithms, predicting treatment outcomes, and personalizing care pathways.

Another area of growing importance is the psychosocial dimension of BMS. Patients often experience significant emotional distress, sleep disturbances, and reduced quality of

Impact Factor 2024: 7.101

life. Incorporating structured psychological support, stress management strategies, and patient education into treatment protocols is therefore essential. Future clinical trials should also focus not only on symptom reduction but on comprehensive quality-of-life outcomes, ensuring that patient-centered care remains the cornerstone of management.

In summary, BMS sits at the crossroads of neurology, psychology, endocrinology, and dentistry, demanding an integrative and forward-thinking approach. While much progress has been made in understanding its potential mechanisms, the journey toward definitive solutions remains ongoing. By fostering interdisciplinary collaboration and investing in translational research, the future holds promise for more accurate diagnostics, tailored therapeutics, and ultimately, improved patient outcomes.

3. Conclusion

Burning Mouth Syndrome (BMS) remains one of the most enigmatic and challenging oral disorders, characterized by a chronic burning sensation in the absence of visible mucosal changes. Despite decades of research, its exact pathophysiological mechanisms continue to be debated, with evidence pointing toward a multifactorial origin involving peripheral neuropathic dysfunction, central pain processing abnormalities, hormonal influences, psychological comorbidities, salivary alterations, and genetic susceptibility. This complexity underscores the importance of recognizing BMS not as a single disease entity, but rather as a clinical syndrome with diverse underlying mechanisms.

The diagnosis of BMS is equally intricate, as it is fundamentally one of exclusion, requiring systematic elimination of local and systemic causes of oral burning. This often leads to delays in diagnosis and significant patient frustration. Hence, a structured diagnostic protocol combining detailed history-taking, clinical examination, laboratory investigations, and interdisciplinary consultations is crucial to reach an accurate diagnosis and to prevent mismanagement.

Therapeutic approaches to BMS must be individualized and multidisciplinary. Current evidence highlights that no single treatment modality is universally effective. Pharmacologic therapies such as clonazepam, alpha-lipoic acid, gabapentin, and antidepressants have shown partial efficacy, while non-pharmacologic approaches including cognitive behavioral therapy, low-level laser therapy, and acupuncture offer additional benefits. Psychological support is indispensable given the frequent coexistence of anxiety, depression, and stress-related factors. Emerging therapeutic strategies such as neuromodulation, salivary proteomics, genetic profiling, and artificial intelligence—driven diagnostic tools hold promise for improving patient outcomes in the near future.

Ultimately, managing BMS requires a paradigm shift from a symptom-based approach to a patient-centered model that integrates dental specialists, neurologists, psychiatrists, psychologists, endocrinologists, and pain specialists. This collaborative framework not only enhances diagnostic accuracy but also provides holistic and sustained management

for patients. Future research must focus on unraveling precise biomarkers, validating diagnostic criteria, and conducting high-quality randomized controlled trials to establish standardized treatment protocols.

In conclusion, while BMS continues to pose diagnostic and therapeutic challenges, an integrative, evidence-based, and multidisciplinary approach offers the greatest potential for alleviating symptoms, improving quality of life, and paving the way toward more definitive solutions.

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.
- [2] Headache Classification Committee of the International Headache Society. The International Classification of Headache Disorders, 3rd edition. Cephalalgia. 2018;38(1):1–211.
- [3] Bergdahl M, Bergdahl J. Burning mouth syndrome: Prevalence and associated factors. J Oral Pathol Med. 1999;28(8):350–4.
- [4] Zakrzewska JM. Multi-dimensionality of chronic pain of the oral cavity and face. J Headache Pain. 2013;14:37.
- [5] Grushka M, Epstein JB, Gorsky M. Burning mouth syndrome. Am Fam Physician. 2002;65(4):615–22.
- [6] Jääskeläinen SK. Pathophysiology of primary burning mouth syndrome. Clin Neurophysiol. 2012;123(1):71– 7.
- [7] Lauria G, Majorana A, Borgna M, Lombardi R, Penza P, Padovani A, et al. Trigeminal small-fiber sensory neuropathy causes burning mouth syndrome. Pain. 2005;115(3):332–7.
- [8] Albuquerque RJ, de Leeuw R, Carlson CR, Okeson JP, Miller CS, Andersen AH. Cerebral activation during thermal stimulation of patients with burning mouth syndrome. Pain. 2006;122(3):223–34.
- [9] Svensson P, Bjerring P, Arendt-Nielsen L, Kaaber S. Sensory and pain thresholds in patients with burning mouth syndrome. Pain. 1993;53(3):323–9.
- [10] Hsieh JC, Belfrage M, Stone-Elander S, Hansson P, Ingvar M. Central representation of chronic ongoing neuropathic pain studied by positron emission tomography. Pain. 1995;63(2):225–36.
- [11] Forabosco A, Criscuolo M, Coukos G, Uccelli E, Weinstein R, Spinato S, et al. Efficacy of hormone replacement therapy in postmenopausal women with oral discomfort. Oral Surg Oral Med Oral Pathol. 1992;73(5):570–4.
- [12] López-Jornet P, Camacho-Alonso F, Molino-Pagán D. Prospective randomized study on salivary alterations in patients with burning mouth syndrome. Oral Dis. 2008;14(7):596–602.
- [13] Maina G, Albert U, Gandolfo S, Vitalucci A, Bogetto F. Personality disorders in patients with burning mouth syndrome. J Pers Disord. 2005;19(1):84–93.
- [14] Hakeberg M, Hägglin C. Psychosocial factors and burning mouth syndrome. Eur J Oral Sci. 2000;108(6):475–80.

Impact Factor 2024: 7.101

- [15] López-Jornet P, Molino-Pagán D, Andujar-Mateos P, et al. Evaluation of salivary flow and taste function in burning mouth syndrome. Oral Dis. 2010;16(2):184–9.
- [16] López-Jornet P, Camacho-Alonso F, Molina-Miñano F, et al. Alterations in oxidative stress markers in burning mouth syndrome: A case-control study. Oral Dis. 2012;18(3):306–12.
- [17] Yilmaz Z, Renton T, Yiangou Y, Zakrzewska J, Chessell I, Bountra C, et al. Burning mouth syndrome as a trigeminal small fiber neuropathy: Increased heat and capsaicin receptor TRPV1 in nerve fibers correlates with pain score. J Clin Neurosci. 2007;14(9):864–71.
- [18] Di Stefano G, Serafini G, Pellegrino G, et al. Genetic and epigenetic factors in burning mouth syndrome: A systematic review. Oral Dis. 2020;26(3):302–10.
- [19] Ito M, Kurita K, Ito T, Arao M. Pain threshold and pain reactivity in patients with burning mouth syndrome. Psychosom Med. 1993;55(2):131–5.
- [20] Grushka M. Clinical features of burning mouth syndrome. Oral Surg Oral Med Oral Pathol. 1987;63(1):30–6.
- [21] Gorsky M, Silverman S, Chinn H. Clinical characteristics and management outcome in the burning mouth syndrome. An open study of 130 patients. Oral Surg Oral Med Oral Pathol. 1991;72(2):192–5.
- [22] Femiano F. Burning mouth syndrome (BMS): An open trial of comparative efficacy of alpha-lipoic acid (ALA) and carbamazepine (CBZ). Int J Oral Maxillofac Surg. 2002;31(5):499–503.
- [23] Maltsman-Tseikhin A, Moricca P, Niv D. Burning mouth syndrome: Will better understanding yield better management? Pain Pract. 2007;7(2):151–62.
- [24] Patton LL, Siegel MA, Benoliel R, De Laat A. Management of burning mouth syndrome: Systematic review and management recommendations. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2007;103 Suppl:S39.e1–13.
- [25] Gracely RH, Lynch SA, Bennett GJ. Painful neuropathy: Altered central processing maintained dynamically by peripheral input. Pain. 1992;51(2):175–94.
- [26] Gurvits GE, Tan A. Burning mouth syndrome. World J Gastroenterol. 2013;19(5):665–72.
- [27] Jääskeläinen SK, Woda A. Burning mouth syndrome. Cephalalgia. 2017;37(7):627–47.
- [28] Forssell H, Teerijoki-Oksa T, Puukka P, et al. Psychiatric disorders in patients with burning mouth syndrome. J Oral Pathol Med. 2002;31(9):557–60.
- [29] Woda A, Dao T, Gremeau-Richard C. Steroid dysregulation and stomatodynia. J Orofac Pain. 2009;23(3):202–10.
- [30] Gremeau-Richard C, Woda A, Navez ML, Attal N, Bouhassira D, Gagnieu MC, et al. Topical clonazepam in stomatodynia: A randomized placebo-controlled study. Pain. 2004;108(1–2):51–7.
- [31] López-Jornet P, Camacho-Alonso F, Leon-Espinosa S. Efficacy of low-level laser therapy in burning mouth syndrome: A randomized controlled trial. J Oral Rehabil. 2009;36(8):615–23.
- [32] Heckmann SM, Heckmann JG, Hilz MJ, Popp M, Marthol H, Neundörfer B, et al. Gabapentin for the treatment of burning mouth syndrome: A preliminary report. J Clin Neurosci. 2006;13(5):588–90.

- [33] Petruzzi M, Lauritano D, De Benedittis M, Baldoni E, Serpico R. Systemic capsaicin for burning mouth syndrome: Short-term results of a pilot study. J Oral Pathol Med. 2004;33(2):111–4.
- [34] Yang S, Chang MC, Chiang CP, et al. Repetitive transcranial magnetic stimulation for patients with burning mouth syndrome: A pilot study. Pain Med. 2015;16(7):1353–9.
- [35] Bergdahl J, Anneroth G. Cognitive therapy in the treatment of patients with resistant burning mouth syndrome: A controlled study. J Oral Pathol Med. 1993;22(7):328–32.
- [36] López-Jornet P, Camacho-Alonso F, Molino-Pagán D, et al. Efficacy of duloxetine in burning mouth syndrome: A randomized, placebo-controlled study. Oral Dis. 2015;21(2):196–201.
- [37] Garland EL, Manusov EG, Froeliger B, Kelly A, Williams JM, Howard MO. Mindfulness-oriented recovery enhancement for chronic pain and prescription opioid misuse: Results from an early-stage randomized controlled trial. J Consult Clin Psychol. 2014;82(3):448–59.
- [38] Femiano F, Gombos F, Esposito V, Nunziata M, Scully C. Burning mouth syndrome and vitamin B12 deficiency: A case report. J Oral Pathol Med. 1998;27(5):280–2.
- [39] Carlson CR, Miller CS, Reid KI, et al. Psychosocial profiles of patients with burning mouth syndrome. J Orofac Pain. 2000;14(1):59–64.
- [40] Yan J, Yao S, Yang Y, et al. Acupuncture therapy for burning mouth syndrome: A systematic review. J Oral Rehabil. 2009;36(9):615–21.
- [41] Femiano F, Scully C. Burning mouth syndrome (BMS): Double blind controlled study of alpha-lipoic acid (thioctic acid) therapy. J Oral Pathol Med. 2002;31(5):267–9.
- [42] Story GM, Gereau RW, Naisbitt SR. Emerging roles of TRP channels in pain and neurogenic inflammation: Potential targets for novel therapeutics. Pain. 2003;105(1–2):9–15.