Indian Journal of Dental Sciences.
September 2012 Issue:3, Vol.:4


www.ijds.in

Review Article

Indian Journal
of Dental Sciences

E ISSN NO. 2231-2293 P ISSN NO. 0976-4003

# An Overview Of Burning Mouth Syndrome

[1] Navneet Sharma
[2] Jasjit Kaur
[3] Divye Malhotra
[4] Gopi Krishna Kolli

[1] Associate Professor,
Deptt of Oral Medicine and Radiology,
[2] Senior Lecturer,
Deptt of Prosthodonticand Crown & Bridge,
[3] Reader, Deptt of Oral and Maxillofacial Surgery,
Himachal Dental College,
Sunder Nagar, Himachal Pradesh.
[4] Reader, Deptt of Oral medicine & Radiology,
Darshan Dental College and Hospital,
Udaipur, Rajasthan, India.

**Address For Correspondence:**
Dr. Navneet Sharma, Associate Professor
Department of Oral Medicine and Radiology,
HDC, Sunder Nagar, Himachal Pradesh.

**Submission :** 05th December 2011
**Accepted :** 17th May 2012

Quick Response Code

**Abstract**
Burning mouth syndrome (BMS) is an oral dysaesthesia that causes chronic orofacial pain in the absence of a detectable organic cause. Affected patients often present with multiple oral complaints, including burning, dryness and taste alterations. Burning mouth complaints are reported more often in women, especially after menopause. Various local, systemic and psychological factors have been found to be associated with BMS, but its etiology is not fully understood. Conditions that have been reported in association with burning mouth syndrome include chronic anxiety or depression, various nutritional deficiencies, type 2 diabetes and changes in salivary function. However, these conditions have not been consistently linked with the syndrome, and their treatment has had little impact on burning mouth symptoms. BMS has been treated with drugs belonging to different pharmacological groups: antidepressants, antipsychotics, antiepileptic drugs, analgesics and mucosal protectors, among others. Although effective therapies have been identified in concrete cases, a treatment modality offering efficacy in most cases of BMS remains to be established. The purpose of this article was to provide the practitioner with an understanding of the local, systemic and psychosocial factors which may be responsible for oral burning associated with BMS, and review of treatment modalities, therefore providing a foundation for diagnosis and treatment of BMS.

**Key Words**
Burning mouth syndrome (BMS), local factors, oral burning, psychosocial factors, systemic factors antidepressants, antipsychotics.

## Introduction

Burning mouth syndrome (BMS) is characterized by burning oral mucosal pain without any visible signs of mucosal pathology. BMS can be considered a chronic pain problem, with symptoms that can last several years.[1] BMS mainly affects middle aged/ old women with hormonal changes or psychological disorders.[2] There has also been no clear consensus on the etiology, pathogenesis and treatment of burning mouth syndrome.[3] BMS is probably of multifactorial in origin, often idiopathic and it represents a disorder with very poor prognosis in terms of quality of life.[4] In literature various synonyms, such as glossodynia, sore mouth, glossopyrosis, sore tongue,oral dysesthesia, stomatodynia, stomatopyrosis has been used for description of quality and location of pain in mouth. Various studies have focused their efforts in differentiating the "true BMS" from other types of unremitting oral burning correlated to different underlying pathologies.[5] International Association for the Study of Pain (IASP) has identified BMS as a "distinctive nosological entity" characterized by "unremitting oral burning or similar pain in the absence of detectable oral mucosal changes." [5] Evidence based research has shown that large number of BMS patients suggests a common background of nerve damage in the pathogenesis of this syndrome. Based on this fact there are two clinic forms of BMS:"primary BMS", or essential / idiopathic BMS for which local or systemic causes cannot be identified and "secondary BMS" resulting from local or systemic factors and responsive to etiology based therapy. This pragmatic approach of dividing BMS into primary and secondary BMS can resolve the diagnostic dilemma of oral burning with clinical abnormality and unexplained oral burning sensation.[6]

## Epidemiology

True prevalence of BMS is difficult to establish due to lack of rigorous diagnostic criteria in many of the published series that do not distinguish between oral burning due to local and systemic factors and true BMS. Estimated prevalence varies between o.7 to 4.5%.[7] BMS is a disorder typically observed in middle-aged and elderly subjects with an age range from 38 to 78 years. Occurrence below the age of 30 is rare and the female-to-male ratio is about 7:1.[8] These differences between gender may perhaps be explained by biological, psychological and sociocultural factors; however such factors have not yet been defined. No studies exist in relation to any occupational, educational or social grouping.[7]

## Etiology

The etiopathogenesis of BMS is still unclear, and the issue has generated considerable controversy in the literature. The most debated aspect is whether BMS should be definitively considered either as a separate entity or as a symptom of different pathologies.[9, 10] Following factors have been categorized in literature as triggering events.[11]

## Local factors

Local factors associated with BMS include, but are not limited to, dental treatment, mucosal diseases, fungal infections, bacterial invasion, allergies, temporomandibular joint dysfunctions, and salivary gland abnormalities.[8] however; there is strong evidence only

for local nerve trauma, oral parafunctional habits, and salivary gland dysfunction.[10,12] Oral salivary quantity and quality have been investigated as causative factors in BMS. In one study, 34% of 150 patients evaluated, demonstrated inadequate saliva or oral dryness.[4] Irregularities in saliva metabolites as protein, potassium, and phosphate concentrations between patients with BMS and control patients have been suggested as causative factors.[13, 14] BMS symptoms may reflect a neuropathic condition possibly involving the central or peripheral nervous system, or both. Nerve injury or dysfunction resulting from oral, facial, or systemic trauma from medical conditions might be the cause of burning sensations in BMS.[1,15] Several studies report that parafunctional habits are observed in patients with BMS. This parafunctional activity (tongue thrusting, bruxism, clenching) is significantly related to anxiety, and the activity most related to a high anxiety score seems to be tooth clenching.[17] Lamey and Lamb [18] have described a lip component to the BMS symptoms. Parafunctional activity of lip licking, lip sucking, lip pressure, and mouth breathing were noted in patients with perioral symptom. Oral infections produced by diverse microorganisms have been associated with this syndrome. Infection by Candida albicans [2,19,20] has been considered one of the most frequent factors in the production of BMS, although some authors question its importance. Mucosal allergic reactions have been reported by Kaaber et al.[21] In this study, 23% of the patients demonstrated an allergic reaction to the substances in dentures and the allergy was determined the cause of BMS. Oral allergies to food and correlation to syndrome-like symptoms has been noted in the literature.[22, 23, 24]

**Systemic factors**

Several systemic factors may influence the prevalence, development, and severity of BMS symptoms. Deficiency diseases, hormonal and immunologic disturbances, and pharmacotherapuetic side effects have been implicated in producing BMS symptoms. The most significant systemic predisposing conditions for BMS are menopausal disorders, diabetes, and nutritional deficiencies.[25, 26.] There is remarkable association between BMS and peri / post menopausal stages. Onset of pain ranges from 3 years before to 12 years after menopause. [27] The most credited theory regards menopausal hormonal changes as a "master player" in BMS onset[28], although estrogen replacement therapy (ERT) does not relieve pain in many cases.[8,29] The variable response to ERT treatment may be due to either the presence/absence of the expression of nuclear estrogen receptors in oral mucosa or the possible activation of reversible/irreversible neuropathic mechanisms.[28] Some patients with BMS were found to have some clinically evident immunologically mediated disease. Antinuclear, antibody, and rheumatoid factor imbalances were noted. HIV and AIDS afflictions have also been correlated with BMS.[30.] It has been suggested that type II diabetes mellitus plays a role in BMS development8 but some studies have mentioned it to be controversial.[18, 31] A possible explanations for this controversy may be that these diabetic patients were erroneously classified as BMS.[32] A significant portion of patients studied had vitamin B1, B2, and/or B6, folic acid deficiencies.[33, 34] But vitamin replacement therapies are not efficient in reducing the symptoms of burning.[35]

**Psychogenic Factors**

Existing studies suggests psychopathologic factors may play an important role in BMS and support the multifactorial etiology, in which physical changes may interact with psychological factors.[36, 37, 38] Many of these patients have symptoms of anxiety, depression and personality disorders. It has also been demonstrated that patients with burning mouth syndrome have a greater tendency towards somatization and other psychiatric symptoms. Majority of the patients exhibit high levels of anxiety and depression as well as pain relief after suitable administrations of psychotropic drugs/medications such as anti-depressants or benzodiazepines. However there is increasing controversy as to whether depression and anxiety are primary [39] or secondary events [15] to the oral pain. Some BMS patients with psychological disorders frequently show other precipitating factors, such as masticatory muscular tensions, denture design errors, and parafunctional habits, all of which are strictly associated with anxiety and depression in these individuals.[17] These findings do not seem to support the hypothesis that BMS is primarily a psychogenic disorder. On the contrary, they draw attention to an overwhelming psychogenic component of the pain in the clinical spectrum of BMS,[40] which may result from the patients' difficulty in coping with their suffering and/or emotional distress.[41] Cancerophobia and anxiety have been documented as etiologic factors, although authors have grouped patients with BMS symptoms in a general diagnostic category of hypochondria.[33] A lower level of socialization and higher levels of somatic anxiety have been observed, so BMS can be considered a chronic pain disorder that adversely affects quality of life.[42]

**Pathogenesis**

The presence of taste changes and sensory anomalies indicate towards the fact that BMS has some neuropathic basis. Neurophysiological studies [43, 44,] suggest that the central and / or peripheral nervous system are implicated in the pain of BMS. This causes a loss of central inhibition and consequently hyperactivity of the trigeminal nociceptive pathway, which in turn carries a more intense response to oral irritants and eventually leads to the appearance of phantom oral pain as a result of this alteration in the taste system. BMS patients may show: (a) abnormal perception of intensities in the pre-pain range and disturbances in the perception of non-nociceptive and nociceptive thermal stimuli [45](b) raised trigeminal nerve sensitivity and alterations in neuronal transmission [46] and (c) disturbances of the mucosal neurovascular microcirculatory system.[47] Central neuropathic mechanisms have been demonstrated following thermal stimulation of the trigeminal nerve in patients with BMS. Patients with BMS show patterns of cerebral activity similar to those that appear in other neuropathic pain disorders, suggesting that the cerebral hypoactivity could be an important element in the pathogenesis of BMS.[48]

**Clinical Presentation**

Burning mouth syndrome has variety of chronic oral symptoms that have been described as continuous chronic discomfort, with spontaneous acute periods, with no clearly identifiable precipitating factor, except stress and other psychological factors. Two specific

clinical features define this syndrome: (1) a "symptomatic triad", which includes unremitting oral mucosal pain, dysgeusia, and xerostomia; and (2) "no signs" of lesion or other detectable change in the oral mucosa, even in the painful area. Oral pain represents the cardinal symptom of BMS. The type of pain experienced by BMS patients is a prolonged "burning" sensation of the oral mucosa, similar in intensity to, but different in quality from, that associated with toothache.[27] However, scalding, tingling, or numb feelings of the oral mucosa have also been reported. The onset of oral pain is generally spontaneous and without any recognizable precipitating factors.[49] But some patients can relate the pain to previous dental procedures or disease.[27], [49] The pain is primarily bilateral and symmetrical on the anterior two-thirds of the tongue (71% - 78%), followed by the dorsum and lateral borders of the tongue, the anterior part of the hard palate, the labial mucosa and gingiva, often appearing at several locations. Other, less frequent locations are the oral mucosa, floor of the mouth, soft and hard palate, and oropharynx. [37,43] To fulfill the diagnostic criteria for BMS, pain episodes must occur continuously for at least 4-6 months. They may last for 12 years or more with an average duration of 3.4 years. [50]. More than one clinical oral-pain pattern may occur in association with local, systemic, and/or psychogenic disorders.4 On the basis of these patterns, it has been suggested that BMS patients may be classified into three types.[51] Type 1 BMS is characterized by a pain-free waking, with burning sensation developing in the late morning, gradually increasing in severity during the day, and reaching its peak intensity by evening.[9] This type is linked to systemic disorders such as nutritional deficiency, diabetes, etc.[4] Type 2 consists of continuous symptoms throughout the day, which, once started, often make falling asleep at night difficult for many individuals.[27] This subgroup of patients often reports mood changes, alterations in eating habits, and a decreased desire to socialize, which seem to be due to an altered sleep pattern.[9,27] Common clinical findings in these subjects include parotid gland hypofunction related to the use of anti-depressant drugs.[52] Type 3 BMS is characterized by intermittent symptoms with pain-free periods during the day. Frequently, these patients show anxiety and allergic reactions, particularly to food additives.[53]. Apart from pain and burning, majority of patients of BMS has persistent taste disorders. The dysgeusic taste is most commonly bitter, metallic, or both.[26] Different alterations in taste perception appear at either threshold or suprathreshold levels.[27] Approximately 46-67% of BMS patients complain of dry mouth (xerostomia).[2,27] In these individuals, the feeling of oral mucosal dryness generally reflects a subjective sensation [54], rather than one objective symptom of salivary gland dysfunction. Subjective xerostomia in BMS patients appears to be related to psychological problems such as depression. Strongest evidence, however, suggests that either feeling or evidence of dry mouth in these subjects is more likely due to idiosyncratic side-effects from an extensive abuse of anticholinergics, such as psychotropic drugs/medications or antihistamines, and diuretics. [55,56] Majority of BMS patients show complete absence of lesions but they may have oral signs related to other associated pathological conditions. Common findings in this subgroup of patients include decreased daily usage of dentures, reduced tongue space, incorrect placement of occlusal table, and increased vertical dimension. [57]

**Management**

Before starting the treatment, it is important to inform the patient about nature of disease and give reassurance, since BMS patients are likely to have consulted numerous specialists who stated that mucosa was healthy and may be convinced that their problems are imaginary.[58] Patients must be made aware, instead, that their pain is "real", the syndrome is common in middle-aged/elderly individuals, and is often linked to some identified conditions. Patient management involves a differential diagnosis for BMS and the discrimination between "Primary BMS" and "Secondary BMS" based on the identification of possible etiologic factors for the syndrome. The most-used medications to treat this syndrome are antidepressants, antipsychotics, sedatives, antiepileptics, analgesics and oral mucosa protectors.[3] The purpose of these medications is to reduce the suffering of the patients, to bring their condition under better control and improve the quality of life.[59,60] The tricyclic antidepressants such as amitriptyline and nortriptyline at low doses are useful in BMS, although some authors contraindicate their use in patients with dry mouth as they can worsen the condition. Amitriptyline and nortriptyline can be used in doses of 10-150 mg per day. Drugs can be prescribed as 10 mg at bedtime, and further dose is increased 10 mg every 4-7 days until oral burning is relieved or side effects occur.[3] Studies have been made to evaluate the efficacy and tolerance of amisulpiride (50 mg/day) and selective serotonin inhibitors: paroxetine (20 mg/day) and sertraline (50 mg/day) in the treatment of BMS, over eight weeks, with a reasonably high efficacy (around 70%).[37,48,61] Topical use of clonazepam (¼ or ½ tablet applied 3 times each day for sucking) has shown partial to complete pain relief in most patients with idiopathic BMS , suggesting a possible local effect of this drug on gamma-amino-butyric-acid receptors (gaba-receptors) within the oral mucosa.[59] Systemic use of clonazepam(0.25-2mg/day)and chlordiazepoxide(10-30mg/day) has also been recommended in literature for symptomatic relief in BMS.[3]. An open study was conducted on 15 patients treated with gabapentin. The starting dose was 300 mg/day, and was increased at a rate of 300 mg every 48 hours to a maximum of 2400 mg/day. Authors concluded that gabapentin exerts little or no effect upon BMS.[61] A possible relationship has also been postulated between estrogen hormone imbalances (seen in menopause) and onset of the sensory alterations characterizing BMS. This subject has generated debate, due to the divergent results reported by the different studies made to assess the effects of hormone replacement therapy (HRT).[37,61,62]. Low doses of capsaicin, applied 3 or 4 times topically on the area where the pain is localized, appear to be quickly effective in alleviating the pain in BMS subjects.[63,64] However, there is a limited number of trials for corroborating its role in BMS pain control, probably because long treatment periods with topical capsaicin are thought to result in depletion of substance P (by causing C-fiber degeneration)[65], with consequent loss of pharmacological effects. Trials have also been made on rinsing with benzydamine hydrochloride at 0.15%, having an analgesic and anti-

inflammatory effect, but finding no significant efficacy, as also found with local anesthetic mouthwash such as lidocaine.[62,66] Alpha lipoic acid is a powerful neuroprotector that prevents damage to nerve cells by free radicals, it regenerates other antioxidants such as vitamins C and E, increasing levels of intracellular glutathione. Several studies suggest that alpha lipoic acid can improve the symptoms in BMS, showing that at two months, 97% of the patients treated with alpha lipoic acid (200 mg, three times a day) experienced an improvement in the symptoms.[61,67]

Patients who do not respond to any of the above treatments (resistant BMS) should undergo "cognitive"[12] or "cognitive/behavior"[68] therapies by qualified psychotherapists, since they probably have, in their BMS spectrum, a strong and complex psychogenic component of the pain. The purpose of psychodynamic therapy is to allow each patient to understand the causes of his/her symptoms. BMS patients, particularly those resistant to treatment, should be offered regular follow-up from two to four times a month during the symptomatic period. Each evaluation should include an analysis of pain levels, personality, psychological functioning, and quality of life.

**Conclusions**

The complex and multifactorial etiology of BMS necessitates collaboration between different specialists for the management of these patients. In addition, it is necessary to carry out trials with a strict agreement on protocols based on clear diagnostic criteria that exclude those cases with a medical or dental cause, as has been suggested in the literature.

**References**


1. Cibirka RM, Nelson SK and Lefebvre CA. Burning mouth syndrome: a review of etiologies. J Prosthet Dent 1997; 78:93-97.
2. Gorsky M, Silverman S Jr and Chinn H. Burning mouth syndrome: a review of 98 cases. J Oral Med 1987; 42:7-9.
3. Grushka M, Epstein J and Gorsky M. Burning mouth syndrome. Am Fam Physician 2002; 65:615-20.
4. Lamey PJ and Lamb AB. Prospective study of etiological factors in burning mouth syndrome. Br Med J 1988; 296:1243-1246.
5. Merskey H, Bugduk N, editors. Classification of chronic pain. Descriptions of chronic pain syndromes and definitions of pain terms. In: Task on taxonomy. Seattle: IASP 1994: Press, p. 74.
6. Balasubramaniam R, Klasser GD and Delcanho R. Separating oral burning from burning mouth syndrome: unraveling a- diagnostic enigma. Australian Dental Journal 2009;54:293-99.
7. Lopez-Jornet P, Camacho-Alonso F, Sanchez-Sites M and Gomez-Garcia F. Burning mouth syndrome: Update. Med Oral Patol Oral Cir Bucal 2010;15(4):e562-8
8. Basker RM, Sturdee DW and Davenport JC. Patients with burning mouths. A clinical investigation of causative factors, including the climacteric and diabetes. Br Dent J 1978;145:9-16.
9. Grinspan D, Fernandez Blanco G, Allevato MA and Stengel FM. Burning mouth syndrome. Int J Dermatol 1995;34:483-487.
10. Zakrzewska JM. The burning mouth syndrome remains an enigma. Pain 1995;62:253-257
11. Jaaskelainen SK, et al. Role of the dopaminergic system in chronic pain-a fluorodopa-PET study. Pain 2001; 90:257-260.
12. Bergdahl J, Anneroth G and Perris H .Cognitive therapy in the treatment of patients with resistant burning mouth syndrome: a controlled study. J Oral Pathol Med 1995;24:213-215.
13. Niedermeier W, et al. Significance of saliva for the denture-wearing population. Gerodontology 2000; 17:104-118
14. Glick D, et al. Relation between idiopathic glossodynia and salivary flow rate and content. Int J Oral Surg 1976;5;161-65
15. Gruska M and Sessle BJ. Burning mouth syndrome. Dent Clin North Am 1991; 35:171-84.
16. Lehman C, Bartoshuk L, Catalanotto F, Kveton J and Lowlicht R. Effect of anesthesia of the chorda tympani nerve on taste perception in humans. Physiol Behav 1995;57:943-951
17. Paterson AJ, Lamb AB, Clifford TJ and Lamey PJ. Burning mouth syndrome: the relationship between the HAD scale and parafunctional habits. J Oral Pathol Med 1995; 24:289-292.
18. Lamey PJ and Lamb AB. Lip component of burning mouth syndrome. OralSurg Oral Med Oral Pathol 1994:78:590-3.
19. Gorsky M, Silverman Jr and 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; 72:192-5.
20. Brooke RI and Seganski DP. Etiology and investigation of the sore mouth syndrome. Dent J 1977; 43:504-6.
21. Dutree-Meulenberg RO, Kozel MM and van Joost T. Burning mouth syndrome: a possible etiologic role for contact hypersensitivity. J Am Acad Dermatol 1992; 26:935-40.
22. Antico A. Oral allergy syndrome induced by chestnut (Castanea sativa ) Ann Allerg Asthma Immunol 1996:76:37-40.
23. Lamey PJ, Lamb AB and Forsvt UA. Atypical burning mouth syndrome. Contact Dermatitis 1987:17:242-3.
24. Lamev PJ, Lamb AB. Hughes A, et al. Type 3 burning mouth svndrome: psychological and allergic aspects. J Oral Pathol Med 1994:23:216-9.
25. Bergdahl J and Anneroth G. Burning mouth syndrome: literature review and model for research and management. J Oral Pathol Med 1993; 22:433-438.
26. Ship JA, Grushka M, Lipton JA, Mott AE, Sessle BJ and Dionne RA. Burning mouth syndrome: an update. J Am Dent Assoc 1995; 126:842-853.
27. Grushka M, Sessle BJ and Miller R. Pain and personality profiles in burning mouth syndrome. Pain 1987; 28:155-167.
28. Forabosco A, et al. Efficacy of hormone replacement therapy in postmenopausal women with oral discomfort. Oral Surg Oral Med Oral Pathol 1992; 73:570-574.
29. Wardrop RW, Hailes J, Burger H and Reade PC. Oral discomfort at menopause. Oral Surg Oral Med Oral Pathol 1989;67:535-540
30. Grushka M, Shupak R and Sessle BJ. A rheumatological examination of 27 patients with burning mouth syndrome. J Dent Res 1986;26:533
31. Mott AE, Grushka M and Sessle BJ. Diagnosis and management of taste disorders and burning mouth syndrome. Dent Clin North Am 1993; 37:33-71.
32. Tourne LP and Fricton JR. Burning mouth syndrome. Critical review and proposed clinical management. Oral

Surg Oral Med Oral Pathol 1992; 74:158-167
33. Main DMG and Basker RM. Patients complaining of a burning mouth. Further experience 'n clinical assessment and management. Br Dent J 1983;154:206-11.
34. Lamey PJ, Hammond A, Allam BF and Mclntosh WB. Vitamin status of patients with burning mouth syndrome and the response to replacement therapy. Br Dent J 1986;160:81-4.
35. Hugoson A and Thorstensson B. Vitamin B status and response to replacement therapy in patients with burning mouth syndrome Acta Odontol Scand 1991;49:367-375.
36. Grushka M, Ching V and Epstein J. Burning mouth syndrome. Adv Otorhinolaryngol 2006; 63: 278-87.
37. Klasser GD, Fischer DJ and Epstein JB. Burning mouth syndrome: recognition, understanding, and management. Oral Maxillofac Surg Clin North Am. 2008;20:255-71
38. Rojo L, Silvestre FJ, Bagan JV and de Vicente T. Prevalence of psychopathology in burning mouth syndrome. Oral Surg Oral Med Oral Pathol. 1994; 78: 312- 6.
39. Pokupec-Gruden JS, Cekic-Arambasin A and Gruden V. Psychogenic factors in the aetiology of stomatopyrosis. Coll Antropol 2000; 24(Suppl 1):119-126.
40. Nicholson M, Wilkinson G, Field E, Longman L and Fitzgerald B. A pilot study: stability of psychiatric diagnoses over 6 months in burning mouth syndrome. J Psychosom Res 2000; 49:1-2.
41. Jerlang BB. Burning mouth syndrome (BMS) and the concept of alexithymia-a preliminary study. J Oral Pathol Med 1997; 26:249-253.
42. López- Jornet P, Camacho-Alonso F and Lucero-Berdugo M. Quality of life in patients with burning mouth syndrome. J Oral Pathol Med. 2008;37:389-94.
43. Fedele S, Fricchione G, Porter SR and Mignogna MD. Burning mouth syndrome (stomatodynia).QJM. 2007;100:527-30.
44. Grushka M, Epstein JB and Gorsky M. Burning mouth syndrome and other oral sensory disorders: a unifying hypothesis. Pain Res Manag 2003;8:133-5.
45. Svensson P, Bjerring P, Arendt-Nielsen L and Kaaber S. Sensory and pain thresholds to orofacial argon laser stimulation in patients with chronic burning mouth syndrome. Clin J Pain 1993;9:207-215.
46. Gao S, Wang Y and Wang Z. Assessment of trigeminal somatosensory evoked potentials in burning mouth syndrome. Chin J Dent Res 2000;3:40-46.
47. Heckmann SM, et al. Oral mucosal blood flow in patients with burning mouth syndrome. Pain 2001; 90:281-86.
48. Brufau-Redondo C, Martín-Brufau R, Corbalán-Velez R and de Concepción-Salesa A. Síndrome de la boca urente.Actas Dermosifiliogr 2008;99:431-40.
49. Tammiala-Salonen T, Hiidenkari T and Parvinen T. Burning mouth in a Finnish adult population. Community Dent Oral Epidemiol 1993;21:67-71
50. Browning S, Hislop S, Scully C and Shirlaw P. The association between burning mouth syndrome and psychosocial disorders. Oral Surg Oral Med Oral Pathol 1987;64:171-174.
51. Lamey PJ and Lewis MA. Oral medicine in practice: burning mouth syndrome. Br Dent J 1989; 167:197-200.
52. Lamey PJ, Murray BM, Eddie SA and Freeman RE. The secretion of parotid saliva as stimulated by 10% citric acid is not related to precipitating factors in burning mouth syndrome. J Oral Pathol Med 2001; 30:121-124.
53. Lamey PJ, Lamb AB, Hughes A, Milligan KA and Forsyth A. Type 3 burning mouth syndrome: psychological and allergic aspects. J Oral Pathol Med 1994; 23:216-219.
54. Bergdahl M and Bergdahl J. Burning mouth syndrome: prevalence and associated factors. J Oral Pathol Med 1999; 28:350-354.
55. Glass BJ. Drug-induced xerostomia as a cause of glossodynia. Ear Nose Throat J 1989; 68:776, 779-781.
56. Bergdahl M and Bergdahl J. Low unstimulated salivary flow and subjective oral dryness: association with medication, anxiety, depression, and stress. J Dent Res 2000;79:1652-58.
57. Svensson P and Kaaber S. General health factors and denture function in patients with burning mouth syndrome and matched control subjects. J Oral Rehabil 1995;22:887-895.
58. Lamey PJ. Burning mouth syndrome: approach to successful management. Dent Update 1998; 25:298-300.
59. Woda A, Navez ML, Picard P, Gremeau C and Pichard-Leandri E. A possible therapeutic solution for stomatodynia (burning mouth syndrome). J Orofac Pain 1998; 12:272-278.
60. Maina G, Vitalucci A, Gandolfo S and Bogetto F. Comparative efficacy of SSRIs and amisulpride in burning mouth syndrome: a single-blind study. J Clin Psychiatry 2002; 63:38-43.
61. Mínguez-Serra MP, Salort-Llorca C and Silvestre-Donat FJ. Pharmacological treatment of burning mouth syndrome: A review and update. Med Oral Patol Oral Cir Bucal 2007;12:E299-304.
62. Patton LL, Siegel MA, Benoliel R and 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.
63. Epstein JB and Marcoe JH. Topical application of capsaicin for treatment of oral neuropathic pain and trigeminal neuralgia. Oral Surg Oral Med Oral Pathol 1994;77:135-140.
64. Scala A, Marini I, Vecchiet F and Checchi L. Diagnostic procedure and supportive care in Burning Mouth Syndrome (abstract). J Dent Res 2003;82 (Spec Iss C) (in press).
65. Simone D and Ochoa J. Early and late effects of prolonged topical capsaicin on cutaneous sensibility and neurogenic vasodilatation in humans. Pain 1991;47:285-294.
66. Zakrzewska JM, Forssell H and Glenny A. Interventions for the treatment of burning mouth syndrome: A systematic review. J Orofacial Pain 2003;17:293-300.
67. López- Jornet P, Camacho-Alonso F and Leon- Espinosa S. Efficacy of alpha lipoic acid in burning mouth syndrome: a randomized, placebo-treatment study. J Oral Rehabil 2009;36:52-7.
68. Humphris GM, Longman LP and Field EA. Cognitive-behavioural therapy for idiopathic burning mouth syndrome: a report of two cases. Br Dent J 1996;181:204-208.

**Source of Support** : Nill, **Conflict of Interest** : None declared