BMS Support Group

Burning Mouth: A Review for Sufferers

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I. Introduction

Burning mouth syndrome (BMS) is an enigmatic, idiopathic, chronic, often painful, clinical entity for which there are yet to be well established standardized validated definitions, diagnostic criteria or classifications. Interestingly, it was first described in the scientific literature by Fox [1] in 1935 and Gilpin [2] in 1936. The various names that have been applied to BMS has created much confusion as this condition has been given many different labels often based upon the quality and/or location of its intraoral pain presentation. Some of the names applied are as follows: glossodynia, glossopyrosis, glossalgia, stomatodynia, stomatopyrosis, sore tongue, burning tongue, scalded mouth syndrome, oral dysesthesia, burning mouth condition and burning mouth syndrome. [3, 4] Unfortunately, the use of these multiple terms has only lead to confusion and uncertainty within the scientific literature and in clinical practice regarding this condition. Overall, BMS is likely more than one disease process with a multifactorial etiology thereby making it a diagnosis of exclusion.

Because of the various definitions and multiple labels applied to BMS it is easy to comprehend the frustration experienced by the patient's and difficulties encountered by the practitioner in evaluating these individuals. This is so because the patient is experiencing continuous burning pain in the mouth while the practitioner is struggling to identify any obvious clinical signs even with the accompaniment of additional diagnostic testing or imaging. This

often produces a dilemma for the majority of practitioners when developing and presenting a definitive diagnosis.

II. Epidemiology

The condition is most commonly reported in postmenopausal women, generally in the fifth to sixth decade of life. Men may also develop BMS with a reported ratio of approximately 1:5 to 1:7 compared to women, depending on the study population. 5, 6 Prevalence appears to increase with age in both men and women. 7 There has been only one study conducted on the prevalence of BMS in relation to ethnicity 8 and the literature is devoid of studies reporting the prevalence of BMS by social, educational or occupational groups.

III. Diagnostic criteria

Over the years there have been several formal diagnostic criteria applied to BMS. Table 1 provides the proposed diagnostic criteria used to identify BMS. Even though there are similarities among some components of these criteria there is no absolute consensus nor has there been validation of any specific criteria.

IV. Classification

There have been several attempts at developing an "ideal" classification system for BMS. It seems the most practical approach in classifying BMS patients is by dividing patients into either primary (essential/idiopathic) BMS (no other evident disease) or secondary BMS (oral burning from other clinical abnormalities). [4] Since secondary BMS is associated with a preexisting condition or cause, it should be remembered that once such a condition is treated the symptoms would either improve or disappear.

V. Clinical signs and symptoms

The clinical presentations of BMS are typically not consistent and will vary from patient to patient. Patients often describe their oral symptoms with the following words: painful, burning, tender, tingling, hot, scalding and numbness yet sometimes the sensation is merely described as discomfort, raw and annoying. BMS is characterized by both positive (burning pain, altered taste and uncomfortable sensation) and negative (taste loss and abnormal sensation) sensory symptoms. [9] The burning is mainly located bilaterally, and symmetrically in the anterior two thirds of the tongue (71% to 78%) followed by the dorsum and lateral borders of the tongue (72%), the anterior aspect of the hard palate (25%) and the labial mucosa of the lips (24%) while often occurring in multiple sites. [10-14] Other less commonly reported sites include the buccal mucosa, floor of the mouth, hard and soft palate and the throat (36%). [12] Approximately 50% of BMS patients experience a spontaneous onset of symptoms without any identifiable triggering factor. [15, 16] However, about 17% to 33% of the patients attribute the onset of their symptoms to a previous illness such as an upper respiratory tract infection, previous dental procedure or medication use (including antibiotic therapy) [16-19] suggesting the possibility of neurologic alterations preceding the onset of burning in some patients. [20-22] Other individuals claim the onset of symptoms relate to traumatic life stressors. [13, 15, 16] Typically, the symptoms occur continuously for months or years without periods of cessation or remission [16] with some reports suggesting an average duration of 2 to 3 years. [23, 24] There have been reports [16] of complete/partial remission (with or without intervention) in approximately 50% of patients and a complete spontaneous remission in approximately 20% of patients within 6 to 7 years of onset. The remission of symptoms, be it complete or partial, are often characterized by a change in pain pattern from a constant to an episodic form. [16, 25]

The pattern of daily symptoms is reportedly constant with fluctuation in pain intensity and with approximately one third of patients experiencing symptoms both day and night. [10, 13, 26] Most patients report minimal symptoms upon awakening, after which the symptoms gradually increase during the day while becoming more aggravating toward the evening. [10, 27] About one third of the patients have difficulty with sleep onset and some may awaken during the night due to the burning pain. [13, 28] The intensity of the burning pain has been described as moderate to severe and in some cases it is comparable to the intensity of toothache pain in regards to severity but not quality. [29] In the majority of patients, the burning sensation intensifies in the presence of personal stressors, fatigue and with the intake of hot/spicy/acidic foods and in about half the patients, the intake of food or liquids and distraction seem to reduce or alleviate the symptoms. [9, 10, 30, 31] BMS patients have a significantly higher incidence of dry mouth, thirst and taste disturbances but they do not differ from healthy controls regarding changes in oral mucosa or dental problems. [10, 15, 32, 33] BMS patients have more non-specific health complaints and more severe menopausal symptoms as compared to healthy controls. [10]

VI. Etiology and Pathophysiology

Currently, the etiology for primary BMS has remained largely unknown. The presumed etiology is best explained as being multifactorial involving the interaction between biological (neurophysiological mechanisms) and psychological factors. [34] Even though multiple local (physical, chemical or biological), systemic and psychological factors have been found related to BMS, several of these factors should be considered as conditions important to the differential diagnosis of oral burning rather than as etiological factors implicated in BMS.

The pathophysiology of BMS continues to be unclear especially with the lack of any visible oral mucosal changes on examination. Current suggested pathophysiology is

multifactorial and encompasses changes to taste, changes in hormone levels, nerve damage, central nervous system changes, autoimmune disorders and psychological factors.

a. New findings: Pain sensitive channels and neuropeptides

Transient receptor potential vanniloid channel type 1 (TRPV-1), a member of a family of nonselective cation channels, is involved in the transmission of pain. [35] In BMS, it is significantly elevated in the papillae of the tongue but not on the epithelium. [35, 36]

The voltage gated sodium channel 1 and 8 (Na_v 1, 8), another ion channel associated with pain sensation, has also been found to be slightly elevated in the subepithelial region of the tongue in BMS. [35] Both TRPV-1 and Na_v 1, 8 are under the regulation of nerve growth factor (NGF) which has been found to be elevated in BMS in both subepithelium and basal tongue epithelium. Since NGF is not produced by the nerve fibers, the elevation in NGF may be the result of increased uptake into a decreased population of remaining nerve fibers. [35]

NGF, synthesized in the areas of the trigeminal distribution, the dental pulp and the cornea [37] supports the growth and maintenance of pain neurons during development and interacts with immune cells such as mast cells to release inflammatory mediators. [38] Substance P is a neuropeptide released by nerve fibers and is an indication of nerve fiber presence. Salivary examination of the presence of neuropeptides in BMS has found elevated NGF but decreased substance P [38] suggesting loss of normal nerve fiber with regeneration and maintenance of pain sensitive fibers. [37]

b. New findings: Deficient dopamine inhibition

Autonomic nervous function studies including deep breathing heart rate, heart rate variability and sympathetic skin responses have shown that some BMS patients have changes in these various activities. [39, 40] These findings are similar to those found in Parkinson disease, a cerebral

degenerative disease associated with dysfunction of the dopamine system, with up to 40% of Parkinson patients reporting burning in the oral cavity. [40] In some BMS patients, positron emission tomography (PET) studies have demonstrated a reduction in dopamine in certain regions of the brain. [41] In fact, several cases have been reported in which BMS has been successfully treated with levodopa [42] supporting the possible involvement of the dopamine system in at least a subpopulation of BMS.

c. New findings: Autoimmune disorder (lichen planus)

Oral burning, which arises from peripheral small fiber neuropathy, can be caused by autoimmune disorders such as Sjogren's syndrome, lupus erythema, inflammatory bowel disease, sarcoidosis and fibromyalgia. [43] Burning can also be a complaint in oral lichen planus, an immunologically medicated mucocutaneous disease. [44] Burning can also occur as a result of a delayed contact sensitivity reaction, with 67% of burning mouth patients showing a positive reaction. [45] Some therefore suggest a connection between activation of the immune system and development of neuropathy leading to burning pain. The exact mechanism of how this damage occurs is currently unknown.

VII. Diagnosis

BMS has for many years remained a diagnosis of exclusion. When mucosa changes are evident, clearly they must be addressed and ruled out to ensure that the burning pain is not the result of a disorder such as lichen planus or fungal infection. Once the mucosal tissues are returned to their normal state, and if the burning persists, the presence of burning mouth pain associated with BMS is suggested.

Testing for burning mouth pain may include studies of salivary flow, taste function, blood tests to rule out systemic factors, contact sensitivity, as well as a clinical history which suggests pain that is usually reduced by stimulation and function.

VIII. Management

Distinct recommendations for the management of BMS patients are somewhat lacking in the literature. From a clinical perspective, the clinician must initially determine if the patient is experiencing signs and symptoms consistent with primary (essential/idiopathic) BMS or secondary BMS in which symptoms are due to underlying local or systemic conditions. [46] Secondary BMS requires appropriate diagnosis and treatment of the underlying condition(s). In primary BMS, the etiology is unclear, so management options are based upon patients' symptomatology.

Often management involves a multidisciplinary team approach, often requiring multiple modifications of the management plan until an effective protocol is achieved. The importance of this approach cannot be overstated as often patients are frustrated by a lack of understanding of this condition among health practitioners. Currently, the clinician has the choice of three approaches or combinations thereof as considerations in management.

a. Behavioral strategies

Behavioral strategies to be considered consist of self help measures such as cessation of parafunctional habits (clenching, bruxism, tongue protrusion) and/or modification of oral care product usage, such as alcohol-free mouthwashes, and the use of products without flavoring agents or irritating components (cinnamic aldehyde, sodium lauryl sulfate, tooth whitening agents, anti-calculus ingredients).[47, 48] Other products for consideration in discontinuing their use include mints, gum or other breath aids.[49] Stress management approaches such as moderate

exercise regimens, yoga, and tai chi may be attempted. Additionally, desensitizing appliances may be considered to reduce oral burning and it may also be used as a habit-breaking appliance. [50] Behavioral strategies utilizing professional assistance include cognitive behavioral approaches (focuses on how beliefs and thoughts influence behavior) used alone or in combination with other therapies and/or group psychotherapy have shown efficacy in decreasing pain intensity. [51-53]

b. Topical therapies

Topical therapies utilizing the following:

- anxiolytics (clonazepam - dissolve and expectorate or oral rinse);
- anesthetics (lidocaine - viscous gel, bupivacaine - lozenge);
- antidepressants (doxepin - cream);
- atypical analgesics (capsaicin - cream);
- non-steroidal anti-inflammatory (benzydamine - not FDA approved for use in the USA);
- antimicrobials (lysozyme-lactoperoxidase - oral rinse);
- mucosal protectants (sucralfate - oral rinse, aloe vera and/or lycopene virgin oil);
- herbal supplement: (chamomile - gel);
- immunosuppressant: (cyclosporine - oral rinse);
- low level laser therapy;

have all been trialed with various rates of success.

3. Systemic therapies

Systemic approaches employing a vast number of medications from various medication categories include:

- antidepressants (amitriptyline, imipramine, nortriptyline, desipramine, trazodone, paroxetine, sertraline, duloxetine, milnacipran);
- anxiolytics (clonazepam, diazepam, chlordiazepoxide);
- anticonvulsants (gabapentin, pregabalin, topiramate);
- antioxidants (alpha lipoic acid);
- atypical analgesics/antipsychotics (capsaicin, olanzipine: amisulpride, levosulpride – both medications are not FDA approved for use in the USA);
- histamine receptor antagonists (lafutidine - not FDA approved for use in the USA);
- monoamine oxidase inhibitors (moclobemide - not FDA approved for use in the USA);
- salivary stimulants (pilocarpaine, cevimiline);
- dopamine agonists (pramipexole);
- herbal supplements (hypericum perforatum or St. John's wort, Catuama);
- vitamin supplementation (Vitamin B, C);
- artificial sweetener (sucralose);
- transcranial magnetic stimulation;
- acupuncture.

Since 2005, there have been five systematic reviews reported in the scientific literature which have exclusively reported on interventions used in the management of BMS. [46, 54-57] Unfortunately, there is only a minor consensus among these specifc systematic reviews as to the thrapy of choice and rankings thereof to guide the clinician in providing an evidence based appraoch for management of this condition. Table 2 provides a summary of the recommendations provided by each of these systematic reviews.

Table 1. Proposed diagnostic criteria to identify BMS.

Source, Year	**Criteria**
Fortuna G et al., [58] 2013	1. Any type of oropharyngeal symptom that can be persistent or intermittent with possible phases of remission/exacerbation during the day; 2. Absence of any clinically and instrumentally detectable oropharyngeal lesion; 3. Absence of any type of local and/or systemic factors such as oral diseases, drugs, trauma, hypersensitivity reactions, physical/chemical agents. Additionally but not mandatory: State of being symptomatic is persistent (typically ≥ 3 months).
ICHD-III beta, [59] 2013	A. Oral pain fulfilling criteria B and C. B. Recurring daily for >2 hours per day for >3 months. C. Pain has both of the following characteristics: 1. burning quality. 2. felt superficially in the oral mucosa. D. Oral mucosa is of normal appearance and clinical examination including sensory testing is normal. E. Not better accounted for by another ICHD-3 diagnosis.
International Association for the Study of Pain, [60]1994, revised 2011	Burning tongue or other parts of oral mucosa, usually bilateral, dysgeusic taste and/or altered taste perception, dry mouth and denture intolerance.
Scala A et al., [4] 2003	1.Daily deep burning sensation of oral mucosa (bilateral); 2. Pain is unremitting for at least 4-6 months; 3. Continuous throughout all or almost all the day; 4. Seldom interferes with sleep; 5. Characteristic symptoms are not getting worse/ sometimes there may be an improvement over the ingestion of food and liquid Additional "inclusion symptomatic criteria" are: 6. Occurrence of other oral symptoms, such as dysgeusia and/or xerostomia; 7. Presence of sensory/chemo-sensory anomalies; 8. Presence of mood changes and/or specific disruption(s) in patient personality traits.

Table 2. Recommendations from BMS systematic reviews (since 2005)

Author, Year	# of studies reviewed	Study design	First line therapy evidence	Alternative therapy
Zakrzewska JM et al., [54] 2005	9 studies	RCT + controlled clinical trials	Insufficient evidence: analgesics, hormones or antidepressants Behavioral: CBT Topical: clonazepam Systemic: alpha-lipoic acid.	
Patton LL et al., [46] 2007	10 studies	Meta-analyses, systematic reviews, RCT + crossover studies	**Recommendations based upon RCT** Behavioral: CBT Topical: clonazepam Systemic: SSRI (paroxetine, sertraline), Antipsychotics (amisulpride)	**Suggestions based upon expert opinion + common clinical practice but not yet evaluated** Topical: capsaicin, doxepine, lidocaine Systemic: TCA, SNRI, Anticonvulsants, Opioids, Benzodiazepenes (clonazepam, alprrazolam)
de Moraes M et al., [55] 2012	12 studies	RCT	Topical: capsaicin, clonazepam Systemic: alpha-lipoic acid.	
Ducasse D et al., [56] 2013	16 studies	RCT	Behavioral: Psychotherapy***** Topical: clonazepam, tongue protector Systemic: clonazepam, SSRI (paroxetine, sertraline)	Systemic: Antipsychotics (amisulpride), catuama (mixture of guarana, catuaba, ginger, and muirapuama) Interventional: lingual nerve block
Kuten-Shorrer et al., [57] 2014	12 studies Note: evaluated the placebo effect in BMS	RCT	The mean placebo response as a fraction of drug response over 10 studies was 72%, suggesting a robust placebo response	Two studies reported no improvement between active intervention + placebo

RCT: Randomized controlled trials
CBT: Cognitive behavioral therapy
SSRI: Selective serotonin reuptake inhibitor
SNRI: Selective norepinephrine reuptake inhibitor
TCA: Tricyclic antidepressant
*****only if Hospital Anxiety and Depression (HAD) score is high

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