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## Burning Mouth Syndrome

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### Abstract

Burning mouth syndrome (BMS) has been considered an enigmatic condition because the intensity of pain rarely corresponds to the clinical signs of the disease. As a result, BMS patients have variously been labelled as depressed, anxious or hypochondriacal and have often been underserved by the medical and dental communities. Recently, there has been a resurgence of interest in this disorder with the discovery that the pain of BMS may be neuropathic in origin and originate both centrally and peripherally. This chapter discusses some of our recent understandings of the etiology and pathogenesis of BMS as well as the role of pharmacotherapeutic management in this disorder.

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Burning mouth syndrome (BMS) is variously referred to as glossopyrosis, glossodynia (when the burning occurs on the tongue only) and syndrome of oral complaints as well as numerous other monikers, although all refer to the same or a similar constellation of symptoms. It is usually described as oral burning pain, sometimes with dysesthetic qualities similar to those present in other neuropathic pain conditions with the absence of clinical and laboratory abnormalities. The dorsal tongue, palate, lips and gingival tissues, individually or in combination, are the most common sites involved. Symptoms are usually bilateral, but can be unilateral as well. In some reports, oral burning pain has been found to be associated with jaw pain [1, 2], taste changes and subjective dry mouth, geographic and fissured tongue [3], painful teeth, loss of a comfortable jaw position, uncontrollable jaw tightness [4–8], headache [5, 9], neck and shoulder pain, increased parafunctional activity, difficulty speaking, nausea, gagging and swallowing difficulties [4].

Although the events preceding the onset of BMS are often not identified, the condition has been reported to follow dental treatment, antibiotic usage and a severe upper respiratory infection [9, 10].

The pain from BMS is constant, progressively increases over the day, and usually decreases during eating. Although it may interfere with onset of sleep, it rarely wakes the patient at night and is at its lowest intensity in the morning [9]. Patients, who are frequently distressed by their unremitting symptoms, may demonstrate psychological abnormalities including anxiety and depression in both questionnaire and psychiatric examinations [11–13]. The presence of emotional issues in BMS appears to be in accord with studies which have demonstrated psychological profiles of distress in the presence of chronic pain [14]. The lack of pathology to account for the pain can be equally frustrating [5, 15].

### **Prevalence**

Epidemiological surveys have reported a prevalence rate of between 0.7–2.6% with an NIH survey estimating close to 1 million burning mouth sufferers in America. Although most prevalent amongst postmenopausal women, men and women of any age can also be affected [16].

### **Differential Diagnosis**

Alternate causes of oral burning pain should be ruled out, including both systemic and peripheral pathology (table 1), before a diagnosis of BMS is entertained. Burning pain can indicate a previously undiagnosed systemic condition. This includes anemia, vitamin B or iron deficiency, untreated diabetes, renal disease, and connective tissue disorders such as Sjögren's syndrome and systemic lupus – both of which can be associated with oral dryness and consequent candidal infections. Some medications such as angiotensin-converting enzyme inhibitors have been reported to be associated with burning pain [10].

Local changes within the oral cavity may cause burning pain and include allergic reactions to dental materials and dentures, and products such as toothpastes, mouth rinses and food constituents such as cinnamon. Candidiasis infection in susceptible patients and painful lesions in the mucosa may also be both causative and treatable. In one small study of the role of viral infection in BMS, 22 subjects complaining of burning mouth were assessed; of these, 9 were

**Table 1.** Differential diagnosis of BMS

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*a Systemic causes*

Nutritional deficiency: vitamin B, iron, zinc

Allergy: food or dental materials

Esophageal reflux disorder

Uncontrolled diabetes

Acoustic neuroma

Central changes including multiple sclerosis, Parkinson's disease, trigeminal neuralgia

Autoimmune disorders: Sjögren's syndrome

*b Local causes*

Oral candidiasis infection

Poorly fitting dentures, restorations

Lichen planus and other oral vesiculobullous conditions

Dry mouth: autoimmune disorders, medication

Viral infection: herpes simplex, herpes zoster

Trauma to lingual or mandibular nerve following dental surgery

Oral inflammatory condition: geographic, fissured tongue

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diagnosed with BMS and the rest were found to have mucosal changes especially erosive lichen planus [17]. While low salivary flow can coexist with BMS and may exacerbate the pain, there is no indication at this time that xerostomia by itself is a primary causative factor [10].

## **Diagnosis**

History taking is the key to diagnosis of BMS. Both diagnosis and management may be difficult because patients often present with multiple oral complaints, may be focused on their symptoms and may be anxious or depressed, which intensifies the pain experience. The diagnosis is based on clinical characteristics, including either a sudden or intermittent onset of pain, bilateral presentation, a progressive increase in pain during the day and the remission of pain with eating (although some foods may intensify the pain) and sleeping. Salivary flows and taste function should be assessed [18]. Important clinical questions are presented in table 2. Neurological imaging and consultation should be considered when patients present with a more complex symptom array, including both sensory and motor changes, to rule out a neurodegenerative disorder such multiple sclerosis, Parkinson's disease, and stroke.

**Table 2.** Clinical features that are helpful in the diagnosis of BMS

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Unilateral or bilateral burning pain localized to tongue, palate, lips and gingival
Pain that gets worse over the day
Decreased pain on eating
Decreased pain with sleep
Absence of clinical finding
Presence of abnormal or dysgeusic tastes, usually metallic, bitter or sour
Complaint of dry mouth in presence of normal flows
Sensory changes or parasthesias including complaints of areas of roughness or irritation

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**Table 3.** Clinical tests that may be helpful

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Hematological tests: CBC, glucose, nutritional factors, autoimmune panel
Oral cultures for fungal, viral or bacterial infections if suspected
MRI to rule out central changes, especially if pain is unilateral, atypical or does not respond to medication
Salivary flows for unstimulated and stimulated whole saliva (<1.5 ml/0.5 min, unstimulated; <4.5 mg/5 min stimulated)
Salivary uptake scan if low salivary flows and Sjögren's syndrome suspected
Allergy testing, if needed, especially to dental panel of allergens
Removal of possibly offending medication including angiotensin-converting enzyme inhibitors

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Considerations in differential diagnosis, diagnostic testing, and clinical history are outlined in tables 1–3.

## **Etiology**

Although many etiologies have been suggested in BMS, spanning the range from nutritional factors to dental intolerances, none have been found to account for a substantial portion of patients. Some of the more recent considerations have been the possibility that BMS is a neuropathic disorder as a result of damage to the taste system, possibly by viral infection.

### *Viral Infection*

In view of the relatively quick onset of burning and dysesthetic pain, the relatively high prevalence of BMS (see above), and the previous demonstration that herpesvirus can lead to neuropathies following oropharyngeal infection [19], a possible association between BMS and herpes viral damage was evaluated in a recent study. In this study, 22 subjects complaining of oral burning pain were

**Table 4.** Percentage of positive Ig findings in serologic tests of the BMS and control groups

	n	HSV, %		CMV, %		HZV, %	
		IgM	IgG	IgM	IgG	IgM	IgG
BMS	9	0	66.7	0	66.7	0	100
Control	13	0	66.7	0	66.7	11.1	88.9

HZV = Herpes zoster virus.

assessed for viral serology, 9 of whom were diagnosed with BMS and the rest were found to have oral mucosal changes and were used as control subjects.

The results of the serologic tests for herpes simplex virus (HSV), cytomegalic virus (CMV) and varicella-zoster virus (VZV) are presented in table 4. No IgM seropositivity for any of the 3 viruses was seen in most patients. All but 1 subject in both the study and control groups were negative for IgM antibody to the herpesviruses tested except for 1 patient with pain compatible with herpes zoster who was found to be positive for varicella-zoster virus antibody. Most subjects in both groups were positive for HSV, CMV and herpes zoster virus IgG and no significant difference was found in the prevalence of the positive findings between the two groups. Although no evidence was found in this preliminary study that would support the presence of an active or past viral infection in BMS subjects, the possibility of a ‘hit and run’ model for viral damage in BMS cannot be ruled out.

#### *Taste Changes in BMS*

Work by Bartoshuk et al. [20] has demonstrated the convergence of taste sensation and pain clinically and experimentally. The chorda tympani nerve leaves the tongue with the lingual nerve (cranial nerve V) and travels through the pterygomandibular space. The inferior alveolar nerve, which conveys sensation from the lower teeth, also passes through the same space. Often, dental anesthesia of the inferior alveolar and lingual nerves required for dental restorations abolishes touch and pain, but also taste on the injected side. The chorda tympani and lingual nerves separate and the chorda tympani passes through the middle ear. Bartoshuk et al. [20], Lehman et al. [21] and Yanagisawa et al. [22] have demonstrated that anesthesia of the chorda tympani behind the tympanic membrane intensifies tastes from the area innervated by the glossopharyngeal nerve at the back of the tongue on the opposite side,

supporting a model of central inhibition between the chorda tympani and glossopharyngeal nerves. According to Bartoshuk and fellow workers, reduction of input into the central nervous system from one taste nerve releases inhibition of other taste.

Tie et al. [23] found that anesthesia of the chorda tympani can intensify pain induced by capsaicin on the contralateral anterior tongue suggesting the presence of central inhibitory interactions between taste and oral pain. Furthermore, the intensification of pain was found to be related to an individual's genetic ability to taste PROP (6-n-propylthiouracil), with the greatest intensification found in 'supertasters' who report the most bitter sensation from PROP testing [24].

Based on these taste/pain interactions, it is believed that BMS could also be the clinical manifestation of taste damage, either to the chorda tympani, with release of inhibition in the glossopharyngeal nerve (taste alterations, alterations in touch and pain) or the trigeminal nerve (touch and pain changes). Consistent with this model, severe taste damage has been found in many BMS patients. Notably, the intensity of the peak oral pain was also found to correlate with the density of fungiform papillae and patients with BMS were primarily supertasters [25]. Furthermore, it has also been suggested that interactions between taste and oral pain were not limited to BMS but involved other orofacial pain complaints as well. For instance, patients with atypical odontalgia (pain appearing to originate from healthy teeth) showed taste damage [26].

It should also be noted that although we are not aware of similar studies linking taste and inhibition of the motor component of the trigeminal nerve, based on reports of increased bruxism in BMS patients [27], as well as increased headaches in BMS [9], the possibility that taste also inhibits the motor component of the trigeminal nerve, leading to muscle hyperactivity in the mastication system, is being considered. The anatomical substrate for this inhibition is known to be present in animal studies which demonstrate projections from the gustatory portion of the nucleus of the solitary tract to the oromotor nuclei in the medulla subserving the masticatory muscles [28].

Other abnormalities have also been noted in BMS, including elevated thresholds for temperature and touch [29], altered pain tolerance [30] as well as changes in blink reflex, corneal reflex, jaw jerk, sensory neurography of the inferior alveolar nerve and trigeminal somatosensory evoked potentials [31]. There may also be an increase in sympathetic output which leads to decreased blood flow [32] in the tongue, altered salivary composition [18, 33], high blood pressure, difficulty sleeping and increased esophageal reflux [9].

A hypothesis based on the taste pathways inhibiting other cranial nerves can explain why conditions such as BMS and AO can often encompass sensory,

motor and sympathetic abnormalities; why multiple orofacial phenomena are linked together in these pain syndromes; why the onset of these problems can occur suddenly; why there is almost always a lack of associated organic mucosal and dental pathology, and why these conditions may respond to centrally acting drugs, especially those affecting the GABAergic pathways [2, 34, 35] which are involved in taste transmission and in neuroinhibition [28].

GABA is known to be an inhibitory neurotransmitter found in the taste system [36–38] and may be a key target. According to Bartoshuk et al. [20], if taste damage produces a sufficient loss of the inhibition normally exerted on central structures mediating oral pain, then replacement of a GABA agonist such as clonazepam might ameliorate the loss of inhibition and relieve the pain in BMS.

Interestingly, GABA agonists such as clonazepam [39] have also been found to have value in the treatment of nausea, coughing and hiccups [40, 41] and in taste disturbances when associated with BMS [34]. Thus, it is possible that the inhibition produced by the taste system is important in controlling other anatomy associated with eating.

## Management

Therapy for BMS involves the use of centrally acting medications for neuropathic pain, such as tricyclic antidepressants, benzodiazepines or gabapentin [42]. Studies support the use of low-dose (0.25–0.75 mg) clonazepam or tricyclic antidepressants (10–40 mg), including amitriptyline, desipramine, nortriptyline, imipramine and clomipramine. Clonazepam is a benzodiazepine used either topically or systemically [1, 34, 35], which appears to have excellent efficacy in the relief of the symptoms related to BMS. In view of only partial or lack of response in some BMS patients taking these medications, other GABA receptor-acting anticonvulsants have been used in combination with clonazepam with apparent success [43]. Topical medications, including clonidine and capsaicin, may be considered for application to local sites. Systemic use of capsaicin has also been suggested [44] as has  $\alpha$ -lipoic acid with or without psychotherapy [45].

### *Polypharmacy in Pain Management*

A recent retrospective study of low-dose anticonvulsant medications used in combination for the management of BMS was carried out. Patients were prescribed up to 0.5 mg clonazepam and asked to add as needed up to 1,200 mg of gabapentin (up to 300 mg 4 times a day); 30 mg of baclofen (in 3 divided doses) and then up to 200 mg of lamotrigine (in 2 divided doses) as needed and pain scores were recorded on a modified adjectival/visual analogue scale. Of the

**Table 5.** Drug combinations used by patients with average doses

	C	G	C/G	C/G/B	L + other	L
Patients	11	4	12	4	4	2
Average dose, mg	0.25	300	0.27/290	0.44/300/25	n/a	50

B = Baclofen; C = clonazepam; G = gabapentin; L = lamotrigine.

45 patients who were diagnosed with BMS and tried the protocol, 1 patient reported an increase in pain after using the protocol and 6 patients did not find any difference; the rest [38] observed some reduction in pain. The average maximum pain rating before treatment was 60.6 and the average maximum pain rating after treatment was 32.1, which was found to be significant ( $p < 0.001$ ) (table 5).

The most common adverse effect reported with the medication protocol was drowsiness followed by dizziness and perceived changes in mood. Eighteen patients reported some side effects at some point of the treatment, and the majority of them were able to resolve the side effects by titrating down the dose of medication. Only 2 patients elected to stop treatment completely because of the side effects.

These results suggest that treatment of BMS may be efficacious with a combination of medications rather than higher doses of a single medication, especially with regard to controlling adverse effects.

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