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Review Article

BURNING MOUTH SYNDROME-A REVIEW

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A B S T R A C T

Burning mouth syndrome is characterized by a painful burning or stinging sensation affecting the tongue or other areas of the mouth without obvious signs of an organic cause on physical examination. A burning mouth sensation can occur in several cutaneous or systemic diseases that must be ruled out prior to making a diagnosis of burning mouth syndrome, since this term is used exclusively to refer to idiopathic forms and is included within the cutaneous sensory disorders. In most cases, patients with burning mouth syndrome have accompanying psychological or psychiatric conditions. Consequently, the syndrome has traditionally been included among the psychogenic dermatoses. However, it is currently unclear whether psychological factors are a cause or a consequence of the syndrome, or whether each exacerbates the other. Recent studies propose the etiology to be neurologic, either neuropathic or related to taste.

INTRODUCTION

Burning mouth syndrome (BMS) is currently defined as a condition in which burning pain in the tongue or other oral mucous membranes occurs in association with normal signs and normal laboratory findings. Although there is no clear understanding of pathogenesis of BMS, recent concepts are dramatically affecting the manner in which clinicians conceptualize this disorder and the way in which these patients are managed.(1) Other synonymous terms are orodynia, glossodynia, glossopyrosis, stomatodynia, stomatopyrosis, and oral dysesthesia

BMS is classified as a cutaneous sensory disorder, a term first used by Koo and Gambla1 to denote situations in which the patient presenting with cutaneous sensory disturbances, such as burning, itching, or stinging, has no apparent lesions that would justify such symptoms. This group of disorders also includes vulvodynia, coccydynia (affecting the anus), burning feet, scalp dysesthesia or red scalp syndrome, and notalgia paresthetica. In some cases, oral and genital dysesthesias have been reported in the same patient.(2,3) Authors of recent studies have proposed a neurologic etiology, either neuropathic or related to taste.

Epidemiology

Based on the makeup of most studies published to date, oral burning appears to be most prevalent in postmenopausal women. It has been reported in 10 to 40 percent of women presenting for treatment of menopausal symptoms. 4)

Etiology and Pathogenesis

The etiology and pathogenesis of BMS are poorly understood and probably multifactorial. We will now discuss a number of factors that have been studied in recent years and may play a role in the development of this syndrome.

Xerostomia

As age increases, salivary flow decreases. Xerostomia is a concomitant symptom in patients with BMS, prevalence varying between 34 and 39%, while Grushka et al. (5) found that this is equal to or greater than 60%. In contrast, some authors consider that the composition of the saliva could play a major role in the pathogenesis of BMS, indicating the importance of the identification and characterization of low molecular weight proteins. A significant increase has been found in levels of sodium, total protein, lysozyme, amylase and immunoglobulins in patients with BMS. Nagler *et al* 6 suggested that these findings indicate a salivary-related local neuropathic mechanism.

Infections

Adler *et al* 7 studied 124 patients with different gastric diseases; 46 had burning sensations with halitosis and tongue hyperplasia, and 78 had other diseases unrelated to BMS. *Helicobacter pylori* in the oral mucosa of 86% of patients who complained of a burning tongue sensation, halitosis, and tongue hyperplasia. By contrast, this microorganism was only

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isolated in 2.6% of the patients who did not report oral symptoms.

Neurologic Abnormalities

The alterations in taste perception and tolerance to pain as a possible cause of the burning sensation. Taste is located fundamentally on the fungiform papillae, finding in certain patients with burning mouth, above all women, an elevated number of said papillae, these individuals being denominated 'supertasters'. This theory proposes that certain people, labeled as supertasters due to the high density of fungiform papillae present on the anterior part of the tongue, are more susceptible to developing burning mouth pain. Supertasters are principally women, and are able to perceive the bitter taste of a substance called PROP (6-n-propiltiouracil (8).

Psychological factors

Studies exist that suggest that psychopathologic factors may play an important role in BMS and support the multifactorial etiology, in which physical changes may interact with psychological factors (9).

Many of these patients have symptoms of anxiety, depression and personality disorders, and it has been demonstrated that patients with burning mouth syndrome have a greater tendency towards somatization and other psychiatric symptoms. Cancerphobia can be present in up to 20-30% of these patients. A lower level of socialization and higher levels of somatic anxiety have been observed, as well as muscular tension, a higher tendency to worry about health and greater sadness. BMS is considered a chronic pain disorder that adversely affects quality of life

Clinical Aspects

The symptoms have been described as continuous chronic discomfort, with spontaneous acute periods, with no clearly identifiable precipitating factor, except stress and other psychological factors. The pain is primarily bilateral and symmetrical on the anterior twothirds 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 locations are the oral mucosa, floor of the mouth, soft and hard palate, and oropharynx. The location of the pain does not seem to affect the course of the disease or the response to treatment. In more than half the patients the symptoms appear spontaneously with no identifiable trigger factors. Approximately 17% to 33% of patients attribute the initiation of the symptoms to a previous condition, such as infection of the upper respiratory airway, dental procedure, or the use of medications. Other patients relate the appearance of symptoms directly with stress (10).

The oral burning sensation usually increases progressively during the day, reaching a maximum intensity at the end of the afternoon / early evening, pain being absent during the night in the majority of patients. Patients do not normally awaken during the night, but do find it difficult to get to sleep. These patients often present mood changes, including irritability, anxiety and depression. The majority of studies describe the coexistence of oral burning with other symptoms, such as dry mouth, dysgeusias, metallic taste, bitter taste or combinations thereof, and/or changes in intensity of taste perception. In addition, dysphagia and atypical facial or dental pain may appear. Experience shows that what the patient defines as 'oral burning' can be identified by diverse sensations. Although the burning or stinging sensation can exist alone, other disorders of oral perception may appear, either alternatively or simultaneously, such as pruritus, roughness, 'sticky sensation', dysphagia, stinging, burning, irritation of the lingual papillae, metallic taste and other dysgeusias, sensation of bad breath, intolerance to prostheses that would include an infinity of subjective perceptions difficult to describe (Table 1).

Clinical factors of BMS

PAIN	
Description	
Intensity	Variable, with peaks of intensity
Pattern	Continuous, no paroxysm
Location	Independent of nerve pathway. Frequently bilateral and symmetric
Pain during sleep	Infrequent
Other symptoms	Dysgeusia and xerostomia
Signs /symptoms	Absence of evident clinical signs Sensory / Chemosensory disorders Psychological profile may be implicated

Diagnosis 8,9

The following steps are required in the diagnosis of BMS:

1. Rule out systemic diseases and conditions that present symptoms similar to those of BMS, such as Sjögren syndrome, diabetes, candidiasis, and iron, folate, zinc, orvitamin B deficiencies
2. Rule out skin diseases, both those that are visible and those that are not so immediately obvious, such as galvanism and contact eczema. Patch tests should be used, especially in patients with intermittent symptoms (test for allergy to chrome or other substances used in dental prosthesis, and to food additives, preservatives, and fragrances) (Table 1).

Detailed Medical History

It is essential to obtain a complete medical, dental, and psychological history, to quantify the sensation of pain on a linear scale from 0 to 10, and to record the characteristics, duration, and timing of symptoms as well as the relationship between insertion of any prosthesis and onset of symptoms.

Examination of the Oral Mucosa

The oral mucosa should be examined carefully to rule out the presence of skin lesions, such as erythema, erosions, depapillation, or any changes characteristic of lichen planus, fissured tongue, geographic tongue, etc. The presence of any of these signs would invalidate the diagnosis of BMS. It should be remembered that autoimmune blistering diseases often begin with disorders that affect the oral mucosa.

Odontological Examination

An odontological examination should be carried out to ascertain whether the patient has any dental problems. This should include revision of any prothesis, occlusion of prosthesis, the likelihood of oral galvanism, and the volume of salivary flow.

Laboratory Tests

The workup should include a complete blood count, blood sugar, iron, serum ferritin, folates, vitamin B12, zinc, and

serology for Sjögren syndrome and H pylori infection. A culture for Candida species must also be ordered. The sample should be taken from the oral mucosa or the palate rather than the dorsum of the tongue, since the results from that area can be deceptive.

Psychiatric Assessment

A psychiatric or psychological assessment should be carried out, particularly when the patient reports a significantly high intake of anxiolytics. Anxiety or depression are observed in 62% of patients with BMS.

Diagnosis of Burning Mouth Syndrome
Detailed medical, dental, and psychological history
Medication
Examination of mucosa
Odontological examination
Patch tests for metals, prothesis, and food products (additives, preservatives, fragrances)
Bacteriologic and mycologic culture
Tongue biopsy, hematoxylin-eosin, immunohistochemistry
Laboratory tests: complete blood count, blood sugar, iron, folates, vitamin B, zinc, serology for Sjögren syndrome
Gastroenterologic examination. Helicobacter pylori test
Psychiatric/psychological assessment

Treatment

Treatment is symptomatic. The remedies used to treat other painful neuropathic abnormalities are also useful in BMS

Treatment of Burning Mouth Syndrome
Symptomatic
Spend time, empathy
Inform the patient. Address cancer phobia
Topical treatment
Capsaicin, Tabasco sauce
Sialagogues
Systemic treatment
Low doses of tricyclic antidepressants
Selective serotonin reuptake inhibitors
Dual-action antidepressants
Antipsychotics
Benzodiazepines
Gabapentin 300-1600 mg/d (start with 100 mg)
Alpha-lipoic acid 600 mg/d
Cognitive-behavioral therapy

Topical Treatment 10

Topical capsaicin has been used as a desensitizing agent in BMS and other disorders characterized by pain and pruritus, but the treatment is not well tolerated by some patients because of its flavour. The mechanism involved is based on the inhibition of substance P. A mouth rinse made of Tabasco sauce mixed with water can be useful in these patients,38 or alternatively one made of hot pepper and water in a dilution of between 1:2 and 1:1.16 Treatment with systemic capsaicin is currently being investigated. Petruzzi *et al* confirmed the efficacy of systemic oral capsaicin 0.25%, but reported a high level of gastric toxicity. Sialagogues are useful when the patient has dry mouth. Another topical treatment used in the form of a mouthwash is benzydamine hydrochloride 0.15% applied 3 times a day, but the efficacy of this regimen has not been shown to be significant.

Systemic Treatment

Tricyclic Antidepressants 11

Low doses of amitriptyline and nortriptyline owe their usefulness in BMS more to the antinociceptive properties of the tricyclics than to their antidepressant effect, and consequently antidepressant doses are not necessary.
Amitriptyline at a dose of 25 to 50 mg/d raises the threshold of sensitivity. The starting dose should be 5 to 10 mg/d at bedtime, and this should be increased by 5 or 10 mg each week until symptoms disappear or side effects occur. After 8 weeks, the dose will have reached 40 mg/d, a regimen that usually provides good results. In some cases, a dose as high as 150 mg/d may be necessary.16 Some authors contraindicate this drug in patients with dry mouth because it could aggravate that condition. Nortriptyline proved useful in a case of stomatodynia associated with penodynia/scrotodynia in which other antidepressants (venlafaxine) had proven ineffective, probably because of the greater efficacy of the tricyclics in controlling neuropathic pain.

Serotonin Reuptake Inhibitors 11

Serotonin reuptake inhibitors have proved useful in some cases of BMS, but not in others.2,16 However, it seems clear that these drugs are of some use, particularly in patientswith depression, and they are better tolerated than certain other antidepressants because they have no anticholinergic effects, in particular dry mouth.41

Dual-Action Antidepressants

Among the dual-action antidepressants-drugs that inhibit both serotonin and noradrenaline-duloxetine is particularly useful at a dose of 30 to 60 mg/d.

Antipsychotics
Risperidone is very effective at a dose of 0.5 mg a day (C. Koblenzer, personal communication).
Benzodiazepines 12

Benzodiazepines are useful, particularly in patients with anxiety disorders. They are effective at low doses, especially in young people. Alprazolam at 0.25 to 2 mg/d is useful. Treatment should be started at 0.25 mg and be increased by 0.25 mg every week until the maximum dose is reached. However, this drug is highly addictive because of its short half-life, and this makes benzodiazepines, which have a longer half-life, preferable. Treatment with low doses of clonazepam have yielded good results in the treatment of BMS pain, probably because it disrupts the underlying neuropathologic mechanism rather than because of any anxiolytic effect.13 Gruska et al16 recommend starting clonazepam at a dose of 0.25 mg/d at bedtime, and increasing the dose by 0.25 mg every 4 to 7 days in 1 full dose or 3 divided doses until symptoms disappear or side effects occur.

In a few cases, benzodiazepines have been associated with the onset of BMS.

Gabapentin 12

The dosage of gabapentin should be established either in combination with benzodiazepines or alone and should range from 300 to 1600 mg/d. Treatment should be started with a dose of 100 mg/d at bedtime and this should be increased by 100 mg/d every week. As dosage increases the medication should be taken in 3 divided doses. The efficacy of this regimen may not be apparent until after at least 1 month of treatment.

Hormone Replacement Therapy 13

Authors who have studied the use of hormone replacement therapy in the treatment of BMS report an improvement in symptoms, especially with tibolone after 3 months of treatment.49

Alpha Lipoic Acid 14,15

Alpha lipoic acid is a powerful neuroprotective agent that limits free radical damage to nerve cells, regenerates other antioxidants, such as vitamins C and E, increases levels of intracellular glutathione, and stimulates the production of nerve growth factors. It also protects membranes by interacting with vitamin C and glutathione, which in turn recycles the vitamin. Thanks to its antioxidant activity, alpha lipoic acid significantly reduces symptoms in most patients with idiopathic dysgeusia and reduces symptoms of peripheral neuropathy in patients with diabetes. Femiano *et al* also reported a significant improvement in BMS symptoms after 2 months of treatment with alpha lipoic acid 600 mg/d in a controlled double-blind study of 60 patients. The improvement was maintained at 1 year in 70% of the patients, a result that supports the hypothesis of a neuropathic etiology for BMS. In another study, the authors found that the improvement occurred particularly in patients not previously treated with tranquilizers, who had a better response than patients previously treated with psychotropic drugs. This finding suggests that the origin of the oral symptoms of BMS may be different in these 2 groups of patients.Patients taking alpha lipoic acid must be prescribed concurrent gastric protection medication.

Psychological Treatment 16

Cognitive-behavioral therapy appears to reduce the intensity of symptoms after a period of 6 months. It is difficult to evaluate the efficacy of the different therapies used because the studies in the literature are not sufficiently uniform in terms of patient selection criteria. Some of these studies enrolled only patients with idiopathic disease (BMS) but others are not comparable because the authors also included patients with similar oral symptoms secondary to systemic causes.48 The complex and multifactorial etiology of BMS makes collaboration between various different types of specialists, crucial in the management of these patients.

CONCLUSION

BMS continues to pose a challenge for those involved in the care of these patients-dermatologists, dentists, and ear, nose, and throat specialists being the physicians most often consulted in these cases (apart from general practitioners). New findings that have emerged during the last few years shed light on the etiology and pathogenesis of BMS and point to a probably neuropathic origin. However, additional studies with strict diagnostic criteria are necessary to allow us to draw reliable conclusions about the etiology and pathogenesis of this syndrome, the real role played by psychological factors, and appropriate treatment (about which very little data is currently available), all of which will help us become more useful to our patients.

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