Burning mouth syndrome: a review and update

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Summary. Burning mouth syndrome (BMS) is mainly found in middle aged or elderly women and is characterized by intense burning or itching sensation of the tongue or other regions of the oral mucosa. It can be accompanied by xerostomia and dysgeusia. The syndrome generally manifests spontaneously, and the discomfort is typically of a continuous nature but increases in intensity during the evening and at night. Although BMS classically has been attributed to a range of factors, in recent years evidence has been obtained relating it peripheral (sensory C and/or trigeminal nerve fibers) or central neuropathic disturbances (involving the nigrostriatal dopaminergic system). The differential diagnosis requires the exclusion of oral mucosal lesions or blood test alterations that can produce burning mouth sensation. Patient management is based on the avoidance of causes of oral irritation and the provision of psychological support. Drug treatment for burning sensation in primary BMS of peripheral origin can consist of topical clonazepam, while central type BMS appears to improve with the use of antidepressants such as duloxetine, antiseizure drugs such as gabapentin, or amisulpride.

Key words. Burning mouth syndrome. Clinical management. Glossodynia. Oral pain. Pain treatment. Stomatodynia.

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Introduction

Burning mouth syndrome (BMS) is a chronic and complex disorder mainly found in middle aged or elderly women, and is characterized by burning or itching sensation or other oral dysesthesias such as gritty sensation or bothersome mucosity on the oral mucosa. However, clinical examination reveals no anomalies, and the syndrome is not accompanied by laboratory test alterations [1]. According to the criteria established by the International Headache Society (ICHD-II), BMS is classified among the central causes of facial pain, and is defined as a spontaneous burning and painful sensation manifesting on the oral mucosa in the absence of exploratory findings or other identifiable local or systemic causes [2].

The syndrome can be accompanied by dysgeusia (strange taste in the mouth) and/or subjective dry mouth sensation or xerostomia. The discomfort is typically present from the time the patient gets up in the morning, though in many cases the intensity of the symptoms increases during the day, becoming particularly bothersome in the evening and at night – though usually without producing sleep disturbances [3].

Burning mouth syndrome is clearly more frequent in women than in men (proportion 7-9:1), and usually manifests between age 50-70 years. The syndrome is very rare in people under 35 years of age, and has never been reported in children or adolescents. The prevalence of BMS varies greatly depending on the literature source (because of differences in the diagnostic criteria used), though most studies describe a range of 0.6-15%. The estimated mean prevalence in the general population is 3.7% [4].

The main symptom (burning sensation) is typically continuous, non-paroxysmal and located on both sides of the tongue (tip and edges) – though the lips, palate, cheek mucosa or entire mouth can also be affected [1,5,6].

Patients with BMS usually suffer psychological problems, with a profile similar to that of other chronic pain patients. Disorders such as anxiety or depression are frequent [7-10], and the patients may experience mood changes, obsessive concern about cancer, and emotional instability [11,12].

Burning mouth syndrome is important due to the prevalence it reaches in women, and when the symptoms persist over time they can have a strong impact upon patient quality of life. The present review offers an update on the most important aspects of BMS, such as its etiopathogenesis, diagnostic criteria, and current management strategies.

Etiopathogenesis

Burning mouth syndrome has been related to local, systemic and psychological factors [13-15], though the underlying etiopathogenic mechanisms remain unclear – possibly because this is a complex syn-

drome comprising a number of different subtypes. In this regard, a distinction must be made between primary (essential or idiopathic) BMS, in which no local or systemic organic alterations capable of explaining the process are identified [16], and secondary BMS, in which the patient discomfort can be attributed to identifiable oral mucosal lesions or known causes, with symptoms improvement following treatment of the latter [17].

Primary BMS

Although no direct cause can be found in primary BMS, in recent years many studies have produced evidence of a possible neuropathic mechanism – though there is controversy over whether the mechanism is of a peripheral or central nature. As long ago as 1987, Grushka et al [18] recorded lesser heat sensitivity on the tip of the tongue than on the skin of the lip in BMS patients. Fomaker et al [19] in turn reported a decrease in taste sensation in these patients compared with the healthy controls. These same authors described a decrease particularly in salty and sweet taste sensation in BMS, with no alterations in acid or bitter taste sensation.

However, the increased heat sensation threshold also appears to be related to an affective-emotional component. Jääkeläinen et al [20] reported a direct association to trigeminal nerve excitation-inhibition with alteration of the palpebral reflex in patients with BMS.

In addition to these perceptive disorders, morphological alterations of the anterolateral mucosa of the tongue have been described in patients with BMS, characterized by a low density of fine subepithelial sensory fibers, which has been associated to axonal degeneration at this level [17]. Such patients have also shown an increase in neural growth factor in saliva [21]. Possible chorda tympani dysfunction has been proposed [22], and taste stimulation has been found to produce a decrease in burning sensation. This would explain why most patients with BMS experience some symptoms relief at mealtimes [23].

On the other hand, there is evidence of a possible central neuropathic mechanism in primary BMS. Grushka et al [24] suggested possible hyperactivity of the motor and sensory system of the trigeminal nerve, followed by central inhibition secondary to gustative damage at chorda tympani and/ or glossopharyngeal nerve level.

There have been reports of diminished somatosensory and taste perception in the tongue of BMS patients [25]. Authors such as Alburquerque et al [26] used functional magnetic resonance imaging of the thalamus to demonstrate a difference in pattern among BMS patients following pain and heat stimuli.

Jääkeläinen et al. [27] used positron-emission tomography (PET) with fluorodopa as radiotracer to reveal a decrease in tracer uptake in the putamen among BMS patients, and postulated the existence of a nigrostriatal dopaminergic system disorder in such individuals.

Grémeau-Richard et al [28] in turn have recorded a heterogeneous response to lingual nerve block with lidocaine in BMS patients. These data suggest that there might be three subclinical types of BMS [29]: a) patients with peripheral neuropathy characterized by alteration of the fine sensory fibers (representing approximately 50% of all cases); b) patients with subclinical trigeminal neuropathy (representing 20-25% of the cases); and c) patients with dopaminergic central descending inhibitory deficiency (representing 20-40% of the cases).

Secondary BMS

Many situations can give rise to symptoms similar to those of primary BMS, but with the presence of an identifiable direct cause. The causal factors in turn can be classified as local, systemic or psychopathological (Table).

Local factors are very common in such cases and require careful evaluation. They include local irritation as well as parafunctional habits that can produce marginal tongue marks or nibbling lesions [30]. On the other hand, these manifestations are very typical of individuals who experience intense stress and anxiety.

Other local causal factors are allergic reactions, galvanism secondary to the dental therapeutic use of different metals in the mouth, and local infections such as oral candidiasis [31,32].

Situations such as geographic tongue have also been associated to BMS [33], in view of their greater prevalence in patients with this syndrome – especially males. Geographic tongue is characterized by the presence of atrophic plaques on the dorsal surface of the tongue, surrounded by leukoedema, and which vary from one day to the next.

Certain systemic processes or diseases likewise have been related to BMS, such as vitamin B complex, folic acid, iron or zinc deficiencies. Chronic anemia can give rise to similar symptoms on the dorsal surface of the tongue, producing a degree of atrophy with disappearance of the papillae [34]. Certain endocrine disorders such as hypothyroid-

ism, diabetes mellitus or hormone depletion during and after menopause have also been implicated [35,36], in the same way as certain gastrointestinal and urogenital disorders [37].

Pekiner et al [38] observed increased serum proinflammatory cytokine levels (particularly IL-2 and TNFα) in patients with BMS. These same authors [39] studied certain ions and cytokines in saliva – no differences being found with respect to the controls, apart from a comparatively higher concentration of IL-6 in the BMS group.

Psychopathological factors have long been associated to BMS, in view of the high incidence of psychiatric disorders in patients of this kind, and due to the clinical benefits obtained with psychotherapy and psychoactive drugs in BMS [40,41]. The most frequent mental problems include current or past depression, and anxiety disorders, though hypochondriac conditions and cancer phobia are also common [42].

Clinical manifestations and diagnosis

The typical patient with BMS is a peri- or postmenopausal woman with burning sensation, itching, numbness or pricking sensation on the tongue (normally in the anterior two-thirds), lips (more on the lower lip than on the upper lip) and/or other oral mucosal zones. These symptoms can be accompanied by taste alterations (dysgeusia), characterized by a metallic taste, and dry mouth sensation (xerostomia). The main symptoms tend to be bilateral, though in rare cases they may be unilateral [43,44].

The onset of the syndrome is usually spontaneous, though in many cases the symptoms may appear after a concrete event which the patient takes to be a triggering factor, such as dental treatment or the administration of a new medication. The discomfort tends to be persistent throughout the day, of a nonparoxysmal nature, and gradually increases in intensity towards the evening and nighttime – though usually without producing sleep disturbances [5].

In many cases the patient has psychological alterations with irritability and anxiety, though other subjects can have antecedents of depression.

The diagnosis of primary BMS remains a challenge for the clinician, since there are no sufficiently objective or universally accepted diagnostic criteria. Standardization would be needed in order to allow comparison of the results of the different studies on the subject [45]. In practice, the diagnosis is usually established after ruling out other possible local or systemic causes, based on an exhaustive oral examination, the laboratory test findings and the patient profile and symptoms [46]. Scala et al [5] proposed a series of diagnostic criteria based on the clinical findings. In many cases certain laboratory test and hematological determinations are required [47], such as vitamin B_{12}, folic acid, ferritin, glucose, TSH, T4, LH, FSH, anti SS-A/anti SS-Ro, anti SS-B/anti SS-La, rheumatoid factor, or ANA.

Imaging techniques are rarely indicated, but may prove useful for identifying the specific causes of

Table. Processes that can give rise to secondary burning mouth syndrome.

Local factors	Mechanical and irritative factors	
	Intraoral galvanism	
	Parafunctional habits	
	Oral infections	Bacterial
		Viral
		Fungal (candidiasis)
	Allergic reactions	
	Poorly fitting dentures	
	Oral mucosal alterations	Oral lichen planus
		Geographic tongue
Systemic factors	Serum deficiencies	Group B vitamins
		Ferritin
		Folic acid
		Zinc
	Anemia	
	Gastrointestinal diseases	
	Urological diseases	
	Endocrine diseases	
	Xerostomia and hyposialia	
	Certain drugs	
Psychopathological factors	Depression	
	Anxiety	

secondary BMS. In this sense, thyroid gland ultrasound exploration can be used if macroscopic thyroid lesions are suspected.

Treatment

The clinical management and effective treatment of BMS have not been clearly established [48]. A number of studies have found that some patients can benefit from certain treatment modalities, but there is no unified and effective protocol applicable to all patients. This may be related to the fact that the natural history of BMS has not been clearly established, since there is a lack of longitudinal cohort-based trials, studies with a sufficient number of patients, and adequate placebo effects [49]. There are also methodological problems in assessing the efficacy and safety of the different treatment regimens. Likewise, the different patient subtypes must be defined in accordance to clearly established etiopathogenic criteria.

The spontaneous resolution of the symptoms of BMS is rarely observed (perhaps in less than 20% of the cases) [1]. Regarding treatment efficacy, older patients or individuals with longer evolving BMS are more refractory and difficult to treat [50].

Considering the available evidence on BMS, oral mucosal irritation and trauma should be avoided, with adequate anxiety control and the provision of psychological support. Irritative parafunctional habits such as teeth clenching or forcing of the tongue against the teeth are to be avoided, and prominent or cutting palatal and lingual cusps should be polished. López-Jornet et al [30] have designed a plastic dental protector that avoids direct tongue irritation caused by parafunctional movements. These authors recommend the combination of a gel containing aloe vera, due to its antiinflammatory effects. Topical lubricants have also been used to lessen the discomfort [51].

On the other hand, cognitive therapy has been shown to be useful in reducing anxiety and discomfort in patients with BMS [52].

The aforementioned measures can be followed by drug treatment to control the pain or the predominant symptom, as well as the associated manifestations. In this regard we have a number of topical products such as peripheral desensitizers or clonazepam, as well as drugs administered via the oral route such as alpha-lipoic acid, antiseizure drugs and antidepressants.

A number of substances administered via the topical route have been used to control burning sensation, such as capsaicin at a concentration of 0.025-0.075% applied 2-3 times a day [53-55]. This drug competes for the VR_1 vanilloid receptors, inhibiting the synthesis and transport of substance P – though it produces clinical intolerance in about 30% of the patients.

Although most of the described treatments have been associated to improvement of the symptoms in studies versus a control group, complete symptoms resolution has only rarely been reported. On the other hand, studies involving placebo have also described symptoms improvement.

Clonazepam is the drug substance with the largest body of supporting evidence in terms of symptoms improvement in patients with BMS [56]. It has been used at an oral dose of 0.75 mg at bedtime and 0.25 mg after lunch, though it is more commonly used as a topical medication in the form of a 0.5 mg/5 mL oral rinse applied during 5 minutes, 2-4 times a day. The response is satisfactory in 61% of the cases, and the medication is relatively safe and well tolerated. Clonazepam has also been used in the form of tablets that are dissolved in the mouth, taking care not to swallow the saliva. This benzodiazepine exerts an effect upon the peripheral GABA receptors, inhibiting pain transmission, and is postulated to suppress neural hyperactivity in cases of deafferentiation.

Clonazepam is not always effective, however, and the success of treatment possibly depends on the level of the neuropathic disturbance. The suggested predictors of clonazepam efficacy are the severity of the initial symptoms and the presence of accompanying discomfort such as dry mouth and dysgeusia [57].

In addition to clonazepam, other second-line drug treatments comprise gabapentin and lamotrigine [58].

Femiano et al [59-61] evaluated the use of alpha-lipoic acid or thioctic acid at a dose of 600 mg/day, and observed patient improvement after two months. Alpha-lipoic acid is a coenzyme that intervenes in energy production and acts as an antioxidant-neuroprotective agent. Other authors have not obtained good results with this treatment, however [62,63].

Antidepressants such as sertraline, duloxetine, amitriptyline and paroxetine [64,65] have also been used in the management of BMS, with variable results, though in general the effects have been acceptable. Of these substances, paroxetine has produced the best results, though in some cases the side effects have required treatment suspension [66].

There have been reports of improvement with amisulpride at a dose of 50 mg/day. This drug is a

benzamide antipsychotic agent with affinity for the type D_2 and D_3 dopaminergic receptors, though in contrast to other classical neuroleptics it has no affinity for other types of receptors [67].

Conclusions

BMS has been an enigma for many years, since the term has been used in reference to a range of disorders with the clinical characteristics of the patient as their only point in common. This is of great importance when considering an effective treatment strategy.

At present, we speak of primary (or essential) BMS and secondary BMS (associated to a known cause). In the presence of a possible local or systemic cause, management should attempt to resolve the latter. However, in the absence of clinically manifest alterations detected upon inspection of the oral cavity or evidenced by the blood test results, a diagnosis of primary BMS should be considered.

Addressing the psychopathological factors typically found in these patients is more complicated. It has been shown that both primary and secondary BMS can be associated to behavioral disorders, and it is still not clear whether these disorders accompany the background disease process (comorbidities), are a consequence of the process, or both [68].

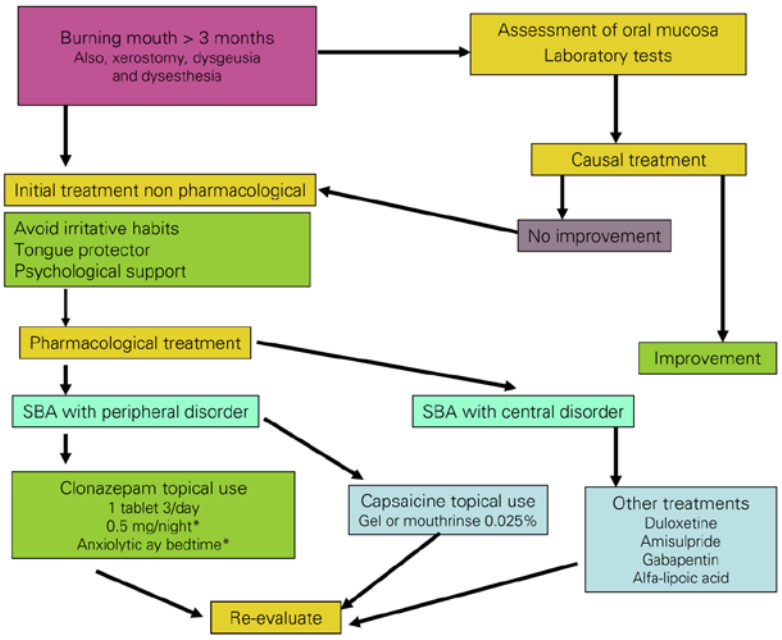
Once the possibility of burning mouth secondary to some identifiable cause has been ruled out, management should aim to avoid possible direct irritative actions upon the oral mucosa. Prominent or cutting tooth edges should be polished, and use can even be made of a protective device such as that described by López-Jornet et al [30]. Likewise, patient anxiety and concern should be reduced by explaining the nature of the symptoms (Figure).

We must attempt to differentiate between possible central or peripheral neuropathy. In this regard, bilateral regional anesthesia of the lingual nerve has been proposed, since persistence of the discomfort following anesthesia may be indicative of a central origin [28].

The treatment of choice in the case of possible primary BMS with peripheral involvement would be topical clonazepam (oral rinses or tablets dissolved in the mouth). Cognitive therapy and the prescription of anxiolytic drugs appear to be effective in patients with important stress and anxiety [69].

In contrast, if a central origin is suspected, or if the aforementioned treatment proves ineffective, we should administer drugs such as amisulpride at low doses (50 mg/day during 24 weeks) or duloxetine (20-40 mg/day during 12 weeks) [70]. Periodic patient re-evaluation is required, with adoption of the same management strategy from the start if no improvement is observed.

Figure. Current criteria treatment algorithm for patients with burning mouth syndrome.



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Síndrome de boca ardiente: revisión y puesta al día

Resumen. El síndrome de boca ardiente (SBA) es un cuadro clínico que padecen mayoritariamente mujeres de edad media o avanzada. Se caracteriza por una sensación muy molesta de ardor o escozor sobre la lengua o en otras zonas de la mucosa bucal. Puede estar acompañado de xerostomía y de disgeusia. Se suele presentar de forma espontánea y tiene un perfil clínico muy característico. Las molestias son continuas, pero aumentan hacia la tarde-noche. Aunque clásicamente se había atribuido a múltiples factores, en los últimos años hay evidencia para relacionarlo con una disfunción neuropática de tipo periférico (fibras C sensitivas o trigeminales) o de tipo central (sistema dopaminérgico nigroestriado). En el diagnóstico hay que descartar lesiones objetivables en la mucosa oral o alteraciones en la analítica sanguínea que puedan ser causa de ardor bucal. El manejo de los pacientes se basa en evitar focos irritativos orales y soporte psicológico. Para el tratamiento farmacológico del ardor en el SBA primario de causa periférica, se puede administrar clonacepam de uso tópico, y pacientes con SBA de tipo central parecen mejorar con el uso de antidepresivos del tipo de la duloxetina, anticonvulsionantes como la gabapentina, o la amisulprida.

Palabras clave. Dolor oral. Estomatodinia. Glosodinia. Manejo clínico. Síndrome de boca ardiente. Tratamiento del dolor.