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The Burning Mouth

JOHN S. MCDONALD, DDS, MS

ABSTRACT Burning in the mouth in and of itself is not all that uncommon. It may result from a variety of local or generalized oral mucosal disorders, or may be secondary to referred phenomena from other locations. Primary burning mouth syndrome, on the other hand, is relatively uncommon. Burning mouth syndrome is an idiopathic pain disorder, which appears to be neuropathic in origin. Thoughts on management of secondary and particularly primary burning mouth syndrome are discussed.

AUTHOR

John S. McDonald, DDS, MS, is an oral and maxillofacial pathologist in private practice in Cincinnati, Ohio, volunteer professor in the Department of Anesthesia for chronic pain management, and a volunteer professor in the Department of Pediatrics, division of pediatric dentistry.

Burning mouth syndrome is one of the more enigmatic oral pain complaints that present in clinical practice. Although not a terribly common clinical complaint in the average private practice, it is in fact frequently encountered in an oral and maxillofacial pathology or oral medicine clinical practice. It is a chronic dysesthetic orofacial pain condition known under a variety of names such as stomatodynia, stomatopyrosis, glossodynia, and glossopyrosis along with a number of other names. It frequently presents as a symptom complex, which may also include xerostomia and/or dysgeusia. It has been grouped along with other idiopathic orofacial pain disorders and other idiopathic pain conditions with a primary complaint of pain that is disproportionate to the evident clinical findings.[1-4]

Problematic is the fact that burning mouth syndrome is characterized by symptoms that can result from a number of local or systemic disorders, some of which can be readily diagnosed while other times no obvious etiology can be found. From a clinicopathologic standpoint two forms of burning mouth syndrome will be discussed: primary burning mouth syndrome, the idiopathic form of the disorder, and secondary burning mouth syndrome, which results from local or systemic disorders that may respond to appropriately directed therapy.[5]

Burning mouth syndrome is usually described as a burning quality, which may vary in severity from aggravating or annoying to agonizing as if the affected area had been scalded or had touched a hot griddle. Tingling and numbness are other features that may be experienced. Affected areas are most commonly said to be the dorsum of the tongue, primarily the anterior tip and lateral borders; the mucosal surfaces of the lips, most often the lower lip; the palate, primarily the anterior

FIGURE 1 Evaluation of the patient with BMS-like symptoms.

hard palate; and gingival tissues. These symptoms, which may occur individually or in combination, are usually bilateral but may be unilateral.[6] Occasionally, the patient will complain the entire mouth burns. The complaint of burning and numbness may be noted concurrently.

The estimated prevalence rate of burning mouth in the general adult population varies widely from 0.7 percent in the U.S. adult civilian population to 15 percent in a Finnish adult population.[7,8] Of note, however, is that on examination of the patients in the Finnish study, half were said to have some clinically observable oral mucosal lesion or candidosis. Burning mouth syndrome affects women much more commonly than men, primarily peri- and postmenopausal females.

Classification

As previously indicated, burning in the mouth can take on two different forms: a primary or idiopathic form of the disease for which there is no evident clinical explanation, and a secondary form derived from the presence of local or systemic factors. In evaluating a patient for burning mouth syndrome, the first and probably most important step is obtaining an accurate clinical history (**FIGURE 1**). Not just that "Doctor, my mouth burns," but the specific areas to which the pain is localized, the pattern of the complaint as to episodic or continuous, time(s) of the day it may be better or worse, if there is such a pattern, sleep pattern (is the sleep disturbed by pain), and the presence or absence of other complaints such as dry mouth or altered taste. A thorough clinical history of the chief complaint then needs to be taken, including a description of the initial presentation, how it has changed over time, a chronological listing of other practitioners who have evaluated the patient for the condition, the various therapies that have been employed, tests that have been done, and the results of any previous tests or therapies. From this the practitioner may then begin to put together in his or her mind a provisional differential diagnosis for the problem. An in-depth health history interview is necessary that includes a history of all medications the patient is taking, previous medications they have taken, the presence of known allergies to any drugs, medications, mouthrinses, dentifrices, chewing gums, cosmetics, etc. A social history is also important and should be geared particularly at present and past psychosocial factors going on in the patient's life. The patient should be questioned as to the use of tobacco, alcohol, caffeine, and the use of any recreational drugs. After all of

TABLE 1

Local factors that may result in burning mouth syndrome-like symptoms

Fissured tongue
Geographic tongue
Hairy tongue
Foliate papillitis
Trauma ■ Physical, i.e., traumatic ulceration, denture irritation, etc. ■ Chemical ■ Thermal, i.e., pizza burn, reverse smoking, etc.
Aphthous stomatitis (RAS) ■ Herpetiform aphthae ■ Aphthae minor ■ Aphthae major (Sutton's disease, PMNR)
Herpes simplex virus infections (HSV) ■ Recurrent labial or intraoral HSV infection ■ Herpes zoster (varicella zoster virus) infection
Oral premalignancy or malignancy

this, a thorough examination of the oral cavity, oropharynx, and adjacent and associated structures should be performed. These are all necessary preliminary steps in determining a diagnosis whether it is suspected the complaint is primary or secondary burning mouth syndrome.

SECONDARY CAUSES OF BURNING MOUTH SYNDROME

Burning in the mouth may arise from a variety of disorders, which may be local or generalized in nature. **TABLE 1** lists many of the local factors that may result in burning mouth syndrome-like symptoms. **TABLE 2** provides an outline of more generalized appearing disorders that may produce burning mouth syndrome-like symptoms. Many of the disorders listed in these two tables may be fairly obvious clinically while others require a differential diagnosis from even a skilled diagnostician and, ultimately, diagnostic testing.

While the list of potential causes for burning in the mouth listed in these tables is long, and even a bit ponderous, a few are much more commonly encountered as secondary causes of burning mouth syndrome than others. For example, fissured and geographic tongues are commonly encountered conditions that may produce a complaint of burning of the tongue. Although far more often asymptomatic, they can produce pain in some individuals, most commonly associated with eating or drinking, particularly spicy foods or liquids. Oral candidiasis is also a frequent cause of burning symptoms in the mouth. Predisposing factors for candidiasis include xerostomia, possibly in combination with gastroesophageal reflux disease, which is either undiagnosed or poorly controlled, and separate or concomitant antibiotic therapy.

If an oral candida infection is suspected and does not respond to initial

TABLE 2

More generalized disorders that may produce burning mouth syndrome-like symptoms

Physical, chemical, drug-induced
- Parafunctional habits
- Contact stomatitis/allergy, i.e., cinnamon allergy, allergy to dentifrices, mouthwashes, cosmetics, denture base allergy, amalgam, gold or other metals
- Fixed drug eruption
- Radiation mucositis and its long-term sequellae
- Chemotherapy

Infection
- Candidiasis, pseudomembranous, acute and chronic erythematous candidiasis including median rhomboid glossitis
- Coliform bacteria, Fusospirochetal infections, Helicobacter pylori
- Gonococcal infection
- HIV infection
- HSV, primary or recurrent (particularly in immunocompromised individuals), VZV infections
- ANUG

Nutritional disorders
- Vitamin B-1, B-2, B-6
- Vitamin B-12
- Iron deficiency
- Folate deficiency

Dermatologic disorders
- Lichen planus, particularly atrophic lichen planus
- Erythema multiforme
- Benign mucous membranes (cicatricial) pemphigoid
- Pemphigus vulgaris
- Lupus erythematosus

Systemic diseases
- Diabetes mellitus
- Uremia
- Crohn's disease
- Blood dyscrasias

Referred pain from other disorders
- Myofascial pain dysfunction, orofacial and paracervical neck musculature, i.e., CN V and cervical nerve distributions
- Gastroesophageal reflux disease
- Trigeminal and glossopharyngeal neuralgia
- Pain referred from tissues in the CN V, CN IX, CN X and cervical nerve distributions

Burning mouth syndrome-like symptoms secondary to disorders of the central nervous system
- Multiple sclerosis
- Parkinson's disease
- Tardive dyskinesia
- Mass lesions involving the brain and CNS

conservative therapies, such as the use of a nystatin rinse or clotrimazole troches, a fungal culture also ordering a mean inhibitory concentration will confirm the diagnosis and provide information on the sensitivity of the fungal organism to other antifungal agents the practitioner may want to utilize. Although xerostomia is often described as part of the symptom complex of idiopathic burning mouth syndrome, its presence alone may produce oral burning. Salivary flow rate can be measured objectively using a modified Schirmer test to confirm the subjective complaint of xerostomia.[9,10] This is important as some patients with a complaint of xerostomia will have objectively measured normal rates of salivary flow.

PRIMARY OR IDIOPATHIC BURNING MOUTH SYNDROME

Although the cause or causes of the primary or idiopathic form of burning mouth syndrome are not truly known, there is an increasing body of evidence pointing to a neuropathic origin. Specific changes in peripheral or central nervous system sensory function and not a psychogenic origin were suggested as early as 1987 by Grushka et al.[11] Ship et al. felt it was likely that burning mouth syndrome reflected a neuropathic condition involving the peripheral and/or central nervous systems.[12] Alterations in sensory function pointing to a possible neuropathic etiology of burning mouth syndrome were also demonstrated by Svensson et al. who reported sensory thresholds as being significantly higher and ratios between pain and sensory thresholds significantly lower on all tested regions.[13] Some objective evidence for a neuropathic etiology for burning mouth syndrome was demonstrated using the eye blink reflex evoked by stimulation of the trigeminal cutaneous nerve branches.[14] Forssell et al. used quantitative sensory testing in addition to the blink reflex to study possible neural mechanisms of burning mouth syndrome pain.[15] They reported abnormal findings in 89 percent of the patients studied by both blink reflex and quantitative sensory testing.

The occurrence of burning mouth syndrome has long been associated with a patient's psychological status. The readers are referred to a paper by Lamb et al. that cited 19 references prior to their 1988 paper addressing the psychological aspects of burning mouth syndrome with the earliest reference dating to 1920.[16] In their critical review of the literature on burning mouth syndrome, Scala et al. pointed out there is little or tenuous evidence to support this view, stating that scientific evidence has generally not supported this belief with the reverse being the case.

THERE IS AN increasing body of evidence pointing to a neuropathic origin.

They interpreted their results as evidence for a generalized, possibly multilevel, abnormality in the processing of somatosensory information in burning mouth syndrome. Of the patients tested with quantitative sensory testing, 76 percent demonstrated abnormal findings in one or more sensory thresholds indicating small fiber dysfunction. More recently, a study was performed comparing superficial biopsies from the lateral aspects of the anterior two-thirds of the tongue in patients with burning mouth syndrome for at least six months with healthy controls.[17] Patients with burning mouth syndrome had a significantly lower density of epithelial fibers than controls with epithelial and subpapillary nerve fibers showing diffuse morphological changes that were thought to reflect axonal degeneration. They concluded that burning mouth syndrome was caused by small-fiber sensory neuropathy. Granot and Nagler hypothesized that the mechanism for development of the idiopathic sensory disturbances of burning mouth syndrome, dysgeusia, and xerostomia is based on a regional neuropathy.[18] They suggested that a regional small fiber neuropathy might affect salivary secretion and oral sensation or alternatively that a primary idiopathic salivary dysfunction might result in sensory neural dysfunction at the receptor level by changing the oral environment.

Burning mouth syndrome, or as also termed in the literature as stomatodynia, has been included in the taxonomy of idiopathic orofacial pain disorders, which includes also atypical odontalgia, atypical facial pain, and facial arthromyalgia.[2-4] It has been proposed these conditions may correspond to a single disease expressed in different tissues characterized by similar or common mechanisms.[2-4] In their recent review of idiopathic pain disorders, Diatchenko et al. suggested that two major contributors to the predilection to develop common idiopathic pain disorders are enhanced pain sensitivity or amplification and psychological distress with genetic variants mediating the activity of physiologic pathways that underlie both of these domains.[1] They believe that as it is highly likely that idiopathic pain disorders share underlying pathophysiological mechanisms and that

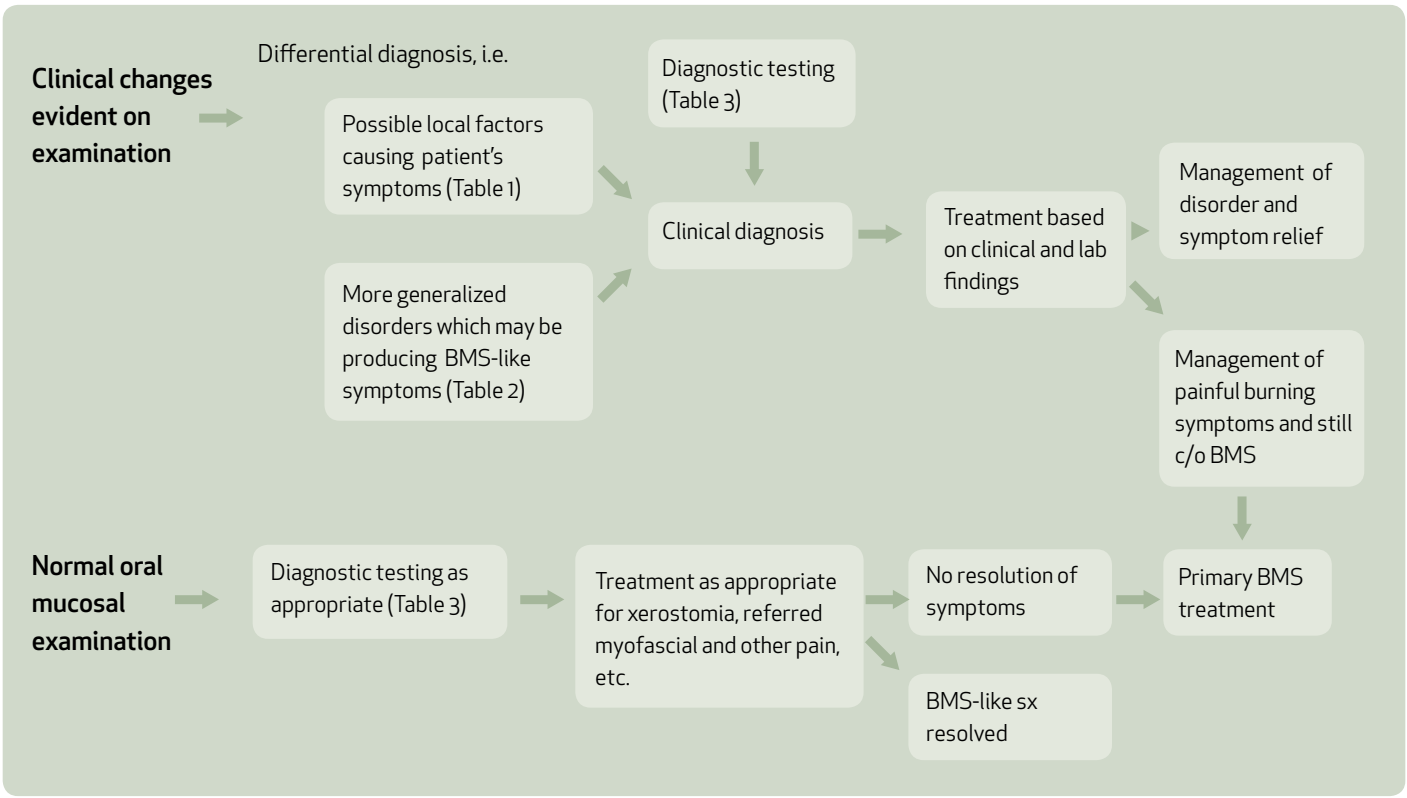

FIGURE 2. Clinical examination.

the same functional genetic variants will often come into play in mediating other types of idiopathic pain disorders.[1,19]

Again, it should be remembered that before entertaining a diagnosis of primary or idiopathic burning mouth syndrome, all secondary factors must be ruled out. Also, it is important to remember there may be apparent secondary factors that overlie true idiopathic burning mouth syndrome that, when resolved, reveal the underlying primary condition.

Treatment

The first step in treating a patient with burning mouth syndrome is an accurate diagnosis. **FIGURE 1** provides an outline for the initial work-up of any chronic pain patient and is a necessary first step in evaluating the patient with a burning mouth. **FIGURE 2** provides an algorithm for the differential diagnosis of the patient's condition. The first step is determining if there are clinical changes evident, either local or generalized, that may produce a burning sensation in the mouth. **TABLE 1** provides a list of local factors that may result in burning mouth syndrome-like symptoms while **TABLE 2** lists more generalized disorders that may also produce these symptoms.

As previously indicated, in some cases where clinical changes are evident, the diagnosis will be fairly obvious. Other times, the cause of the disorder is far less clear-cut, particularly in the case of contact stomatitis or allergy, nutritional and systemic disorders, and when the pain is referred from another site. In many cases, diagnostic testing as outlined in **TABLE 3** is necessary to either confirm the provisional clinical diagnosis or to provide the diagnosis when the underlying disorder is unknown. Without a clear and concise plan to the diagnostic process, the correct diagnosis may not be considered and the clinician is left to take an incorrect approach at managing the disorder. In the case of burning mouth syndrome secondary to other factors and after establishing a clinical diagnosis, the appropriate treatment is pursued, aimed at resolution of the symptoms. In some cases, it will become apparent the evident clinical disorder was overlying and not related to the burning symptoms, and the diagnosis of primary burning mouth syndrome is then made.

When the oral mucosal examination is normal, then diagnostic testing may still be appropriate. This should include, first and foremost, salivary testing, particularly measuring salivary flow rate. A quick, easy, and reliable test in this regard is the modified Schirmer test.[9,10] Salivary pH testing may also be employed. In some patients with a complaint of oral burning and objectively established xerostomia, improving hydration by increasing water intake to normal levels, generally considered to be 64-fluid ounces qd, and decreasing or eliminating caffeine will significantly or com-

TABLE 3

Diagnostic testing for burning mouth syndrome-like symptoms

Salivary testing, including but not limited to ■ Objective testing of salivary flow rate (modified Schirmer test) ■ Salivary pH testing
Fungal and possibly bacterial or viral cultures
Laboratory studies ■ CBCD ■ B-1, B-2, B-6, B-12 levels ■ Blood glucose followed by a fasting glucose tolerance test if high
Oral biopsy with immunofluorescence testing in the case that one or more of the mucocutaneous disorders are suspected
Allergy testing
Clinical assessment ■ Cranial nerve examination ■ Musculoskeletal examination ■ Gastrointestinal consult to R/O GERD if suspected
CT or MRI scans as necessary, depending on the differential diagnosis and clinical findings

pletely mollify the patient's complaint.

In the end, after potential specific etiologies for burning in the mouth with normal clinical evaluations have been ruled out, the clinician is faced with an enigmatic situation. First and foremost, the patient needs reassurance that the tissues are clinically healthy and their condition is not related to any form of cancer or other serious disease. The patient needs reassurance that their condition is not imaginary, i.e., it is not just in their head. The practitioner needs to express cautious optimism and discuss that while there is no one effective treatment regimen, there are a variety of potential treatment protocols that may be employed. The patient needs to understand the chronic nature of their condition with a goal of successful management.

As there is no definitive therapy for primary or idiopathic burning mouth syndrome, the concept of symptomatic therapy should be embraced. Because of the chronic nature of this condition and the fact that recent literature points to the neuropathic nature of this disorder, pharmacologic therapy, either topical or systemic, may naturally be considered. As in assessing other chronic pain complaints, a 10 cm visual analog scale, can be used to assess the severity of the patients pain (0 = no pain; 10 = the worst pain imaginable) and their overall improvement (0 = no improvement; 10 = complete resolution of symptoms). Severity of pain should be assessed at the time of the initial examination, i.e., asking the patient to rate their pain at it worst and at its usual level, and determine if there is a daily pattern to their pain. The patient's pain should also be rated at the start of therapy and assessed at follow-up intervals.

It is also appropriate to employ the concept of escalation of therapy, i.e., topical versus systemic therapy, using the alternative with the fewest side effects first. With a possible neuropathic etiology in mind, the use of topical capsaicin should be considered. It has been shown that topical application of capsaicin can partially or completely mollify the pain in primary burning mouth syndrome.[20]

The effect of capsaicin on the pain in burning mouth syndrome will depend on the underlying pathophysiological mechanism of the process involved in the patient's pain. The effect of the capsaicin is to desensitize the c-fiber nociceptors, thus exciting significant effects on painful disorders arising from these afferents.[21] The authors in this study proposed that capsaicin would be most suitable for treating neuropathic pain symptoms characterized by exaggerated heat pain sensation. Their data also reflected a resistance of A-delta nociceptors to capsaicin. From this it may be inferred that the lack of consistent results from capsaicin therapy points out the heterogeneity of the underlying neuronal mechanisms producing the patient's pain.[22]

For a capsaicin rinse, a Tabasco sauce/water mixture using one part Tabasco sauce (approximately 300 ppm capsaicin and two parts water is rinsed around the mouth for approximately 15 seconds and then expectorated.[23] A 1:3 ratio may be used if the recommended concentration is too objectionable. This procedure is repeated every two to three waking hours for three to four days. If relief is achieved, the interval between rinses may be increased according to the length of pain relief is achieved. A pilot study using systemic capsaicin has also been reported.[24] It was shown to be therapeutically effective for short-term management of burning mouth syndrome, although major gastrointestinal side effects were noted.

Topical or systemic uses of a variety of medications have been considered as treatment for primary burning mouth syndrome. Woda et al. studied the effect of local application of clonazepam for patients with burning mouth syndrome.[25] The subjects were to suck on between one-quarter and one-half of a 0.5 mg

tablet, taking care not to swallow, and expectorating after three minutes. Following their proposed treatment protocol, one-third of patients had experienced total relief of pain, one-third had partial improvement, and one-third had no improvement. The average outcome of all patients' improvement was 52 percent.

Zakrzewska et al. undertook a Cochrane review of interventions for burning mouth syndrome.[26] Nine trials were included in their review. They reported on three interventions as demonstrating a reduction in burning mouth syndrome symptoms: alpha-lipoic acid (thioctic acid), clonazepam, and cognitive behavioral therapy. Two randomly controlled trials were performed comparing alpha-lipoic acid to cellulose starch controls as efficacy against burning mouth syndrome. In the first study, a 20-day trial using 600 mg per day followed by 200 mg per day for 10 days, significant improvement was said to be noted in up to two-thirds of patients receiving alpha-lipoic acid compared to about 15 percent of those using a placebo.[27] In the second study, 200 mg of alpha-lipoic acid was used three times a day for 60 days, again using a cellulose starch pill for control.[28] A statistically significant improvement was noted in 97 percent of patients who used alpha-lipoic acid over two months compared to 40 percent for the placebo group. Follow-up at 12 months showed improvement was maintained completely in 73 percent of patients compared to controls where all patients had noted some deterioration in their improvement. A trial was then undertaken comparing alpha-lipoic acid to bethanacol, lactoperoxidase, or placebo (xylitol in distilled water).[29]

Gremeau-Richard et al. studied the effect of topical clonazepam on burning mouth syndrome.[30] This study demonstrated that sucking a 1 mg tablet of clonazepam three times daily for 14 days resulted in an improvement of pain symptoms in two-thirds of the included patients. They also noted the treatment was not effective in all patients and concluded that like other idiopathic pain, burning mouth syndrome probably results from several mechanisms and topical administration of clonazepam may only be effective when the primary mechanisms are peripheral. Finally, it has been pointed out that in cases of burning mouth syndrome resistant to other therapies, a psychological origin should be considered. Bergdahl et al. in the last study accepted in this review, reported on the effect of cognitive therapy in patients with resistant burning mouth syndrome after odontological and medical treatments were employed.[31] Odontological treatment consisted of diseases diagnosed on estimation of saliva secretion rate and candidal investigation. The control group of patients received attention/placebo therapy. Of the patients receiving cognitive therapy, 27 percent of patients were "cured" during a six-month follow-up period and a reduced intensity of symptoms was noted in almost all of the patients. The attention/placebo group did not show any decrease in intensity of burning mouth syndrome. The authors concluded that if burning mouth syndrome remains after the patient had been appropriately treated from a dental and medical standpoint, their pain was most likely of psychological origin.

IN CASES OF burning mouth syndrome resistant to other therapies, a psychological origin should be considered.

To reiterate, treatment of primary burning mouth syndrome is usually directed at symptomatic relief. As there is evidence it is by and large a neuropathic pain disorder, then, if topical therapies are ineffectual, systemic medications aimed at other neuropathic conditions may be considered. These may include the use of benzodiazepines such as clonazepam as already mentioned, tricyclic antidepressants such as amitriptyline or nortriptyline (side effects of xerostomia may preclude their use), and anticonvulsants such as gabapentin used alone or in combination.[32]

Grushka et al. reported on the use of clonazepam taken orally in escalating doses on burning mouth syndrome.[33] Their dosages ranged from 0.25 mg at sleep to a total dose of 2 mg per day taken in three divided doses. Of the 30 patients in their study, 13 (43 percent) reported at least some improvement and continued to use the medication; eight (27 percent) had noted at least some improvement but had chosen to discontinue its use because of side effects or for other reasons; and nine (30 percent) had reported no benefit from using clonazepam. More recently, Grushka et al. reported a retrospective study using "polypharmacy" consisting of various combinations of low-dose anticonvulsant medications in combination for management of burning mouth syndrome.[34] Medications used included clonazepam, gabapentin, baclofen, and lamotrigine in various combinations. The average maximum pain rating reported was 60.6 prior to treatment, with the average maximum pain rating said to be 32.1 after therapy.

Finally, the rate of spontaneous

remission of burning mouth syndrome has been studied. Grushka et al. reported at least partial remission in nearly 50 percent of patients with burning mouth syndrome with seven years of onset of their symptoms.[34] They also reported a change from constant to cyclic burning during the same time period for some patients still experiencing some pain. More recently, Sardella et al. in a retrospective study looked into the spontaneous remission rate of patients with this disorder.[35] Their data showed complete spontaneous remission in 3 percent of patients within five years after the onset of burning mouth syndrome. They speculated the wide range in remission rate between patients in the previously cited study and their study might be explained through a larger follow-up period in the earlier study.

Conclusion

Burning in the mouth is a most nefarious complaint that may be a challenge to diagnose and, dependent on the ultimate diagnosis, treatment may be even more enigmatic. The first step in management is in arriving at an accurate diagnosis and determining if the burning is secondary to local factors or more generalized disorders as listed in **TABLES 1** and **2**. Initial therapy includes addressing any of these factors that may be present to attempt to mollify the burning. Even in the presence of secondary factors, the primary form may be uncovered.

In its primary form, there are a variety of potential treatment options that may be employed as discussed in this paper. Throughout, the patient must be treated with reassurance and great care using, in the context of its benign but chronic nature, escalation of therapy combined with the principle of doing no harm. Treatment requires almost as much patience on the clinician's part as on the patient's often with less-than-hoped-for results for both parties. ■■■■

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TO REQUEST A PRINTED COPY OF THIS ARTICLE, PLEASE CONTACT John S. McDonald, DDS, MS, University of Oral Pathology Service, University Medical Arts Building, 222 Piedmont Ave., Suite 8400, Cincinnati, Ohio, 45219.