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DIFFERENT ASPECTS OF OROFACIAL PAIN (PART I) GLOSSODYNIA/ STOMATODYNIA

RAZLIČITI ASPEKTI OROFACIJALNOG BOLA (PRVI DEO) GLOSODINIJA/ STOMATODINIJA

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Abstract

Stomatodynia/Glossodynia, also known as burning mouth syndrome is mostly considered to be a psychosomatic disorder characterised by painful sensations within the oral cavity, without detectable abnormalities of the mucous membranes or underlying medical disorder. The burning sensation can affect the tongue, lips, gums, hard pallet, soft pallet, and the internal mucosa of the cheeks and throat. Frequently, patients also complain of xerostomia and dysgeusia. Glossodynia has many different causes, including infection, mechanical irritation, allergic reaction, chemical irritation, underlying disease, and dry mouth. Rarely, glossodynia is related to an inherited genetic defect. Emotional disorders, such as anxiety and depression, as well as an extreme fear of cancer are often associated with burning mouth syndrome. We refer not only to symptoms and possible causes, but also to demographic spread, clinical examination and management.

INTRODUCTION

Glossodynia/Stomatodynia (G/S), also known as burning mouth syndrome (BMS), stomatopyrosis or orodynia is defined by the International Association for the Study of Pain (IASP) in 1983. as a burning pain in the tongue or other oral mucous membranes associated with normal signs and laboratory findings. Still there is no clear understanding of its aethiology or pathogenesis and diagnosis remains basicly clinical [(1)].

Demographics consideration of G/S

Clinical and epidemiologic studies give strong evidence that G/S is mostly prevalent in older women. In most studies, prevalence rates vary from 0.5% to 15%. Although women appear to be preferentially affected in clinical populations, several recent epidemiological studies [(2, 3)] suggest that this difference may be smaller than refered initially. Most studies suggest that prevalence increases with age [(4)]. These differences in prevalence rates and gender among studies may be related to the criteria used to diagnose G/S in both clinical and epidemiological studies.

Most G/S patients report spontaneous onset of pain without known precipitating factors [(5, 6)]. Approximately 17% to 33% of subjects attribute the onset of their oral burning to a previous illness, such as an upper respiratory infection, or a previous dental procedure, which suggests the possibility of neurologic changes preceding the onset of burning in some patients. The distribution of oral sites (most often anterior tongue, anterior hard palate, and the lower lip) does not appear to affect the response to treatment (see later discussion) or the course of the disorder, which can persist many years after onset [(7, 8)].

Pain patterns

Although nighttime pain does not appear to be a problem for most persons with burning mouth syndrome, the pain often gradually increases throughout the day[(9)] and reaches maximum intensity by late overlying. As a result, it is not uncommon for persons with G/S to report difficulty falling asleep at night and interrupted sleep. Reported mood changes such as irritability and decreased desire to socialize have been hypothesized to be related to altered sleep patterns[(9, 10)]. Pain intensity in G/S have been reported to be moderate to intense and may be related to alterations in personality characteristics such as depression and anxiety, which have been documented in other groups of patients with chronic pain [(2)].

Spontaneous remission

At least a partial remission occure within 6 to 7 years after onset in approximately 1/3 to 1/2 of subjects, as reported in the few studies that have examined spontaneous remission in G/S [(3)]. No significant differences in age, sex, duration of disease, or distribution of burning sites have been found among individuals who experienced either partial or complete remission [(10)].

Is G/S really a syndrome?

Most clinical studies suggest that in G/S, oral burning is accompanied by dry mouth, thirst, and dysguesic or altered taste [7, 9, 11]. In an age- and sex-matched control study, Grushka et al.[12] found that approximately 70% of patients with BMS (compared to 11% of controls) reported either change in their ability to taste foods or a persistent dysgeusic taste (especially bitter or metallic). These findings were confirmed later by studies at the Connecticut Chemosensory Research Clinic[13]. In an epidemiologic study Lipton et al. [14] also reported that 40% of patients with burning complaints also experienced an alteration of taste. The dys-geusic taste can be reduced by stimulation with food and rinsing with a local anesthetic rinses in contrast to the oral burning, which decreases with food and increases frequently with topical anesthesia stimulation, respectively. Taste disturbances were also demonstrated objectively in patients with G/S by using electric stimuli and oral tastants (sweet, salt, sour, and bitter) at both threshold and supra-threshold levels.

In contrast to taste changes, most studies have not demonstrated decreased salivary flow in BMS despite subjective complaints of dry mouth and thirst (see later discussion). Many studies however, have demonstrated qualitative changes in saliva despite unaltered flow rates. Although it does appear that disturbances in taste and qualitative changes in saliva are linked directly to oral burning, the cause of this relationship still remains unclear.

CLINICAL EXAMINATION AND FINDINGS

Evaluatin of systemic conditions

Despite a widespread belief that G/S may be the result of certain systemic diseases or nutritional deficiencies, no consistent relationship has been found to support this belief [15, 16, 17] despite the demonstration that more than 58% of persons with G/S show mildly abnormal results for immunologic features such as rheumatoid and antinuclear factor, and 50% have no consistent relationship between G/S and a connective tissue disorder such as Sjogren's syndrome. The relevance of the association between the other pain sensations noted to be more frequent in BMS, including other facial pains, pains in other parts of the body, headache pain and BMS, also remains unclear.

Factors of local influence

In accordance with the International Association for the Study of Pain (IASP) definition of G/S from 1983. oral burning is believed to be unassociated with any local soft- or hard-tissue changes, a theory supported by most studies. Other studies, have reported a higher incidence of soft-tissue lesions such as gingivitis, periodontitis, ulcerative or erosive lesions, and geographic, fissured, scalloped, or erythematous tongue in G/S, which may be one possible source of neuropathic changes underlying G/S in some patients [18].

Changes based on psychophysical status

Burning pain, the main feature of G/S, is also a characteristic feature of some posttraumatic nerve injuries. However, in the later conditions, additional sensory abnormalities that manifest as changes in perception of touch, temperature, two-point discrimination, and pain are often present [19]. Psychophysical testing of many of these modalities in persons with G/S, however, failed to reveal any abnormalities in perception at any of eight intraoral and facial sites tested compared to control subjects, besides the abnormalities of taste (see previous discussion). The only exception in one study was heat pain tolerance, which was found to be reduced significantly at the tongue tip (but not at the lower lip) in patients with G/S. These findings that were extended in further investigation, confirmed the absence of sensory abnormalities in perception of stimulus extent in persons with G/S as compared to controls [20].

On the contrary, qualitative and quantitative differences in some sensory functions of persons with BMS have been determined with argon laser stimulation. A recent report that used quantitative sensory examination, tongue and face telethermography, and selected tongue biopsy demonstrated subclinical polyneuropathy in 50% of patients including loss of function in small diameter nerve fibers [21]. Other recent investigation demonstrated abnormalities in the blink reflex of patients with BMS associated with disease duration, which they believe suggests a possible generalized pathologic changes of the nervous system and supports the concept of modification in peripheral or central nervous system processing in G/S [22].

Additional data are required to support this concept, such as sheding light on the mechanism of nerve injury in G/S and anatomic identification of the types of nerve fibers involved in this type of neuropathy.

Saliva influence

Most salivary flow rate studies in subjects with G/S have not demonstrated a significant decrease in salivary output [4, 7, 10, 14]. In contrast, many studies of stimulated and unstimulated whole saliva and stimulated parotid and stimulated submandibular saliva have demonstrated significant alterations in salivary levels of factors such as proteins [2, 3, 9], mucin, and immunoglobulins in G/S. Additional differences have been noted in salivary pH, buffering capacity, electrical resistance, and conductance in G/S [7, 14, 21]. The qualitative changes in saliva appear to be caused by selective changes that occur in G/S including altered sympathetic tone at the time of menopause, rather than by an overall reduction of flow rate [7].

PROBABLE CAUSES

Psychologic dysfunction

Although current evidence clearly indicates a strong psychologic component within G/S, there is still no evidence of a close causal relationship between psychogenic factors and burning mouth syndrome [4, 7, 8, 21, 22, 23].

Diabetes mellitus

Recent findings of a low prevalence of abnormal glucose tolerance in G/S patients, suggest that: diabetes may not be an important cause of mouth burning [10, 18]. This is in contrast to the known development of painful peripheral neuropathies in some patients with diabetes [10] caused by what is believed to be nerve fiber defects [9].

Denture allergy, mechanical irritation, parafunctional habits, and galvanic currents studies have not supported neither a chemical irritation nor an allergic reaction to dental materials as a significant cause of G/S (there was a little support for galvanic currents as a causative factor) [26]. On the contrary, mechanical irritation [7] may be a more likely candidate for burning effect when associated with dentures because errors in denture design and parafunctional habits (eg. tongue thrusting, grinding, and clenching) [16, 20] have been identified more frequently in persons with G/S.

Menopausal factors

Although most subjects who participate in clinical studies of G/S are postmenopausal women [3, 7, 9], hormone replacement therapy has been ineffective in reducing oral symptoms, in most cases [15]. One reason for this lack of significant effectiveness may be that only women with nuclear estrogen receptors in the oral mucosa may benefit from estrogen replacement. Another reason may be that the changes at the time of menopause are irreversible and not responsive to estrogen replacement. One of these changes at the time of menopause may be the loss of bitter taste sensation (carried by the chorda tympani), with interruption of input leading to loss of inhibition of oral pain and the precipitation of spontaneous pain [10].

Nutritional factors

Nutritional deficiency, especially iron, B12, and folic acid, are linked to the onset of G/S (26). Most recent reports, however, have failed to indicate a higher-than-expected prevalence of nutritional deficiencies in G/S [8, 16, 26]. Despite the suggestion of some investigators, replacement of B1, B2 and B6 for the treatment of G/S did not produce consistent results [16, 18].

Neuropathic changes

It was suggested that oral burning may be the result of a centrally based neuropathic condition that results in the release of peripheral inhibition since it was observed that oral burning in G/S increases after the use of an anesthetic rinse [6]. This is in accordance with taste studies that demonstrated that loss of inhibitory interactions between the central projection areas of the chorda tympani and glossopharyngeal taste nerves after peripheral injury to either nerve can result in the production of phantom taste sensations or dysgeusias. Preliminary spatial taste testing in patients with G/S has provided further support for the possibility that for some persons with BMS, oral burning may result from the loss of inhibition of nociceptive trigeminal fibers secondary to selective injury of either the chorda tympani or glossopharyngeal nerves. Additional support for the possibility of the occurrence of oral burning caused by peripheral nerve injury, including the chorda tympani, is suggested by the findings of other authors [23]. They demonstrated that taste loss, burning pain of the tongue and lips, tingling, and drooling can follow mild nerve injury after local anesthetic block to the inferior alveolar and lingual nerves. The presence of preganglionic para-sympathic fibers to the submandibular and sublingual salivary glands in the chorda tympani, in conjunction with the evidence that injury of the chorda tympani is possible through mild injury (including local anesthetic agents), and the finding that injury of one cranial nerve can result in loss of inhibition of another, suggests that one possible mechanism for G/S may be mild damage of chorda tympani that results in alterations of taste, oral dryness, and pain caused by loss of inhibition of the trigeminal nerve [2].

Other causes

Recently, a number of case reports of oral burning (Lev scalded mouth syndrome) secondary to the use of angiotensin-converting enzyme (ACE) inhibitors such as captopril, enalapril, and lisinopril, which have remitted after discontinuation of the medication [23], also documented the association of G/S with the use of other medications, including antihypertensives. Loss of taste sensation has also been reported to occur with use of acetyl-cholin esterase (ACE) inhibitors, which suggests a link between pain and taste [7].

G/S has also been found to be associated with a sleep disorder, a chronic tongue thrusting habit, parafunctional activity, and myofascial referral sites from nearby trigger points.

MANAGEMENT

Treatment of burning mouth syndrome is highly individualized and depends on your particular signs and symptoms and on the underlying cause or causes, if they can be identified. Most people with burning mouth syndrome can control their symptoms through tailored treatment plans.

In a small number of clinical trials, tricyclic antidepressants (TCAs) have been found to be effective in alleviating idiopathic G/S in some patients. The beneficial effects of the TCAs (ie. amitriptyline, desipramine, nortriptyline, imipramine, and clomipramine) for chronic pain relief have been corroborated in other studies that indicate that low doses of TCAs may act as analgesics.

Interest in the benzodiazepines, especially chlordiazepoxide and clonazepam, for the treatment of orofacial pain, including G/S, has been renewed recently [4, 19]. Some studies have reported [22-26] reduction of pain in approximately 67% of subjects with G/S at doses 15 mg to 30 mg daily of chlordiazepoxide. Studies using clonazepam, which is also used as an anticonvulsant have shown benefit in temporomandibular disorders at low doses (0.375 mg daily) [21] and in G/S at higher doses (0.5 mg to 6 mg daily), with an average reported daily dose of 2 mg. The use of clonazepam is in accordance with recent literature, which has demonstrated the effectiveness of various anticonvulsants, including clonazepam, for the management of other pain states, including trigeminal neuralgia, diabetic neuropathy, and migraine prophylaxis [23].

Benzodiazepines are gamma-amminobutyric acid (GABA) receptor agonists that bind to both peripheral and central receptor site. Benzodiazepines interact with specific receptors on the GABA receptor – chloride channel macrornolecular complex. Binding of these drugs facilitates the inhibitory actions of GABA. In the presence of benzodiazepines, chloride ion conductance is increased because of increased frequency of GABA-mediated chloride ion channel openings. Some of the benzodiazepines, including diazepam and clonazepam, possess selective anticonvulsant

action with suppression of convulsions at doses that do not cause sedation. Clonazepam appears to differ from other benzodiazepines as it seems to bind more to central than peripheral benzodiazepine receptors and have a greater effect on the brain serotoninergic system.

Several recent studies have also demonstrated the effect of clonazepam in G/S [11, 21]. A meta study [26] conducted on 30 persons with G/S reported preliminary evidence of the effectiveness of low-dose clonazepam (average 0.87 mg to 1,36 mg) in 70% of subjects in reducing oral burning (and in some cases in reducing taste dysgeusias as well). In the second study, topical application was also found to be effective in reducing oral burning [11]. This degree of responsiveness is higher than the spontaneous remission rate of G/S (see previous discussion). Although it is possible that this class of drugs may exert its effect on oral burning by acting as a sedative/hypnotic, this possibility appears unwarranted because it is effective topically and because the maximal effect of clonazepam was observed at the lower dose range, with no increase in symptom relief at increasing doses, despite the linear corrrelation between dose and. serum concentration [19].

CONCLUSION

Considering a certain number of possible causes of G/S, we strongly recommend you to see a doctor as soon as possible if symptoms don't disappear in a day or two. If you have diabetes, athyroid disorder, or some other underlying illness, you should report the symptoms to your physician as soon as possible, because your symptoms may be related to theunderlying disease or condition.

Apstrakt

Stomatodinija/glosodinija, poznata i kao sindrom usta koja gore, se se najčešće se smatra psihosomatskim poremećajem koji se karakteriše gorućim bolom u ustima bez vidljivih abnormalnosti sluzokoža ili drugih poremećaja. Ovaj bol može da zahvati jezik, usne, desni, tvrdo i meko nepce, kao i sluzokožu unutrašnje strane obraza i grla. Pacijenti se često žale i na kserostomiju i disgeziju. Uzročnici glosodinije su različiti, između ostalog na infekcije, mehaničke i hemijske iritacije, alergijske reakcije, osnovna bolesti i suva usta. U retkim slučajevima, uzrok glosodinije je nasledni genetski defekt. Emocionalni poremećaji, kao što su anksioznost, depresija i izraziti strah od malignih bolesti, često su povezani sa ovim sindromom usta koja gore. U radu su opisani ne samo simptomi i mogući uzroci, već i demografski faktori, kliničko dijagnostikovanje i lečenje.

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