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Management of burning mouth syndrome

Leczenie zespołu pieczenia jamy ustnej

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Abstract

In this review, we discuss some practical strategies that can be useful for clinicians in the interdisciplinary and individualised management of patients with burning mouth syndrome. Burning mouth syndrome (stomatodynia, glossodynia) is a chronic, idiopathic pain syndrome accompanied by a sensation of pain or burning, scalding, pinching, numbness or stinging without clinical changes in oral cavity mucosa in the absence of abnormalities in additional tests. In addition to the above symptoms, burning mouth syndrome is characterised by a sensation of oral dryness (xerostomia), distortion of the sense of taste (dysgeusia), and food hypersensitivity. Patients may further report mood swings, anxiety, insomnia, personality disorder, chronic fatigue, headache or carcinophobia. Burning mouth syndrome has a clear predisposition to perimenopausal females, and significantly affects not only the quality of life of patients but also that of their families. The aetiopathogenesis of the disease is not fully understood, and treatment predominantly focuses on symptomatic relief. Topical therapies involve benzydamine, clonazepam, capsaicin, lidocaine, lactoperoxidase, and sucralfate. Antidepressants, antiepileptics, atypical neuroleptics, and benzodiazepines have been used in systemic pharmacological management of burning mouth syndrome. Burning mouth syndrome therapy should be a combination of pharmacological and neuromodulating effects with psychological support for both patients and their families. The therapeutic strategy should be highly personalised, interdisciplinary, and holistic.

Keywords: burning mouth syndrome, treatment, oral burning, stomatodynia

Streszczenie

W przeglądzie omawiamy kilka praktycznych strategii, które mogą być przydatne dla klinicystów w interdyscyplinarnym i zindywidualizowanym leczeniu pacjentów z zespołem pieczenia jamy ustnej. Zespół pieczenia jamy ustnej (stomatodynia, glosodynia) to przewlekły, idiopatyczny zespół bólowy, któremu towarzyszy uczucie bólu lub pieczenia, parzenia, szczypania, drętwienia lub kłucia bez zmian klinicznych błony śluzowej jamy ustnej i odchyleń w badaniach dodatkowych. Poza powyższymi objawami objawia się uczuciem suchości w jamie ustnej (kserostomia), zaburzeniami odczuwania smaku (dyzgeuzja) oraz nadwrażliwością pokarmową. Pacjenci mogą ponadto zgłaszać wahania nastroju, niepokój, bezsenność, zaburzenia osobowości, chroniczne zmęczenie, ból głowy lub kancerofobię. Zespół pieczenia jamy ustnej szczególnie często występuje u kobiet w okresie okołomenopauzalnym i wywiera znaczący wpływ na jakość życia zarówno chorych, jak i ich rodzin. Etiopatogeneza choroby nie jest w pełni wyjaśniona, a leczenie koncentruje się głównie na łagodzeniu objawów. Strategia terapeutyczna powinna być wysoce spersonalizowana, interdyscyplinarna i holistyczna. Terapie miejscowe obejmują benzydaminę, klonazepam, kapsaicynę, lidokainę, laktoperoksydazę i sukralfat. W ogólnoustrojowym leczeniu farmakologicznym stosowano leki przeciwdepresyjne, przeciwpadaczkowe, atypowe neuroleptyki i benzodiazepiny. Prowadzenie pacjenta obciążonego zespołem pieczenia jamy ustnej powinno być połączeniem działania farmakologicznego i neuromodulacyjnego ze wsparciem psychologicznym zarówno pacjentów, jak i ich rodzin. Strategia terapeutyczna powinna być wysoce spersonalizowana, interdyscyplinarna i holistyczna.

Słowa kluczowe: zespół pieczenia jamy ustnej, leczenie, pieczenie jamy ustnej, stomatodynia

INTRODUCTION

In this review, we discuss some practical strategies that can be useful for clinicians in the interdisciplinary and individualised management of patients with burning mouth syndrome (BMS). BMS is a chronic, idiopathic pain syndrome accompanied by a sensation of pain or burning, scalding, pinching, numbness or stinging (stomatodynia, glossodynia) with neither clinical changes in oral cavity mucosa nor abnormalities in any additional diagnostic tests. In addition, BMS features may include a sensation of oral dryness (xerostomia), distortion of the sense of taste (dysgeusia), and food hypersensitivity. Apex or anterior two-thirds of the tongue, hard palate, lower lip, and rarely the floor of the mouth, cheek mucosa or gums are the most common pain locations in the mouth. These symptoms of moderate to severe pain intensity are usually symmetric. Stress, fatigue and speaking increase the severity of pain, whereas eating and chewing gum relieve this sensation. Pain lasts during the day, increasing in the evening and subsiding at night (Bergdahl and Bergdahl, 1999). The classic clinical manifestation of BMS is a triad of symptoms (glossodynia, xerostomia, and dysgeusia) persisting for at least three months (Grushka et al., 1987). The International Headache Society describes BMS as intra-oral burning or dysaesthetic sensation recurring daily for more than two hours a day for more than three months without clinically evident causative lesions [Headache Classification Committee of the International Headache Society (IHS), 2013]. Moreover, patients may report mood swings, anxiety, insomnia, personality disorder, chronic fatigue, headache or carcinophobia (Formaker and Frank, 2000; Scala et al., 2003). The hypothesis of the psychogenic origin of pain in BMS has been proposed for many years (Browning et al., 1987). The latest published results of histological, neurophysiological, brain imaging and quantitative sensory testing studies classified BMS as "painful cranial neuropathies" (Lauria et al., 2005; Puhakka et al., 2016; Reyes-Sevilla, 2020).

EPIDEMIOLOGY AND AETIOLOGY OF BMS

The prevalence of primary BMS in middle-aged adults and elderly population is estimated to be 0.7% to 3.7%. The peak incidence occurs in the seventh decade of life. BMS is more frequently seen in women, with an average female/male ratio of 7:1 (Bergdahl and Bergdahl, 1999; Kohorst et al., 2015; Tu et al., 2019).

It is worth mentioning that clinical symptoms of BMS may be secondary to systemic conditions (anaemia, diabetes, thyroid diseases, perimenopausal hormonal changes, gastroesophageal reflux disease), deficiencies (B vitamins, folic acid, iron), food allergies, depression, neurosis, bruxism, medications (such as angiotensin-converting enzyme inhibitors), dental diseases, poorly fitting dentures or improper oral hygiene. These symptoms are defined as secondary BMS (Jääskeläinen, 2012; Scala et al., 2003).

The neuroscience pathogenesis of BMS is concerned with the distraction of nociceptive perception at various levels of peripheral and central nervous sensory transmission (Jääskeläinen, 2012). Jääskeläinen (2012) distinguished three neurological subtypes of BMS: small fibre peripheral neuropathy of the oral mucosa (50–65%), trigeminal neuralgia (20–25%), and dopaminergic system dysfunction (20–40%). Patients with BMS are affected by disturbed thermal perception (hypoesthesia, hypoalgesia, thermal allodynia), along with morphological changes of small fibres of the peripheral nervous system characterised by axonal degeneration (Forssell et al., 2002). Studies on trigeminal nerve function (blink reflex, evoked potentials) have revealed abnormalities in many patients (Jääskeläinen et al., 1997). Impairment of dopaminergic transmission in the nigrostriatal dopamine system has also been noted (Hagelberg et al., 2003). Furthermore, steroid dysregulation and autonomic nervous system dysfunction may play a role in the pathogenesis of BMS (Jääskeläinen, 2018; Jääskeläinen and Woda, 2017). Some other observations relate to a decrease in the concentration of neurokinin A and substance P, as well as an increase in salivary cortisol and nerve growth factor levels (Nosratzehi et al., 2017).

CLINICAL CLASSIFICATION OF BMS

Clinically, BMS can be divided into two types, central and peripheral (Jääskeläinen and Woda, 2017). The central type does not respond to topical treatments and is often associated with psychiatric comorbidities. Anxiety, depression, and schizotypal and obsessive-compulsive personality disorders are reported by more than 50% of patients with BMS (Galli et al., 2017). Patients with the peripheral type of BMS respond well to topical therapies. Hence, the first-line management for patients reporting a burning sensation in the oral cavity includes the identification and elimination of both local and systemic factors leading to the development of secondary BMS (Jääskeläinen, 2018). If the elimination of potential aetiopathological factors has not resulted in a significant relief of the patient's clinical symptoms, primary BMS may be suspected (Scala et al., 2003). It should be determined whether the origin of oral mucosal pain is the central or peripheral nervous system. There is no method for effectively distinguishing peripheral and central nervous system disorders, and the diagnosis of primary BMS can be considered one of exclusion in the present state of knowledge (Jääskeläinen and Woda, 2017). Furthermore, according to Jääskeläinen's division of neurological disorders and the coexistence of several types, it has been suggested that the diagnosis of BMS can be a challenge (Jääskeläinen and Woda, 2017).

TREATMENT OF BMS – PHARMACOLOGIC STRATEGIES

BMS treatment is demanding for the physician and should include symptomatic treatment and therapy targeted at the mechanisms of neuropathic pain (Jääskeläinen, 2012).

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The initial step of treatment usually involves topical anti-inflammatory, analgesic, local anaesthetic, antibacterial, and protective medications. These include benzydamine, clonazepam, capsaicin, lidocaine, lactoperoxidase and sucralfate. Topical medications are effective for the treatment of peripheral BMS.

Benzydamine is an analogue of indazole, and belongs to the group of non-steroidal anti-inflammatory drugs. Benzydamine is used in the form of lozenges or irrigation solutions (0.15% three times a day). The drug has anti-inflammatory, anti-swelling, local anaesthetic, disinfecting, and antiaggregant properties, and reduces the tension of smooth and striated muscles. It should be emphasised that benzydamine is used as the first-line medication in the pharmacological management of BMS for new and undiagnosed patients (Klasser et al., 2012; Ślebioda and Szponar, 2014). Despite the satisfactory results obtained in the treatment of mucosa inflammation of various aetiology, benzydamine is not effective in all patients with BMS. Research found that the clinical application of benzydamine hydrochloride oral rinses in the treatment of patients with BMS did not demonstrate significant efficacy in comparison with the use of a placebo solution (Sardella et al., 1999).

Clonazepam belongs to benzodiazepine analogues and has proven to be beneficial in the treatment of peripheral BMS. Topical treatment (three-minute sucking of a 1 mg clonazepam tablet with expectoration, three times a day) ensures a high concentration of the drug locally, in the oral cavity (McMillan et al., 2016). Clonazepam is a ligand for the GABA A-benzodiazepine receptor complex widely distributed in the central and peripheral nervous system, as well as nerve fibres of the oral mucosa (Basit and Kahwaji, 2023). Stimulation of the central GABA A-benzodiazepine receptor increases the GABA inhibitory effect, leading to anticonvulsant, sedative, hypnotic, anxiolytic and myorelaxant effects (Basit and Kahwaji, 2023). Peripheral GABA A-benzodiazepine receptors are not fully understood. It may be assumed that changes in peripheral GABA A-benzodiazepine receptor density are related to the development of BMS (McMillan et al., 2016). In addition, buccal hyperalgesia is associated with receptor density changes related to age and deficiency of steroid hormones (McMillan et al., 2016). Topical effectiveness of clonazepam in peripheral BMS seems to confirm this aetiopathogenesis (Shin et al., 2023). However, systemic clonazepam (0.25–0.75 mg of clonazepam a day) or combination therapy may be required in some cases (McMillan et al., 2016; Shin et al., 2023).

Capsaicin is an alkaloid found primarily in the *Capsicum* genus and is responsible for the spicy and searing taste of chili peppers (Hayman and Kam, 2008). The alkaloid is reported to have a benefit in relieving topical pain due to its high affinity to the transient receptor potential vanilloid subtype 1 (TRPV1), and subsequent nociceptive neurons desensitisation (Jørgensen and Pedersen, 2017). Capsaicin is an interesting alternative method of BMS treatment. It is used as 0.001% and 0.025% gel, 0.02% rinse or even a solution of 5–6 drops of Tabasco sauce in a tablespoon of water (Jørgensen and Pedersen, 2017; Peikert et al., 1991). However, long-term therapy is not possible due to gastric symptoms (Petruzzi et al., 2004).

Lidocaine is a local anaesthetic used in the treatment of BMS. Patients may take advantage of lidocaine rinses or a lingual nerve block. Trigeminal nerve block described in the literature effectively reduces pain in 50% of patients with BMS (Grémeau-Richard et al., 2010).

Lactoperoxidase secreted from mammary, salivary, and other mucosal glands is part of the innate immune response with bactericidal and antiviral activity. Lactoperoxidase is applied topically in the form of mouthwashes (McMillan et al., 2016).

Sucralfate is a complex of aluminium hydroxide and sulphated sucrose, successfully used in the treatment of BMS (1 g suspension of sucralfate four times a day or chewable tablets containing 1 g of sucralfate four times a day). Applied topically, the drug forms a cytoprotective shield on the oral mucosa (Campisi et al., 1997; McMillan et al., 2016).

Central BMS symptoms can also be reduced by systemic medications. These include antidepressants, atypical neuroleptics, anticonvulsants, and benzodiazepine analogues.

Antidepressants are effectively used in the treatment of BMS coexisting with affective and anxiety disorders. Tricyclic antidepressants (clomipramine 25–75 mg/day, amitriptyline 75–100 mg/day, doxepin 75–100 mg/day), serotonin reuptake inhibitors (sertraline 50 mg/day, paroxetine 20 mg/day), serotonin and noradrenaline reuptake inhibitors (duloxetine 60 mg/day), and serotonin reuptake inhibitors with 5-HT_2 serotonin receptor antagonism (trazodone 200 mg/day) are usually used (Solberg et al., 2014; Tu et al., 2019). A two-phase effect of antidepressants has been reported. During the first five to seven days, analgesia is visible, then gradually for one to two months there is a reduction of BMS symptoms such as taste disturbances and dry mouth sensation. Stabilisation and complete therapeutic response occur within three to six months of treatment implementation (Liu et al., 2018; Solberg et al., 2014; Tu et al., 2019). However, complete remission may not be achieved in all patients with BMS (Solberg et al., 2014; Tu et al., 2019). Maina et al. (2002) have observed a reduction in BMS symptoms in 70% patients after eight weeks of paroxetine and sertraline administration that were equally effective. Beneficial effects of duloxetine, amitriptyline, as well as clomipramine for rapid disease remission, have also been confirmed (Adamo et al., 2021; Kim et al., 2014; Maina et al., 2002).

The atypical neuroleptic olanzapine (2.5–5 mg/day), is a second-generation thienobenzodiazepine derivative with potent antagonist activity of central serotonin and dopamine receptors, particularly 5-HT_{2A} and D2, and also muscarinic, α1-adrenergic and/or H1 histamine receptors (Ueda et al., 2008). Olanzapine as an inhibitor of the dopaminergic transmission of the mesolimbic and nigrostriatal

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pathways can be an effective drug in the treatment of BMS. Clinical observations indicate that the drug is well tolerated and brings rapid and long-lasting pain relief in patients with BMS (Ueda et al., 2008). Amisulpride (50 mg/day) also brings long-term improvement of BMS (Rodriguez-Cerdeira and Sanchez-Blanco, 2012). It is worth mentioning that quetiapine is ineffective in the treatment of BMS (Maina et al., 2002; Rodriguez-Cerdeira and Sanchez-Blanco, 2012).

Gabapentin is an anticonvulsant and analgesic analogue of gamma-aminobutyric acid. Gabapentin (300 mg/day) with alpha-lipoic acid (600 mg/day) is more efficacious in BMS treatment than gabapentin alone (López-D'alessandro and Escovich, 2011).

Organic alpha-lipoic acid as an antioxidant and anti-inflammatory compound may improve some neurophysiological biomarkers and demonstrate neuroprotective effects (Femiano, 2002; Palacios-Sánchez et al., 2015).

NON-PHARMACOLOGIC THERAPIES FOR BMS

Psychotherapy, cognitive-behavioural therapy or psychological support administered for several weeks lead to effective pain relief that lasts up to six months. Based on studies, psychotherapy helps achieve symptom improvement in 70% of patients with BMS (Bergdahl et al., 1995; Miziara et al., 2009).

Tongue protectors were found to be effective especially in the case of tongue traumatisation and in patients with secondary BMS. The protectors consisted of a transparent, low-density polyethylene sheath covering the tongue from the tip to the posterior third. Patients used the protectors for 15 minutes three times a day for two months with the therapeutic aim of avoiding continuous rubbing against the teeth or dentures (López-Jornet et al., 2011).

Photobiostimulation (low-level laser therapy procedure, LLLT) initiates biochemical reactions leading to the normalisation of cell membrane potential and stimulating cell growth and nutrition. The clinical effects may include analgesia, and anti-inflammatory and biostimulatory results (Barbosa et al., 2018; Bardellini et al., 2019).

Acupuncture has not failed to demonstrate clinical efficacy. However, small groups of patients suffering from BMS may benefit from this method (Susano et al., 2017; Yan et al., 2012).

Electroshock therapy, repetitive transcranial magnetic stimulation (rTMS) or electroconvulsive therapy (ECT) may be effective in patients with severe and treatment-resistant BMS (Suda et al., 2008; Umezaki et al., 2016).

CONCLUSION

BMS is a chronic pain syndrome that affects the quality of life of both patients and their families. The aetiology and pathogenesis of the disease are not fully understood, and treatment predominantly focuses on symptomatic relief. The management of BMS and its effective treatment remains a significant challenge for clinicians (Alsabbagh and Ouanounou, 2022; Reyad et al., 2020). Current therapies lead only to some degree of symptomatic control, but not to a desirable cure of the disease. Based on the complexity and clinical observations of BMS, it has been assumed that the most promising therapies should be highly personalised, interdisciplinary, and holistic, including a combination of pharmacological and neuromodulation methods, along with psychological support and practical education for both patients and their families.

Conflict of interest

The authors report no financial or personal relationships with other individuals or organisations that could adversely affect the content of the publication and claim ownership of this publication.

Author contributions

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