Ref: Ro J Med Pract. 2023;18(3)
DOI: 10.37897/RJMP.2023.3.2

Burning mouth syndrome (BMS) - possible pathogenesis related to pain pathways

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Abstract

Burning mouth syndrome (BMS) has been admitted relatively recently as a condition/syndrome within oral pathology, bordering various other medical specialities, which may collaborate to determine the diagnosis and especially to achieve therapeutic success. From a clinical point of view, BMS can present itself in 2 forms: the primary/essential form, whose etiopathogenesis is unexplained, justifying the permanent concern as a topic of research at the international level, and the secondary form, in which the causes of the syndrome can be identified and treated/ removed, allowing healing.

Particular to the primary form of BMS is the discrepancy between the extent of subjective pain felt by the patient as "burning" and the lack of any objective (clinical) signs in the oral mucosa. In this form of BMS, the pathogenesis of pain can be explained by invoking the role and importance of the field and some favourable factors. In recent years it has been noticed that changes in taste perception and pain tolerance could be possible causes of the "burning" sensation. Thus, the involvement of the field allowed the following hypothesis to be issued, namely that taste is generated mainly at the level of the fungiform lingual papillae.

Keywords: Burning mouth syndrome (BMS), cranial nerve (CN), orofacial pain, chronic pain

INTRODUCTION

The common pathway for the cortical transmission of orally generated taste and pain

The taste receptor. Taste buds and their function

Just like any other analyzer, the peripheral segment of the reflex arc that contributes to the perception of taste is the taste bud. Structurally, it contains the taste receptors, represented by specific cells, with a renewal capacity every 10-12 days (physiological apoptosis). Taste buds are distributed throughout the mucosa of the oral cavity and oropharynx, being spread over the soft palate, tonsillar pillars, nasopharyngeal area, epiglottis, pharynx, and upper larynx [1,2].

On the surface of the tongue, the taste buds are spread unevenly in the structure of the lingual papillae. These are defined as anatomical structures in the form of ridges or elevations of the lingual mucosa and which, according to appearance, are divided into 4 categories:

- circumvallate, in number from 8 to 10, present on the dorsal side of the tongue, arranged in two lines that form the lingual V-shaped line;
- foliated, small vertical folds parallel to each other, located on the edges of the tongue in the middle portion;
- filiform, thin and elongated, located diffusely on the dorsal surface of the tongue.

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Article History:
Received: 30 August 2023
Accepted: 7 September 2023

- fungiform, rounded, 1-2 mm in diameter, darker red color, disseminated among the filiform papillae [2,3].

Adults have approximately 10.000 taste buds, and children have a little more. After the age of 45, the taste buds degenerate, which explains the reduction of the taste sensation with age [2].

Primary taste sensations

The fundamental tastes are perceived differently by the taste buds, as follows:

- The sweet taste is perceived especially in the front area of the tongue;
- Sour and salty taste in the lateral lingual areas;
- Bitter taste in the circumvallate papillae on the back of the tongue [2].

Guyton and Hall (2006) also distinguish a 5th primary taste, namely umami (a Japanese word which in translation means "savouriness" and which designates a sensation of pleasant taste, qualitatively different from the other four fundamental tastes) [4]. Umami is the dominant taste in foods containing L-glutamate, such as Parmesan cheese, fermented cheese, seaweed soup called "dashi" etc.

Innervation of the tongue

The collection of information on language sensitivity is performed by:

- the trigeminal nerve (cranial nerve V), which through its sensitive branches provides general sensitivity (tactile, pain, thermal) in the anterior 2/3 of the tongue;
- the facial nerve (CN VII), which through the chorda tympani nerve provides sensory innervation in the anterior 2/3 of the tongue;
- the glossopharyngeal (CN IX) and vagus (CN X) nerves, which are responsible for the gustatory sensation of the root of the tongue and the mucous membrane of the valleculae (CN X).

Damage to the fibers that conduct the sensitive-sensory excitations of the pair of V and/or VII cranial nerves can affect the sensitivity of the anterior 2/3 of the tongue. As an argument in support of the previous statement, there is the possibility of the occurrence of sensitivity disorders after trauma through local anesthesia and the extraction of the wisdom tooth [5].

Damage to the fibers of the pair of cranial nerves IX changes the sensory sensitivity at the base of the tongue. In medical practice, this can occur secondary to tonsillectomy.

RESULTS

The afferent pathway of the taste reflex arc. Regulation of salivary secretion on the descending (effectory) pathway.

In recent years, it has been observed that changes in the perception of taste and pain tolerance could be possible causes of the burning sensation. Taste is fundamentally located at the level of fungiform lingual papillae, and in certain patients with non-specific algic syndrome, especially women, there is a high number of such papillae present on the anterior part of the tongue, these patients being called "supertasters" [6].

Etiologically, any factor that affects the papillae can lead to changes in taste (for example, infections, trauma, injuries, contact sensitivity, nutritional deficiency, etc.) Estrogen deficiency at menopause accentuates sensory changes [5].

The afferent pathway of the reflex arc is identically structured with other similar but functionally different ones containing three neurons: the protoneuron, the deutoneuron, and the third thalamic neuron.

The protoneuron, depending on which nerve the sensory fibers belong to, is located differently. So:

- for the nerve impulses that are conducted to the cerebral cortex via the VII nerve (the chorda tympani nerve - a branch of the facial nerve), the first neuron of the gustatory pathway is represented by the geniculate ganglion;
- for those belonging to the glossopharyngeal nerve (CN IX), by the Andersch and Ehrenritter ganglia (as roots of the protoneuron), which collect the gustatory nerve excitations from the posterior ⅓ of the tongue;
- and for the gustatory impulses from the base of the tongue, pharynx, soft palate, epiglottis, and larynx that are transmitted via the vagus nerve (CN X), the protoneuron is represented by the nodose ganglion [2,4].

DISCUSSIONS

According to Grushka (2014), peripheral lesions of the trigeminal (V), facial (VII), glossopharyngeal (IX), and vagus (X) cranial nerves induce changes in taste, variously localized pain, including dental, sensation of "dry mouth" or jaw constriction in patients. Changes in occlusion and altered tactile sensitivity may also occur (foreign body sensation, loaded tongue, etc.) [5].

From these ganglia, the gustatory impulses are led to the deutoneuron, located in the nucleus of the solitary tract in the medulla [2].

It must be said that from the deutoneuron level, the nerve impulse carrying gustatory information travels:

a) ascending, towards the third thalamic nucleus.

The axons of the deutoneurons, ascending, reach the level of the ventral posteromedial nucleus of the thalamus. It represents the root of neuron III of the sensory reflex arc;

b) descending, making connections that become effectory pathways.

From this level (the solitary tract in the medulla), the gustatory excitation present as a gustatory nerve influx is tridirectional. A large number of impulses reach the superior and inferior salivary nuclei directly, from where commands are sent to the secretory effectors, differentiated, with a triple destination:

1. from the inferior salivary nucleus in the medulla, through the glossopharyngeal nerve, for the parotid glands;
2. from the superior salivary nucleus in the bridge, through the facial nerve, for the submandibular and sublingual glands. The pairs of cranial nerves VII and IX parasympathetically innervate the main salivary glands. Parasympathetic stimulation stimulates saliva secretion, while sympathetic stimulation decreases secretion and makes it more viscous [5].
3. also, from the level of the solitary tract, collateral branches depart towards the reticulated nucleus of the reticulated formation, from where, on the effector pathway of the reticulospinal tract, fibers reach both the salivary glands and the facial muscles, the masticatory muscles, the muscles involved in swallowing and the motor muscles of the tongue [1,2].

Damage to the nerve fibers of the pairs of cranial nerves VII and IX can reduce salivary flows. In conclusion, damage to pairs of cranial nerves V, VII, IX, or X can lead to altered taste sensation and changes in salivary flow. In addition, damage to one nerve allows other nerves to become hyperactive, leading to dysaesthesia [5].

In conclusion, the ascending pathway of the gustatory nerve impulses to the cortex is continued by the fibers that leave the nucleus of the solitary tract, pass the opposite side, and go up to the third neuron of the pathway, represented by the posterior-median ventral nucleus of the thalamus [1,2]. Also from the level of the solitary tract, numerous gustatory impulses are transmitted through the brain stem directly to the superior and inferior salivary nuclei, and these centers transmit impulses to the submandibular, sublingual and parotid salivary glands, to regulate the secretion of saliva during food ingestion and digestion [4].

The cortical projection of the thalamic fibers occurs in the lower area of the ascending parietal gyrus, near the motor centers of the tongue, masticatory, and swallowing muscles. The projection ends in cortical area 43 of the parietal lobe (according to Brodmann's numbering, 1909), in the operculum region of the insular lobe [2].

The areas are located in the lower part of the postcentral gyrus of the parietal cortex (the somatosensory area I), where they penetrate deeply into the Sylvian fissure and the adjacent operculum insular area [4].

At the level of the postcentral gyrus, the gustatory cortical areas are located lateroventrally and rostral to the projection area of tactile impulses from the tongue, located in the somatosensory area I. At the level of the cerebral cortex, in the projection areas, the generation of functional disinhibition that induces activation or even hyperactivation is observed [4].

The hyperactivity of the trigeminal nociceptive pathways, in turn, produces a much more intense response under the action of oral irritants and leads to the appearance of the sensation of oral pain [7].

Dysregulation of taste sensation and pain sensitivity, respectively

Xerostomia is a frequently encountered symptom in dental medicine, with complex multifactorial etiopathogenesis. The symptom is due to a reduction in the amount of saliva or qualitative changes in the saliva, but it can also occur in people with a normal amount of saliva [8].

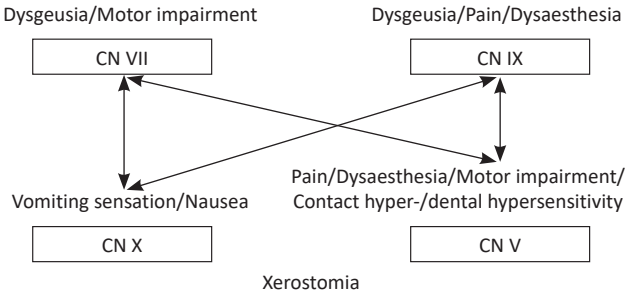

FIGURE 1. *A common pathway for the cortical transmission of taste and orally-generated pain. The pairs of cranial nerves V, VII, IX, and X are interconnected. Damage to the gustatory fibers of the VII or IX nerves results in loss of inhibition and increased sensitivity-sensory hyperexcitability of other cranial nerves [5]*

Qualitative taste disturbance can be classified based on:

a) clinical criteria, using the symptom described by the patient (subjective element);
b) objective sensory determinations,

in: total/partial ageusia/specific ageusia, total/partial hypogeusia and dysgeusia.

Analyzing the pain sensation in various patients, different characteristics of pain perception can be distinguished: hyperaesthesia, hyperalgesia, hypoalgesia, and allodynia [9].

The presence of gustatory changes and the fact that many patients with BMS are orofacial pain suggest an interaction between taste and nociceptive mechanisms, which would connect the sense of taste in the chorda tympani and/or glossopharyngeal nerves with oral pain in the central nervous system.

As an argument in support of the previous statements, there is the possibility that even the ascending pathways of taste and pain are common [7].

CONCLUSIONS

In BMS, due to the intersection of the gustatory and the pain sensation in the anatomical pathway presented above, taste disorders induce pain, be it atypical, under the aspect of subjective perception.

Therefore, it is hypothesized that at the level of the tympanic and/or glossopharyngeal nerves, there is an interaction between the nerve influx carrying information about taste and the nociceptive mechanisms, with the purpose of connection.

By means of qualitative and quantitative sensory tests and the subjective gustatory examination of each individual patient, it can be determined if there are neurosensory differences in patients with primary BMS and whether they coexist with gustatory and neurosensory deficits.

The hypothesis of an alteration in the gustatory system suggests that gustatory stimuli have an inhibitory influence on the trigeminal nociceptive system. Consequently, hypogeusia/ageusia due to peripheral nerve degeneration in patients with BMS could lead to reduced taste sensitivity. At the cortical level, this would result in central de-inhibition of the trigeminal nociceptive pathway, leading to faulty processing of transmitted information and a modified perception of painful sensitivity (increased) in the oral region.

Anatomical connections between gustatory sensitivity and oral pain support the idea that deterioration of the gustatory system might be associated with abnormal sensations, such as a burning oral sensation.

In explaining the pain mechanism of BMS, the role and significance of predisposing factors and conditions have been invoked. Thus, BMS could represent a sensation of oral pain induced in predisposed individuals through the deterioration of the gustatory system.

Conflict of interest: none declared
Financial support: none declared

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