applied sciences

MDPI

Review

Photobiomodulation as a Therapeutic Strategy in Burning Mouth Syndrome: A Scoping Review

Francesco Spadari [1,2], Federica Pulicari [1,2,*], Martina Ghizzoni [3], Massimo Porrini [1,2], Moreno Bosotti [1,2] and Matteo Pellegrini [1,2,*]

[1] Department of Biomedical Surgical and Dental Sciences, University of Milan, 20122 Milan, Italy; francesco.spadari@unimi.it (F.S.); massimo.porrini@unimi.it (M.P.); moreno.bosotti@unimi.it (M.B.)
[2] Maxillo-Facial and Odontostomatology Unit, Foundation IRCCS Ca' Granda Ospedale Maggiore Policlinico, 20122 Milan, Italy
[3] Section of Dentistry, Department of Clinical, Surgical, Diagnostic and Pediatric Sciences, University of Pavia, 27100 Pavia, Italy; martina.ghizzoni01@universitadipavia.it
* Correspondence: federica.pulicari@studenti.unimi.it (F.P.); matteo.pellegrini@unimi.it (M.P.)

Abstract: Burning mouth syndrome (BMS) is considered an atypical oral clinical-symptomatological condition because its etiopathogenesis is not yet fully clarified. It is mainly characterized by the symptom of burning, which occurs chronically and with various intensities. It is essential for making a diagnosis of BMS, clinical negativities, and instrumental investigations. It mainly affects the female sex, in the pre-post-climactic phases. A peripheral neuropathic matrix of the pain symptoms has been repeatedly demonstrated. However, this subjectivity is associated with personalities with anxiety-depressive traits, affective-behavioral difficulties, and disorders of the psycho-algogenic sphere. Numerous treatments are reported in the literature, which have rarely met lasting healing parameters. In this clinical landscape, photobiomodulation therapy (PBMT) can be considered a possible therapeutic alternative. Our study aims to present a scoping review of how photobiomodulation is used in BMS therapy and to analyze the outcome of the therapy. A literature review focused on the photobiomodulation treatment for burning mouth syndrome was conducted in the main scientific databases: PubMed, SCOPUS, and Web of Science. The results of our research highlight encouraging results regarding photobiomodulation, as in all studies, there is a reduction in symptoms.

Keywords: low-level laser therapy; photobiomodulation; burning mouth syndrome; neuropathic pain

Citation: Spadari, F.; Pulicari, F.; Ghizzoni, M.; Porrini, M.; Bosotti, M.; Pellegrini, M. Photobiomodulation as a Therapeutic Strategy in Burning Mouth Syndrome: A Scoping Review. *Appl. Sci.* **2023**, *13*, 8880. https://doi.org/10.3390/app13158880

Academic Editor: Bruno Chrcanovic

Received: 9 May 2023
Revised: 6 July 2023
Accepted: 31 July 2023
Published: 1 August 2023

1. Introduction

Burning mouth syndrome (BMS) is categorized as an idiopathic oral condition due to its yet-to-be-fully-understood etiology and pathogenesis, along with its atypical symptomatology and clinical characteristics [1–3]. In 1994, the International Association for the Study of Pain (IASP) identified three distinct groups of chronic oral and facial pain with neurogenic, vascular, and idiopathic origins.

Subsequently, the Headache Classification Committee of the International Headache Society (IHS) in 2013 and 2018, along with the International Classification of Orofacial Pain (ICOP) in 2020, defined BMS as "Intraoral burning or dysaesthesia sensation, recurring daily for more than 2 h per day over more than 3 months, without clinically evident causative lesions". A diagnosis of BMS is confirmed when individuals report oral burning symptoms without any observable clinical signs, and diagnostic investigations reveal a normal condition [2–4].

When oral symptomatology has a definable local and systemic cause, and locally there are clinical signs or instrumental tests that testify to a disease, it is correct to speak of secondary BMS [5,6].

Factors that can cause intraoral burning include iron, zinc, vitamin B12 or folic acid deficiencies, pharmacological causes, hyposalivation, Sjögren's syndrome, erosive

and ulcerative lesions of the mucosa, oral infections, inappropriate prostheses, and para-functional habits [5,6]. When we have an oral burning pain condition, without clinical signs and with an unknown origin, we will speak instead of primary BMS or idiopathic BMS [6–8].

The various etiological and pathophysiological aspects would lead to considering primary BMS as a complex multifactorial clinical condition, although it has also been associated with psychological and endocrinological disorders [9–12].

In the late 1980s, neurophysiological, neuropathological, and imaging studies led to the hypothesis that BMS could be a neuropathic pain condition [13–16]. Therefore, we can consider at least three lines of research that have characterized the studies on the etiology and pathogenesis of idiopathic BMS: a peripheral and central neurological basis, and an endocrinological and psychological matrix [8]. The pathophysiology of BMS is still unknown [8].

Three distinct subclasses of primary burning mouth syndrome (BMS) can be identified. The first subgroup (50–65%) is characterized by peripheral neuropathy affecting the small diameter fibers of the intra-oral mucosa. The second subgroup (20–25%) comprises patients with subclinical trigeminal lingual nerve system pathology, which clinically resembles the other two subgroups. The third subgroup (20–40%) aligns with the concept of central pain, potentially linked to the reduced function of dopaminergic neurons in the basal ganglia [8]. The development of neuropathic pain in primary BMS is thought to be closely related to small fiber damage. Recent studies in primary BMS have emphasized the role of small myelinated Aδ fibers and unmyelinated C fibers [15,17,18]. Additionally, approximately 50% of patients with primary BMS experience significant pain relief after lidocaine anesthesia of the lingual nerve, suggesting a peripheral origin of the pain [18]. The taste and pain neurological circuits appear to be intricately interconnected. Damage to the chorda tympani results in the inhibition of its function, leading to reduced control over the activity of other nerves. Consequently, deafferentation of the Aδ gustatory fibers of the chorda tympani nerve may result in lingual burning pain symptoms [19,20]. Alternatively, the diminished signaling of Aδ channels with preserved C-fiber function could also contribute to the burning pain experienced in primary BMS [8,21].

Patients with BMS often report dysgeusia or phantom taste sensations. [8,21–25]. In the pathogenesis of primary BMS, the possible involvements of the central nervous system related to the sensorium of the mouth and face regions have been considered [26,27]. Alterations in the basal ganglia would cause signs of dopaminergic system dysfunction, like Parkinson's disease, and bilateral oral pain distribution. The dopaminergic system has a pain-inhibitory role [25–30]. The blink reflex habituation, reflecting depletion of endogenous dopamine in the putamen, as is usually seen in Parkinson's disease, suggests the hypothesis that ineffective endogenous inhibitory pain control by the brain dopamine-opioid system predisposes the individual patient to chronic neuropathic pain [27,28]. Furthermore, patients with primary BMS report similar benefits from sleep duration as those with Parkinson's disease, due to increased brain dopamine tone during nighttime sleep. Also, patients diagnosed with major depression reported BMS, and this pathology has been associated with decreased brain dopamine. Genetic studies provide other insights into low brain dopamine tone in the etiology of BMS. The C957T dopamine D2 receptor polymorphism affects D2 receptor function in the striatum and influences the synaptic concentration of endogenous dopamine; homozygotes for the T allele have the lowest dopaminergic tone in the striatum and appear to be more common in patients with facial neuropathic pain, including primary BMS T alleles that are also found in patients with facial neuropathic pain, including primary BMS [29–32]. Sex hormones have neuroprotective effects and modulate the function of various neurotransmitters and peripheral receptors [33,34]. In 2009, it was highlighted that in subjects after menopause, there was a deficiency or dysfunction of adrenal steroids with decreased neuroprotective effects on neural tissues [13]. It has been seen that a deficiency of ovarian hormones would lead to an atrophy of the lining epithelia, including the lingual

and oral ones. Interestingly, this epithelial atrophy was observed in parallel with the reduction of unmyelinated C fibers [35–37]. The high prevalence of anxiety disorders and depression seen in BMS is associated with a history of chronic post-traumatic stress and chronic major anxiety leading to impaired adrenal steroid production. Among the various dysfunctional pain disorders, BMS is characterized by underlying hypercortisolism [38–40], which reaches 70% in primary BMS [41]. Initially, it was thought that dry mouth was only a subjective impression of BMS patients and not due to reduced actual salivary flow. However, it was possible to confirm that, compared to healthy subjects, the salivary flow was slightly reduced when unstimulated and did not change when stimulated with lemon [42]. It has been hypothesized that neuroprotective steroid deficiency leads to reduced function of the minor salivary glands and contributes to the induction of xerostomia. It has been confirmed that alterations of the emotional-instinctive and psycho-anxiety dimensions are conditions commonly encountered in BMS patients. However, no significant differences were found between the emotional and psychological characteristics of BMS patients and chronic pain patients [31,43–46]. Interestingly, most of the psychiatric and personality disorders associated with BMS have low brain dopamine tones in common. [47–51]. The purpose of our scoping review is to investigate whether photobiomodulation can be useful in the treatment of the disease and which protocols have been studied up to now.

2. Materials and Methods

2.1. Focused Questions

Can photobiomodulation be useful in the treatment of burning mouth syndrome? What are the protocols used in photobiomodulation therapy?

2.2. Eligibility Criteria

This review followed specific inclusion and exclusion criteria. The inclusion criteria were as follows: (I) the study model should be interventional studies, observational studies, cohort studies, or case series/case reports studies; (II) participants must be diagnosed with burning mouth syndrome; (III) the intervention under investigation should be photobiomodulation; and (IV) the outcome of interest should be clinical results for neuropathic pain treatment. Only studies that met all these inclusion criteria were considered in the review.

On the other hand, certain studies were excluded based on the following exclusion criteria: (I) articles published in non-English languages that were available only in abstract form; (II) duplicate studies that were identical or substantially overlapped with other included studies; (III) studies not relevant to the purpose of the full-text articles, or not suitable for addressing the focused questions, such as those analyzing different supplementary treatments or whose content did not match the abstract; (IV) ex vivo or experimental animal studies, which did not involve human participants; (V) studies lacking approval from an ethics committee; and (VI) narrative reviews, systematic reviews, or systematic and meta-analysis reviews, which were not in the scope of this particular review.

2.3. Search Strategy

In accordance with the JBI methodology for scoping reviews, the research process involved three main steps: (i) an initial limited search conducted on databases like PubMed (MEDLINE), Scopus, and Web of Science; (ii) selection of key terms from the retrieved articles to create an effective search strategy; and (iii) an additional search of the reference lists of all included articles to identify further relevant research.

Furthermore, the review followed the PCC model, which revolves around three essential elements: population (individuals undergoing PBMT procedures), concept (PBMT as a treatment for neuropathic pain), and context (without restricting the review to any specific cultural aspect or setting). The review focused on analyzing the

abstracts of studies that examined the effects of photobiomodulation procedures and their clinical outcomes.

Throughout the literature review process, the preferred reporting items for scoping reviews (PRISMA-ScR) consensus guidelines were adhered to (see Table S1, Supplementary Materials).

2.4. Research

The Medical Subject Heading (MeSH) terms used for the search included burning mouth syndrome, stomatopyrosis, photobiomodulation, photobiomodulation therapy, and low-level laser therapy. An electronic search was conducted on PubMed (MEDLINE), Scopus, and Web of Science databases, focusing on articles published between 2010 and 2023. The data extraction phase took place from February 2023 to April 2023, with the last search performed on 15 April 2023.

Four reviewers (F.P., M.Po., M.G., and M.Pe.) carried out the search, and any disagreements or discrepancies were resolved through consensus, with input from two additional reviewers (F.S. and M.B.). Titles and abstracts of the initially retrieved articles were thoroughly analyzed, and studies that were not relevant were excluded. Full texts of all relevant articles were reviewed and scrutinized, with findings documented, and similar studies meeting the inclusion criteria were identified. The present protocol has been registered on the Open Science Framework platform with the registration https://osf.io/a9hqu (accessed on 8 May 2023). The detailed strategies employed for each electronic database search are presented in Table S2 (Supplementary Materials).

2.5. Quality Assessment of Included Studies

A methodological quality risk of bias assessment was used in this review, JBI critical appraisal for randomized controlled trials.

3. Results

The primary search identified 186 articles based on MeSH terms. Following this, 160 articles were removed (5 abstracts of articles published in non-English languages, 98 duplicates, and 57 because they were not pertinent), and 26 articles were screened based on title and abstracts. The remaining 15 full-text articles were assessed for eligibility. Additionally, 4 full-text articles were further excluded because they were irrelevant articles. The 11 relevant articles were finally included and analyzed in this review. The flow chart of the review process is described in Figure 1.

Risk of Bias

The JBI critical appraisal tool was applied to assess the risk of bias in the studies included in this review (Table 1), using the judging criteria for risk of bias shown in Table S3 (Supplementary Materials).

Table 1. The risk of bias in randomized controlled trials is represented by symbols (green for low risk of bias, yellow for high risk of bias, and blue for uncertain or unavailable data and medium risk of bias).

Author and Year of Publication	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10	Q11	Q12	Q13	Overall Appraisal
Bardellini et al., 2019 [52]	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
De Pedro et al., 2020 [53]	✓	✓	✓	–	–	✓	✓	✓	✓	✓	✓	✓	✓	✓
Hanna et al., 2022 [54]	✓	✓	✓	✓	✓	–	✓	✓	✓	✓	✓	✓	✓	✓
Lončar-Brzak et al., 2022 [55]	✓	✓	✓	?	?	?	✓	✓	✓	✓	✓	✓	✓	✓
Scardina et al., 2020 [56]	✓	✓	✓	?	?	?	✓	✓	✓	✓	✓	✓	✓	✓
Sikora et al., 2018 [57]	✓	✓	✓	?	?	?	✓	✓	✓	✓	✓	✓	✓	✓
Spanemberg et al., 2015 [58]	✓	✓	✓	✓	–	✓	✓	✓	✓	✓	✓	✓	✓	✓
Sugaya et al., 2016 [59]	✓	✓	✓	?	?	?	✓	✓	✓	✓	✓	✓	✓	✓
Sun et al., 2021 [60]	✓	✓	✓	?	?	?	✓	✓	✓	✓	✓	✓	✓	✓
Arduino et al., 2016 [61]	✓	✓	✓	?	?	?	✓	✓	✓	✓	✓	✓	✓	✓
Valenzuela et al., 2017 [62]	✓	✓	✓	✓	?	?	✓	✓	✓	✓	✓	✓	✓	✓

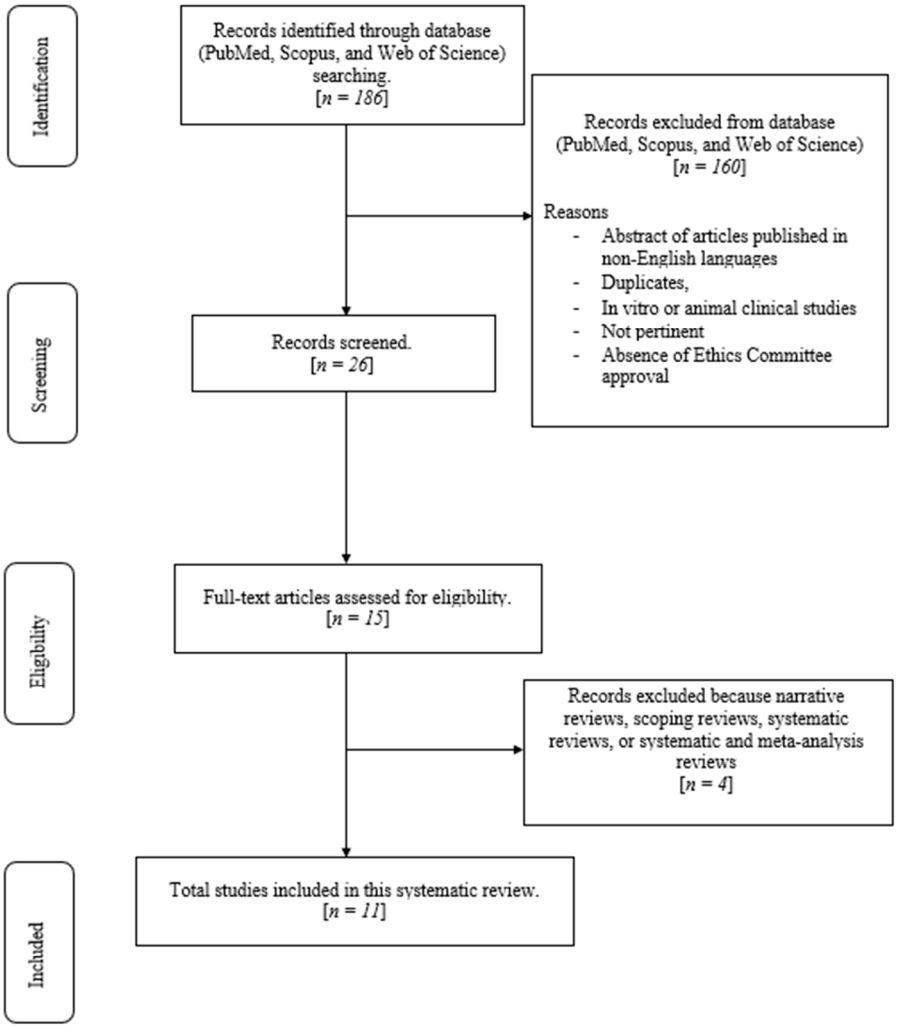

Figure 1. Flow chart of the review process.

4. Discussion

The response to treatment in patients with primary BMS is influenced by various pathological factors, including neuropathic components, central vulnerability, and psychiatric comorbidity. These problems are often intricate and interconnected, and addressing them requires a comprehensive approach rather than relying on a single therapy [50]. Like other neuropathic pain conditions, BMS poses challenges in treatment. On average, only 40% of patients experience benefits from their current neuropathic pain medications [51].

Another issue related to BMS treatment is the lack of consistent and continuous medication usage. While psychotropic medications show effectiveness in BMS treatment, approximately 15% of patients discontinue their medication. Implementing motivational interviewing can enhance appropriate adherence to medication [52,53]. Often, a combination of medications is recommended, along with treatments utilized for other neuropathic pain conditions [52,53]. Tricyclic antidepressants such as amitriptyline and clomipramine have been tried for BMS, with only 19% of patients reporting very poor outcomes and side effects such as dry mouth [54,55]. Antidepressants belonging to the group of selective serotonin reuptake inhibitors (SSRIs) such as chlordiazepoxide, diazepam, amisulpride, paroxetine, or sertraline hydrochloride have also been recommended with good results, but with the most side effects such as dizziness, insomnia, nausea, and somnolence [54,55].

Trazodone was not effective in improving BMS symptoms [56]. Anticonvulsants such as gabapentin and pregabalin are routinely recommended in the pharmacotherapy of BMS,

but therapeutic success has not been achieved [55,56]. Systemic and local treatment with clonazepam should be considered in the treatment of BMS.

Clonazepam is a benzodiazepine and a γ-aminobutyric acid (GABA) receptor agonist. This receptor is widely present in the central nervous system and peripheral tissues, and this drug acting on this receptor may have positive results in the treatment of this syndrome [55–58]. Pure small-fiber peripheral neuropathy may be better controlled with local clonazepam and central mechanisms may benefit more from systemic clonazepam [55–58]. Clonazepam, like other benzodiazepines, can cause side effects, mainly drowsiness, impairment of memory and cognitive functions, and dependence on long-term use. Although systemic absorption is low, no side effects have been reported with topical application [55–58].

α-lipoic acid (ALA) has been used to treat diabetic neuropathy because it is thought to act as a com- and coenzyme, produce energy (ATP), improve glucose metabolism and stimulate nerve growth factor (NGF) production. In addition, ALA stimulates the elevation of cellular glutathione levels and may prevent peripheral neuropathy [59–63].

There have been indications for the use of capsaicin in the pharmacological management of burning pain. Capsaicin induces desensitization and depletion of substance P leading to analgesia. A double-blind cross-over study revealed that a 0.025% capsaicin mouth rinse significantly reduced pain with no side effects [63–66]. Additionally, various topical treatments, such as saliva substitutes, may be helpful when a peripheral mechanism is suspected [66].

To talk about photobiomodulation, it is necessary to start from the assumption that light is a type of electromagnetic radiation and is considered an indispensable source of energy. A photochemical reaction is normally induced by the interaction of light, particularly ultraviolet, visible, and near-infrared light, with external matter. There are numerous examples of interactions between light and biological systems: photosynthesis; the reactions of the photoreceptors of the retina; the synthesis of vitamin D [67].

Since 2015, we have been talking about photobiomodulation (PBM) to describe the scientific basis of the use of non-ionizing electromagnetic energy to trigger photochemical changes within cellular structures and tissues [68].

PBM has analgesic and anti-inflammatory therapeutic effects, can stimulate biological tissues, and has documented antimicrobial effects [68].

Therefore, the correct use of the PBM can relieve painful symptoms, significantly reduce an inflammatory process, and accelerate and improve the healing of damaged tissues [69].

The mechanism of action of PBM is not yet fully understood. The most popular theory is that light is absorbed by mitochondrial cytochrome C oxidase (CcOx), causing an increase in adenosine triphosphate (Adenosine TriPhosphate ATP) [69,70].

PBM, on the other hand, can induce a brief rise in reactive oxygen species (ROS) before decreasing oxidative stress. Nitric oxide (NO) competes with oxygen and binds to CcOx in cells with varying degrees of hypoxia, limiting oxygen use and thus mitochondrial cell respiration. PBM may restore this inhibition by dissociating NO from its binding location on CcOx, allowing mitochondria to enhance ATP synthesis and cellular energy [69,70].

Another possible mechanism of PBM could be an increase in the concentrations of Ca^{2+} ions, important for cellular metabolism and homeostasis, and cellular signal transmissions. Furthermore, the cellular temperature changes after a PBM must be considered [67–73].

PBM is not considered a heat treatment; however, selective absorption by CcOx can lead to intracellular thermal micro-changes that can positively influence the behavior of cells and tissues [73,74].

The analgesic properties of PBM have been repeatedly recognized. It has been seen to reduce chronic low back pain, chronic neck pain, and other types of chronic pain such as BMS [49,74–79].

Furthermore, at the oral level, it has proved to be useful in preventing and treating oral mucositis in cancer patients, in reducing pain and swelling after third molar extraction,

in suffering from the temporomandibular joints, in orthodontic pain, and in reducing inflammation of the oral lichen [49,80–88].

Primary BMS is to be considered a chronic oral pain pathology where PBM could perform a part of the new local therapeutic strategies. Indeed, in recent years, with the diagnostic and cognitive advances of the disease, PBM has emerged as a potential non-invasive treatment with no apparent side effects.

Many therapeutic experiences have provided encouraging results in the reduction of oral burning symptoms. However, further therapeutic experiences will be needed to confirm and better protocol PBM treatments and correctly relate them to the clinical and symptomatic data of patients with BMS [57–60].

In the analysis of the results in the literature, there are some studies where an improvement in pain symptoms in patients with BMS has been found. Sleep values were compared and measured with the VAS and the improvement rates ranged from 4 to 15% [58,59,62].

The study conducted by Scardina et al. highlights the improvement in microcirculatory patterns after photobiomodulation was applied in patients with BMS, analyzed by videocapillaroscope. The improvement of the pattern has been noticed lasting for a long time after the therapy [56].

As regards the parameters used by the authors of the articles analyzed, the wavelengths used are variable, from a minimum of 685 nm [55] to a maximum of 1064 nm [38] with a preference; however, for the wavelength of 810–830 nm [52–54,56,57,59,62]. All the studies confirm the improvement of the symptoms. For the evaluation of the symptoms, the OHIP-14 questionnaires and the VAS scale were mainly used [52–62]. In fact, all the studies report an improvement in VAS scale values and OHIP values, especially after almost three applications of photobiomodulation. The follow-up shows a prolonged analgesic effect, which was found by Hanna et al., even nine months after discontinuation of treatment. In all studies, the absence of side effects of photobiomodulation is highlighted, even in follow-ups. The therapy is well accepted by the patients and easy to perform for the operator. Finally, it should be noted that the results of the improvement in the quality of life are recorded on average after seven weeks of application of the therapy, with an average of one session per week.

Table 2 shows the results of the literature review that satisfies the proposed research questions are summarized.

Table 2. Results of the literature review.

Author and Year of Publication	Type of Laser	Power	Wavelength	Application Method	Number of Application Spots	Time of Application	Number of Sessions	Questionnaires for Symptoms Evaluation	Outcome
Bardellini et al., 2019 [52]	Diode Laser	3.2 W	660–970 nm	Scanning method	Variable per patient, depending on the most painful points	3′ 51″ each spot	10 applications once a week	VAS, OHIP-14	Symptoms improvement
De Pedro et al., 2020 [53]	Diode Laser	0.6 W	810 nm	Scanning method, pulsed wave	56 points (3 in the vestibular mucosa of the 4 quadrants, 4 in each lip mucosa, 6 in each of the two buccal mucosa, 6 in the hard palate, 4 on each lateral edge of tongue, 6 in the dorsum of the tongue and 4 sublingual points, with a distance in between of 2 mm.)	10 s each spot	10 applications once a week	VAS, SF-36, OHIP-14, Epworth, SCL-90, McGill	Symptoms improvement
Hanna et al., 2022 [54]	Diode Laser	200 mW	810 nm	Scanning method, continuous wave	9 spots	30 s each spot	10 applications twice a week	EQ-5D-5L	Symptoms improvement
Lončar-Brzak et al., 2022 [55]	GaAlAs	30 mW	685 nm	Scanning method	3 spots	381 seach spot	10 applications once a day	OHIP-14, VAS	Symptoms improvement
Scardina et al., 2020 [56]	Diode Laser	4 W	800 nm	Scanning method, continuous wave	4 spots	300 seach spot	8 applications twice a week	VAS	Symptoms improvement, changes in microcircular pattern
Sikora et al., 2018 [57]	GaAlAs	100 mW	830 nm	Scanning method, pulsed wave	Variable per patient, depending on the most painful points	5 min each session	10 applications once a week	VAS, OHIP-14, OHIP-CRO-14	Symptoms improvement
Spanemberg et al., 2015 [58]	Diode Laser	100 mW and 35 mW	830 nm and 685 nm	Scanning method, continuous wave	apex of the tongue (3 points), side of the tongue (4 points), dorsum of the tongue (10 points), buccal mucosa (8 points), labial mucosa (5 points), hard palate (8 points), soft palate (3 points), and gums or alveolar ridge mucosa (3 points per sextant)	50–58 seach spot, 50 sfor 830 nm wave and 58 sfor 685 nm wave	10 applications once a week	OHIP-14	Symptoms improvement
Sugaya et al., 2016 [59]	GaAlAs	120 mW	790 nm	Scanning method, continuous wave	Variable per patient, depending on the most painful points	50 seach spot	4 applications twice a week	VAS	Symptoms improvement

Table 2. *Cont.*

Author and Year of Publication	Type of Laser	Power	Wavelength	Application Method	Number of Application Spots	Time of Application	Number of Sessions	Questionnaires for Symptoms Evaluation	Outcome
Sun et al., 2021 [60]	Nd: YAG	100 mW	1064 nm	Scanning method, pulsed mode	Variable per patient, depending on the most painful points	30 sper cm^2	4 applications once a week	VAS	Symptoms improvement
Arduino et al., 2016 [61]	GaAlAs	300 mW	980 nm	Scanning method, continuous wave	Variable per patient, depending on the most painful points	10 seach spot	5 applications once a week	VAS, McGill, OHIP-49, Hospital Anxiety and Depression Scale	Symptoms improvement
Valenzuela et al., 2017 [62]	GaAlAs	1 W	815 nm	Scanning method, continuous wave	Variable per patient, depending on the most painful points	6 seach spot	4 applications once a week	VAS, OHIP-14	Symptoms improvement

Abbreviations: EQ-5D-5L, European Quality of Life-5 Dimensions-5 Levels; McGill, McGill Pain Scale; OHIP-CRO-14, Oral Health Impact Profile-Croatian-14; OHIP-14, Oral Health Impact Profile-14; OHIP-49, Oral Health Impact Profile-49; SF-36, Short Form Health Survey-36; SCL-90, Symptom Checklist-90.

5. Conclusions

Various treatment options for BMS exist, but their efficacy based on evidence-based research remains unsatisfactory due to the diversity of studies and treatments employed. Among the treatments investigated, clonazepam and ALA have shown promising results in multiple studies, but further research with larger sample sizes is required to establish them as a first-line treatment for BMS patients. Notably, some studies have demonstrated similar outcomes between treatment and placebo evaluations, emphasizing the importance of exploring the psychological and/or psychiatric characteristics of patients. A multidisciplinary approach may be necessary for addressing BMS treatment. Photobiomodulation represents a potential therapeutic strategy for managing burning mouth symptoms. However, further investigation is warranted to fully understand its effectiveness in BMS treatment. Numerous studies on patients confirm that laser treatment can alleviate symptoms, with long-lasting effects. The advantages of this therapy are the easy execution by the clinician, the lack of adverse effects, and the good compliance by the patient. Despite this, it should be emphasized that it would be advisable to conduct future studies on larger patient samples; to standardize the operating protocols both in terms of therapeutic sessions, wavelengths to be used, and tests necessary to formulate a diagnosis precise; and correct classification of the symptomatology.

Supplementary Materials: The following supporting information can be downloaded at: https://www.mdpi.com/article/10.3390/app13158880/s1, Table S1: PRISMA-ScR Checklist; Table S2: Search strategies for electronic databases; Table S3: JBI critical appraisal checklist for randomized controlled trials, a tool used for risk of bias assessment.

Author Contributions: Conceptualization, F.S.; methodology, F.S.; software, M.P. (Matteo Pellegrini); validation, M.P. (Matteo Pellegrini), F.P. and M.P. (Massimo Porrini); formal analysis, M.P. (Massimo Porrini); investigation, F.P.; resources, M.G.; data curation, M.B.; writing—original draft preparation, F.S.; writing—review and editing, F.P.; visualization, M.B.; supervision, F.S.; project administration, M.P. (Matteo Pellegrini). All authors have read and agreed to the published version of the manuscript.

Funding: This research received no external funding.

Institutional Review Board Statement: Not applicable.

Informed Consent Statement: Not applicable.

Data Availability Statement: Upon request to the corresponding author, the data are available for use. The protocol of the review was registered with the Open Science Framework (OSF) with https://osf.io/a9hqu (accessed on 8 May 2023) and registered from osf.io/svkgn.

Conflicts of Interest: The authors declare no conflict of interest.

References

1. Merskey, H.; Bogduk, N. *Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms*, 2nd ed.; IASP Task Force on Taxonomy; IASP: Washington, DC, USA, 1994.
2. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache disorders, 3rd edition (beta version). *Cephalalgia* **2013**, *33*, 629–808. [CrossRef] [PubMed]
3. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition. *Cephalalgia* **2018**, *38*, 1–211. [CrossRef] [PubMed]
4. Orofacial Pain Classification Committee of the International Headache Society (IHS). International Classification of Orofacial Pain, 1st edition (ICOP). *Cephalagia* **2020**, *40*, 129–221. [CrossRef]
5. Nasri-Heir, C. Burning mouth syndrome. *Alpha Omega* **2012**, *105*, 76–81.
6. Scala, A.; Checchi, L.; Montevecchi, M.; Marini, I.; Giamberardino, M. Burning mouth syndrome: Overview and patient management. *Crit. Rev. Oral Biol. Med.* **2003**, *14*, 275–291. [CrossRef]
7. Jääskeläinen, S.K.; Woda, A. Burning mouth syndrome. *Cephalalgia* **2017**, *37*, 627–647. [CrossRef]
8. Jääskeläinen, S.K. Pathophysiology of primary burning mouth syndrome. *Clin. Neurophysiol.* **2012**, *123*, 71–77. [CrossRef]
9. Van der Ploeg, H.M.; van der Wal, N.; Eijkman, M.A.; van der Waal, I. Psychological aspects of patients with burning mouth syndrome. *Oral Surg. Oral Med. Oral Pathol.* **1987**, *63*, 664–668. [CrossRef]

10. Davies, S.; Underhill, H.C.; Abdel-Karim, A.; Christmas, D.M.; Bolea-Alamanac, B.M.; Potokar, J.; Herrod, J.; Prime, S.S. Individual oral symptoms in burning mouth syndrome may be associated differentially with depression and anxiety. *Acta Odontol. Scand.* **2016**, *74*, 155–160. [CrossRef] [PubMed]
11. Umezaki, Y.; Miura, A.; Watanabe, M.; Takenoshita, M.; Uezato, A.; Torihara, A.; Nishikawa, T.; Toyofuku, A. Oral cenesthopathy. *Biopsychosoc. Med.* **2016**, *10*, 20. [CrossRef]
12. Imamura, Y.; Shinozaki, T.; Okada-Ogawa, A.; Noma, N.; Shinoda, M.; Iwata, K.; Wada, A.; Abe, O.; Wang, K.; Svensson, P. An updated review on pathophysiology and management of burning mouth syndrome with endocrinological, psychological and neuropathic perspectives. *J. Oral Rehabil.* **2019**, *46*, 574–587. [CrossRef] [PubMed]
13. Woda, A.; Dao, T.; Gremeau-Richard, C. Steroid dysregulation and stomatodynia (burning mouth syndrome). *J. Orofac. Pain.* **2009**, *23*, 202–210.
14. Grushka, M.; Sessle, B.J.; Howley, T.P. Psychophysical assessment of tactile, pain and thermal sensory functions in burning mouth syndrome. *Pain* **1987**, *28*, 169–184. [CrossRef] [PubMed]
15. Lauria, G.; Majorana, A.; Borgna, M.; Lombardi, R.; Penza, P.; Padovani, A.; Sapelli, P. Trigeminal small-fibre sensory neuropathy causes burning mouth syndrome. *Pain* **2005**, *115*, 332–337. [CrossRef] [PubMed]
16. Hagelberg, N.; Forssell, H.; Rinne, J.O.; Scheinin, H.; Taiminen, T.; Aalto, S.; Luutonen, S.; Någren, K.; Jääskeläinen, S. Striatal dopamine D1 and D2 receptors in burning mouth syndrome. *Pain* **2003**, *101*, 149–154. [CrossRef]
17. Lauritano, D.; Spadari, F.; Formaglio, F.; Zambellini Artini, M.; Salvato, A. Aspetti eziopatogenetici, clinico-diagnostici e terapeutici della sindrome bocca bruciante. *Minerva Stomatol.* **1998**, *47*, 239–251.
18. Grémeau-Richard, C.; Dubray, C.; Aublet-Cuvelier, B.; Ughetto, S.; Woda, A. Effect of lingual nerve block on burning mouth syndrome (stomatodynia). *A randomized crossover trial. Pain* **2010**, *149*, 27–32.
19. Bartoshuk, L.M.; Snyder, D.J.; Grushka, M.; Berger, A.M.; Duffy, V.B.; Kveton, J.F. Taste damage: Previously unsuspected consequences. *Chem. Senses* **2005**, *30*, i218–i219. [CrossRef]
20. Kolkka-Palomaa, M.; Jääskeläinen, S.K.; Laine, M.A.; Teerijoki-Oksa, T.; Sandell, M.; Forssell, H. Pathophysiology of primary burning mouth syndrome with special focus on taste dysfunction: A review. *Oral Dis.* **2015**, *21*, 937–948. [CrossRef]
21. Imura, H.; Shimada, M.; Yamazaki, Y.; Sugimoto, K. Characteristic changes of saliva and taste in burning mouth syndrome patients. *J. Oral Pathol. Med.* **2016**, *45*, 231–236. [CrossRef]
22. Eliav, E.; Kamran, B.; Schaham, R.; Czerninski, R.; Gracely, R.H.; Benoliel, R. Evidence of chorda tympani dysfunction in patients with burning mouth syndrome. *J. Am. Dent. Assoc.* **2007**, *138*, 628–633. [CrossRef] [PubMed]
23. Nasri-Heir, C.; Gomes, J.; Heir, G.M.; Ananthan, S.; Benoliel, R.; Teich, S.; Eliav, E. The role of sensory input of the chorda tympani nerve and the number of fungiform papillae in burning mouth syndrome. *Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod.* **2011**, *112*, 65–72. [CrossRef] [PubMed]
24. Formaker, B.K.; Frank, M.E. Taste function in patients with oral burning. *Chem. Senses* **2000**, *25*, 575–581. [CrossRef]
25. Craig, A.D. How do you feel? Interoception: The sense of the physiological condition of the body. *Nat. Rev. Neurosci.* **2002**, *3*, 655–666. [CrossRef] [PubMed]
26. Le Pichon, C.E.; Chesler, A.T. The functional and anatomical dissection of somatosensory subpopulations using mouse genetics. *Front. Neuroanat.* **2014**, *8*, 21. [CrossRef]
27. Khalid, S.; Tubbs, R.S. Neuroanatomy and neuropsychology of pain. *Cureus* **2017**, *9*, e1754. [CrossRef] [PubMed]
28. Chen, Z.S.; Wang, J. Pain, from perception to action: A computational perspective. *iScience* **2022**, *26*, 105707. [CrossRef]
29. Kim, M.J.; Kho, H.S. Understanding of Burning Mouth Syndrome Based on Psychological Aspects. *Chin. J. Dent. Res.* **2018**, *21*, 9–19.
30. Kenchadze, R.; Iverieli, M.; Okribelashvili, N.; Geladze, N.; Khachapuridze, N. The psychological aspects of burning mouth syndrome. *Georgian Med. News.* **2011**, *194*, 24–28.
31. Taiminen, T.; Kuusalo, L.; Lehtinen, L.; Forssell, H.; Hagelberg, N.; Tenovuo, O.; Luutonen, S.; Pertovaara, A.; Jääskeläinen, S. Psychiatric (axis 1) and personality (axis 11) disorders in patients with burning mouth syndrome or atypical facial pain. *Scand. J. Pain* **2011**, *2*, 155–160. [CrossRef]
32. Jääskeläinen, S.K.; Lindholm, P.; Valmunen, T.; Pesonen, U.; Taiminen, T.; Virtanen, A.; Lamusuo, S.; Forssell, H.; Hagelberg, N.; Hietala, J.; et al. Variation in the dopamine D2 receptor gene plays a key role in human pain and its modulation by transcranial magnetic stimulation. *Pain* **2014**, *155*, 2180–2187. [CrossRef] [PubMed]
33. Coronel, M.F.; Labombarda, F.; González, S.L. Neuroactive steroids, nociception, and neuropathic pain: A flashback to go forward. *Steroids* **2016**, *110*, 77–87. [CrossRef] [PubMed]
34. Rupprecht, R.; Holsboer, F. Neuroactive steroids: Mechanisms of action and neuropsychopharmacological perspectives. *Neurosci. Trend.* **1999**, *22*, 410–416. [CrossRef]
35. Sardella, A.; Gualerzi, A.; Lodi, G.; Sforza, C.; Carrassi, A.; Donetti, E. Morphological evaluation of tongue mucosa in burning mouth syndrome. *Arch. Oral Biol.* **2012**, *57*, 94–101. [CrossRef]
36. Walf, A.A.; Frye, C.A. A review and update of mechanisms of estrogen in the hippocampus and amygdala for anxiety and depression behavior. *Neuropsychopharmacology* **2006**, *31*, 1097–1111. [CrossRef] [PubMed]
37. Lokuge, S.; Frey, B.; Foster, J.; Soares, C.; Steiner, M. Depression in women: Windows of vulnerability and new insights into the link between estrogen and serotonin. *J. Clin. Psychiatry* **2011**, *72*, e1563–e1569. [CrossRef] [PubMed]

38. Gunn, B.G.; Cunningham, L.; Mitchell, S.G.; Swinny, J.D.; Lambert, J.J.; Belelli, D. GABAA receptor-acting neurosteroids: A role in the development and regulation of the stress response. *Front. Neuroendocr.* **2015**, *36*, 28–48. [CrossRef]
39. Lamey, P.-J.; Freeman, R.; Eddie, S.-A.; Pankhurst, C.; Rees, T. Vulnerability and presenting symptoms in burning mouth syndrome. *Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod.* **2005**, *99*, 48–54. [CrossRef] [PubMed]
40. Koike, K.D.; Shinozaki, T.D.; Hara, K.D.; Noma, N.D.; Okada-Ogawa, A.D.; Asano, M.D.; Shinoda, M.D.; Eliav, E.D.; Gracely, R.H.; Iwata, K.D.; et al. Immune and endocrine function in patients with burning mouth syndrome. *Clin. J. Pain* **2014**, *30*, 168–173. [CrossRef]
41. Buchanan, J.; Zakrzewska, J. Burning mouth syndrome. *BMJ Clin. Evid.* **2010**, *7*, 1301.
42. Spadari, F.; Venesia, P.; Azzi, L.; Veronesi, G.; Costantino, D.; Croveri, F.; Farronato, D.; Tagliabue, A.; Tettamanti, L. Low basal salivary flow and burning mouth syndrome: New evidence in this enigmatic pathology. *J. Oral Pathol. Med* **2015**, *44*, 229–233. [CrossRef] [PubMed]
43. Agha-Hosseini, F.; Mirzaii-Dizgah, I.; Mansourian, A.; Khayamzadeh, M. Relationship of stimulated saliva 17β-estradiol and oral dryness feeling in menopause. *Maturitas* **2009**, *62*, 197–199. [CrossRef] [PubMed]
44. Agha-Hosseini, F.; Mirzaii-Dizgah, I.; Mirjalili, N. Relationship of stimulated whole saliva cortisol level with the severity of a feeling of dry mouth in menopausal women. *Gerodontology* **2012**, *29*, 43–47. [CrossRef] [PubMed]
45. Tokura, T.; Kimura, H.; Ito, M.; Nagashima, W.; Sato, N.; Kimura, Y.; Arao, M.; Aleksic, B.; Yoshida, K.; Kurita, K.; et al. Temperament and character profiles of patients with burning mouth syndrome. *J. Psychosom. Res.* **2015**, *78*, 495–498. [CrossRef]
46. Galli, F.; Lodi, G.; Sardella, A.; Vegni, E. Role of psychological factors in burning mouth syndrome: A systematic review and meta-analysis. *Cephalalgia* **2017**, *37*, 265–277. [CrossRef]
47. Al Quran, F.A. Psychological profile in burning mouth syndrome. *Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod.* **2004**, *97*, 339–344. [CrossRef]
48. Lamb, A.B.; Lamey, P.J.; Reeve, P.E. Burning mouth syndrome: Psychological aspects. *Br. Dent. J.* **1988**, *165*, 256–260. [CrossRef]
49. Pozza, D.H.; Fregapani, P.W.; Weber, J.B.; de Oliveira, M.G.; de Oliveira, M.A.M.; Neto, N.R.; de Macedo Sobrinho, J.B. Analgesic action of laser therapy (LLLT) in an animal model. *Med. Oral Patol. Oral Cir. Bucal* **2008**, *13*, E648–E652.
50. Jääskeläinen, S.K.; Rinne, J.O.; Forssell, H.; Tenovuo, O.; Kaasinen, V.; Sonninen, P.; Bergman, J. Role of the dopaminergic system in chronic pain—A fluorodopa-PET study. *Pain* **2001**, *90*, 257–260. [CrossRef]
51. Wood, P.B. Role of central dopamine in pain and analgesia. *Expert Rev. Neurother.* **2008**, *8*, 781–797. [CrossRef]
52. Bardellini, E.; Amadori, F.; Conti, G.; Majorana, A. Efficacy of the photobiomodulation therapy in the treatment of the burning mouth syndrome. *Med. Oral Patol. Oral Cir. Bucal* **2019**, *24*, e787–e791. [CrossRef] [PubMed]
53. De Pedro, M.; López-Pintor, R.M.; Casañas, E.; Hernández, G. Effects of photobiomodulation with low-level laser therapy in burning mouth syndrome: A randomized clinical trial. *Oral Dis.* **2020**, *26*, 1764–1776. [CrossRef] [PubMed]
54. Hanna, R.; Bensadoun, R.J.; Beken, S.V.; Burton, P.; Carroll, J.; Benedicenti, S. Outpatient Oral Neuropathic Pain Management with Photobiomodulation Therapy: A Prospective Analgesic Pharmacotherapy-Paralleled Feasibility Trial. *Antioxidants* **2022**, *11*, 533. [CrossRef]
55. Lončar-Brzak, B.; Škrinjar, I.; Brailo, V.; Vidović-Juras, D.; Šumilin, L.; Andabak-Rogulj, A. Burning Mouth Syndrome (BMS)-Treatment with Verbal and Written Information, B Vitamins, Probiotics, and Low-Level Laser Therapy: A Randomized Clinical Trial. *Dent. J.* **2022**, *10*, 44. [CrossRef] [PubMed]
56. Scardina, G.A.; Casella, S.; Bilello, G.; Messina, P. Photobiomodulation Therapy in the Management of Burning Mouth Syndrome: Morphological Variations in the Capillary Bed. *Dent. J.* **2020**, *8*, 99. [CrossRef] [PubMed]
57. Sikora, M.; Včev, A.; Siber, S.; Vučićević Boras, V.; Rotim, Ž.; Matijević, M. The Efficacy of Low-Level Laser Therapy in Burning Mouth Syndrome—A Pilot Study. *Acta Clin. Croat.* **2018**, *57*, 312–315. [CrossRef] [PubMed]
58. Spanemberg, J.C.; López López, J.; de Figueiredo, M.A.; Cherubini, K.; Salum, F.G. Efficacy of low-level laser therapy for the treatment of burning mouth syndrome: A randomized, controlled trial. *J. Biomed. Opt.* **2015**, *20*, 098001. [CrossRef]
59. Sugaya, N.N.; Silva, É.F.; Kato, I.T.; Prates, R.; Gallo, C.B.; Pellegrini, V.D. Low Intensity laser therapy in patients with burning mouth syndrome: A randomized, placebo-controlled study. *Braz. Oral Res.* **2016**, *30*, e108. [CrossRef]
60. Sun, C.; Xu, P.; Zhang, Q.-Q.; Jiang, W.-W. Nd:YAG photobiomodulation treatment in burning mouth syndrome: A pilot study. *Laser Dent. Sci.* **2021**, *5*, 53–60. [CrossRef]
61. Arduino, P.G.; Cafaro, A.; Garrone, M.; Gambino, A.; Cabras, M.; Romagnoli, E.; Broccoletti, R. A randomized pilot study to assess the safety and the value of low-level laser therapy versus clonazepam in patients with burning mouth syndrome. *Lasers Med. Sci.* **2016**, *31*, 811–816. [CrossRef]
62. Valenzuela, S.; Lopez-Jornet, P. Effects of low-level laser therapy on burning mouth syndrome. *J. Oral Rehabil.* **2017**, *44*, 125–132. [CrossRef] [PubMed]
63. Bushnell, M.C.; Ceko, M.; Low, L.A. Cognitive and emotional pain control and its interruption in chronic pain. *Nat. Rev. Neurosci.* **2013**, *14*, 502–511. [CrossRef] [PubMed]
64. Finnerup, N.B.; Attal, N.; Haroutounian, S.; McNicol, E.; Baron, R.; Dworkin, R.H.; Gilron, I.; Haanpää, M.; Hansson, P.; Jensen, T.S.; et al. Pharmacotherapy for neuropathic pain in adults: A systematic review and meta-analysis. *Lancet. Neurol.* **2015**, *14*, 162–173. [CrossRef]

65. Yamazaki, Y.; Hata, H.; Kitamori, S.; Onodera, M.; Kitagawa, Y. An open-label, noncomparative, dose escalation pilot study of the effect of paroxetine in treatment of burning mouth syndrome. *Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod.* **2009**, *107*, e6–e11. [CrossRef]
66. Maina, G.; Vitalucci, A.; Gandolfo, S.; Bogetto, F. Comparative efficacy of SSRIs and amisulpride in burning mouth syndrome: A single-blind study. *J. Clin. Psychiatry* **2002**, *63*, 38–43. [CrossRef]
67. Birkinshaw, H.; Friedrich, C.M.; Cole, P.; Eccleston, C.; Serfaty, M.; Stewart, G.; White, S.; Moore, R.A.; Phillippo, D.; Pincus, T. Antidepressants for pain management in adults with chronic pain: A network meta-analysis. *Cochrane Database Syst. Rev.* **2023**, *5*, CD014682.
68. Heckmann, S.M.; Heckmann, J.G.; Ungethüm, A.; Hujoel, P.; Hummel, T. Gabapentin has little or no effect in the treatment of burning mouth syndrome—Results of an open-label pilot study. *Eur. J. Neurol.* **2006**, *13*, e6–e7. [CrossRef] [PubMed]
69. Cui, Y.; Xu, H.; Chen, F.; Liu, J.; Jiang, L.; Zhou, Y.; Chen, Q. Efficacy evaluation of clonazepam for symptom remission in burning mouth syndrome: A meta-analysis. *Oral Dis.* **2016**, *22*, 503–511. [CrossRef]
70. Gremeau-Richard, C.; Woda, A.; Navez, M.L.; Attal, N.; Bouhassira, D.; Gagnieu, M.C.; Laluque, J.F.; Picard, P.; Pionchon, P.; Tubert, S. Topical clonazepam in stomatodynia: A randomized placebocontrolled study. *Pain* **2004**, *108*, 51–57. [CrossRef]
71. Rodrìguez de Rivera Campillo, E.; López-López, J.; Chimenos-Küstner, E. Response to topical clonazepam in patients with burning mouth syndrome: A clinical study. *Bull. Group. Int. Rech. Sci. Stomatol. Odontol.* **2010**, *49*, 19–29. [PubMed]
72. Marino, R.; Torretta, S.; Capaccio, P.; Pignataro, L.; Spadari, F. Different therapeutic strategies for burning mouth syndrome: Preliminary data. *J. Oral Pathol. Med. Off. Publ. Int. Assoc. Oral Pathol. Am. Acad. Oral Pathol.* **2010**, *39*, 611–616. [CrossRef]
73. Tirosh, O.; Sen, C.K.; Roy, S.; Kobayashi, M.S.; Packer, L. Neuroprotective effects of alpha-lipoic acid and its positively charged amide analogue. *Free Radic. Biol. Med.* **1999**, *26*, 1418–1426. [CrossRef] [PubMed]
74. Christy, J.; Noorani, S.; Sy, F.; Al-Eryani, K.; Enciso, R. Efficacy of alpha-lipoic acid in patients with burning mouth syndrome compared to that of placebo or other interventions: A systematic review with meta-analyses. *J. Dent. Anesth. Pain Med.* **2022**, *22*, 323–338. [CrossRef]
75. Femiano, F.; Scully, C. Burning mouth syndrome (BMS): Double blind controlled study of alpha-lipoic acid (thioctic acid) therapy. *J. Oral Pathol. Med.* **2002**, *31*, 267–269. [CrossRef] [PubMed]
76. Reyad, A.A.; Mishriky, R.; Girgis, E. Pharmacological and non-pharmacological management of burning mouth syndrome: A systematic review. *Dent. Med. Probl.* **2020**, *57*, 295–304. [CrossRef]
77. Silvestre, F.J.; Silvestre-Rangil, J.; Tamarit-Santafé, C.; Bautista, D. Application of a capsaicin rinse in the treatment of burning mouth syndrome. *Med. Oral Patol. Oral Cir. Bucal* **2012**, *17*, e1–e4. [CrossRef]
78. Vyklický, L.; Nováková-Toušová, K.; Benedikt, J.; Samad ATouška, F.; Vlachova, V. Calcium-dependent desensitization of vanilloid receptor TRPV1: A mechanism possibly involved in analgesia induced by topical application of capsaicin. *Physiol. Res.* **2008**, *57* (Suppl. S3), S59–S68. [CrossRef]
79. De Souza, F.T.; Amaral, T.M.; dos Santos, T.P.; Abdo, E.N.; Aguiar, M.C.F.; Teixeira, A.L.; Kummer, A.M.; Abreu, M.H.N.G.; Silva, T.A. Burning mouth syndrome: A therapeutic approach involving mechanical salivary stimulation. *Headache* **2012**, *52*, 1026–1034. [CrossRef] [PubMed]
80. Cronshaw, M.; Parker, S.; Arany, P. Feeling the heat: Evolutionary and microbial basis for the analgesic mechanisms of photobiomodulation therapy. *Photobiomodul. Photomed. Laser Surg.* **2019**, *37*, 517–526. [CrossRef]
81. Kato, I.T.; Pellegrini, V.D.; Prates, R.A.; Ribeiro, M.S.; Wetter, N.U.; Sugaya, N.N. Low-level laser therapy in burning mouth syndrome patients: A pilot study. *Photomed. Laser Surg.* **2010**, *28*, 835–839. [CrossRef]
82. Chow, R.T.; Johnson, M.I.; Lopes-Martins, R.A.; Bjordal, J.M. Efficacy of low-level laser therapy in the management of neck pain: A systematic review and meta-analysis of randomized placebo or active-treatment controlled trials. *Lancet* **2009**, *374*, 1897–1908. [CrossRef] [PubMed]
83. Zadik, Y.; Arany, P.R.; Fregnani, E.R.; Bossi, P.; Antunes, H.S.; Bensadoun, R.J.; Gueiros, L.A.; Majorana, A.; Nair, R.G.; Ranna, V.; et al. Systematic review of photobiomodulation for the management of oral mucositis in cancer patients and clinical practice guidelines. *Support. Care Cancer* **2019**, *27*, 3969–3983. [CrossRef] [PubMed]
84. Aras, M.H.; Güngörmüş, M. The effect of low-level laser therapy on trismus and facial swelling following surgical extraction of a lower third molar. *Photomed. Laser Surg.* **2009**, *27*, 21–24. [CrossRef] [PubMed]
85. Salmos-Brito, J.A.; de Menezes, R.F.; Teixeira, C.E.; Gonzaga, R.K.; Rodrigues, B.H.; Braz, R. Evaluation of low-level laser therapy in patients with acute and chronic temporomandibular disorders. *Lasers Med. Sci.* **2013**, *28*, 57–64. [CrossRef]
86. Doshi-Mehta, G.; Bhad-Patil, W.A. Efficacy of low-intensity laser therapy in reducing treatment time and orthodontic pain: A clinical investigation. *Am. J. Orthod. Dentofac. Orthop.* **2012**, *141*, 289–297. [CrossRef]
87. Akram, Z.; Abduljabbar, T.; Vohra, F.; Javed, F. Efficacy of low-level laser therapy compared to steroid therapy in the treatment of oral lichen planus: A systematic review. *J. Oral Pathol. Med.* **2018**, *47*, 11–17. [CrossRef]
88. Romeo, U.; Del Vecchio, A.; Capocci, M.; Maggiore, C.; Ripari, M. The low level laser therapy in the management of neurological burning mouth syndrome. *A pilot study. Ann. Stomatol.* **2010**, *1*, 14–18.