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Review

The Impact of Prophylactic Photobiomodulation Therapy on Reducing Oral Mucositis in Patients with Leukemia: A Narrative Review

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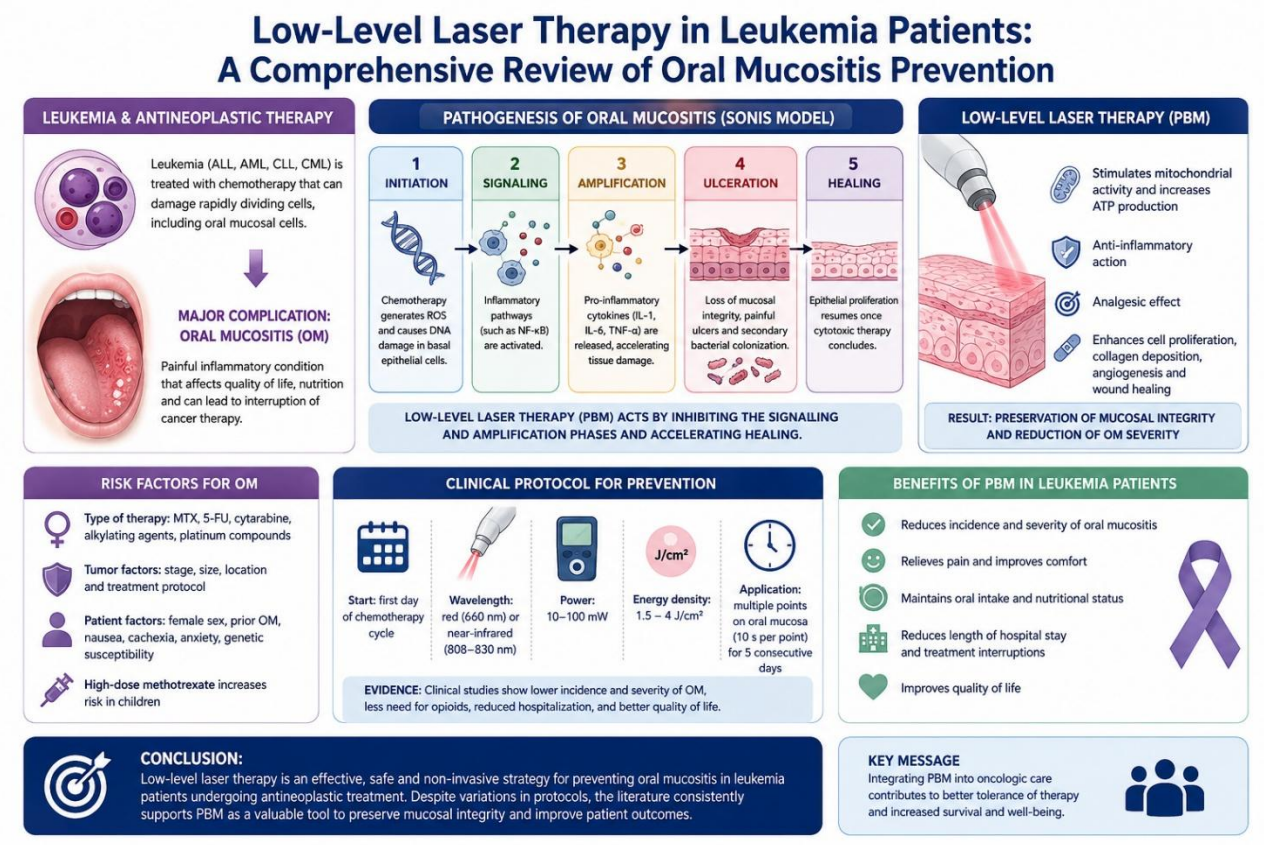
Abstract

Background and Objective: Leukemia is a malignant neoplasm characterized by disordered proliferation of blood cells originating in the bone marrow and lymphoid tissues. It can be classified into several subtypes; the four principal ones are acute myeloid leukemia (AML), chronic myeloid leukemia (CML), acute lymphoblastic leukemia (ALL), and chronic lymphoblastic leukemia (CLL). During antineoplastic therapy, the oral cavity is frequently affected because the drugs used cause tissue damage, stimulating the release of inflammatory cytokines and favoring the development of ulcerated and erythematous lesions. Among these manifestations, oral mucositis (OM) stands out as one of the most common and painful complications, significantly impacting patients' quality of life. OM is also a major dose-limiting factor and may lead to treatment interruption. Low-level laser therapy (LLLT), in turn, acts through photophysical and biochemical mechanisms that enhance cellular metabolism, stimulate mitochondrial activity, and exert analgesic, anti-inflammatory, and reparative effects on the mucosa. Thus, it represents an effective preventive measure with positive clinical results and is well-tolerated, non-toxic, and free of side effects. This study aimed to conduct a literature review on the impact of prophylactic laser therapy in reducing this condition. **Materials and Methods:** A literature search was conducted in PubMed and SciELO for articles published between 2020 and 2025. Boolean operators (AND, OR) were utilized to combine descriptors including "Low-Level Laser Therapy", "Oral Mucositis", and "Leukemia". The method included inclusion/exclusion criteria and search strategies. **Results:** Forty-three articles were initially identified, and after applying strict inclusion/exclusion criteria, twenty-five studies were selected for narrative synthesis. **Conclusion:** This review allows us to conclude that using laser therapy in leukemia patients undergoing antineoplastic treatment is a relevant strategy to prevent OM.

Clinical application: The benefits include longer survival, pain relief and control, shorter hospital stays, and improved quality of life. Regarding protocols, there is no standardization of preventive strategies, as application parameters may vary among professionals. However, the literature is generally consistent that – regardless of the technique employed – low-level laser therapy is effective in reducing OM.

Keywords: Low-level laser therapy, Oral mucositis, Leukemia

Graphical Abstract



1. Introduction

Leukemias are neoplasms that affect the hematopoietic system and arise from the uncontrolled proliferation of a cell clone with defects in differentiation and apoptosis. This abnormal growth replaces normal blood cells, resulting in malignant disease. [1,2] Although its etiology is unknown, it is believed that the neoplasm results from a combination of genetic and environmental factors such as smoking, excessive radiation exposure, hereditary diseases, and genetic syndromes. [3]

Leukemia can be classified as acute or chronic according to the maturation level of leukemic cells or the clinical course.[4] According to the lineage affected, it can be divided into Acute Myeloid Leukemia (AML) and Acute Lymphoblastic (or Lymphocytic) Leukemia (ALL).[5] Treatment generally includes cycles of chemotherapy (CT), central nervous system (CNS) prophylaxis, and, in specific cases, bone marrow transplantation.[6] Average duration ranges from 2 to 2.5 years and is divided into three stages: remission induction, intensification (or consolidation), and maintenance (or continuation). [7]

Oral mucositis (OM) is one of the most common and painful complications of antineoplastic therapy. It arises a few days after treatment begins and is characterized by erythema that may progress to ulcerations covered by pseudomembranes, which favor bacterial proliferation. Loss of mucosal integrity leads to extensive ulcers that can involve different areas of the oral cavity. It is estimated that approximately 40% of patients receiving chemotherapy develop OM.[8,9] For chemotherapy-induced OM, factors such as drug class, dose, frequency, and duration directly influence occurrence and severity. OM presents different severity grades; the more advanced the stage, the greater the overall clinical complications. [10]

In addition to causing severe pain and reducing the patient's quality of life, OM is a major dose-limiting toxicity. Severe OM frequently necessitates the delay, reduction, or complete discontinuation of chemotherapy, which directly and negatively impacts overall patient survival. To mitigate this, international guidelines, including those from the Multinational Association of Supportive Care in Cancer and the International Society of Oral Oncology (MASCC/ISOO), strongly recommend photobiomodulation (PBM) as a prophylactic intervention for high-risk patients. PBM utilizes specific wavelengths of light to trigger favorable biochemical changes, specifically enhancing adenosine triphosphate (ATP) production and deoxyribonucleic acid (DNA) repair, to prevent tissue breakdown.

In this context, low-level laser therapy (LLLT) shows promising results for OM management, acting effectively both in prevention and treatment. Several studies demonstrate that, when properly applied, it is a safe, well-tolerated, painless approach with no toxicity or side effects.[11] By emitting red light, LLLT modulates gene expression in relevant cells involved in microcirculation, anti-apoptotic pathways, antioxidant activity, and DNA repair. This cellular stimulus increases ATP production, accelerating tissue healing and repair synthesis.[12] Given that OM is one of the most common sequelae of antineoplastic therapy in patients with leukemia, this paper aims to highlight, through a literature review, the role of laser therapy in preventing and reducing the incidence and severity of OM. It addressed the techniques used and the therapeutic benefits, reinforcing their importance for patients' systemic status.

Despite these recommendations, there remains significant heterogeneity in clinical protocols and inconsistencies in the literature regarding the optimal dosimetry for leukemia patients specifically. This narrative review aims to synthesize the scattered clinical evidence, critically evaluate the discrepancies in current protocols, and highlight the impact of prophylactic laser therapy in this specific immunodeficient population.

2. Materials and Methods

2.1 Study Design

This narrative literature review was conducted to evaluate the prophylactic use of laser therapy in reducing oral mucositis in patients with leukemia. The search strategy utilized the following Boolean equations: ("Low-Level Laser Therapy" OR "Photobiomodulation") AND ("Oral Mucositis") AND ("Leukemia"). The search was conducted on November 26th, 2025, covering literature without date restriction, but in English and Portuguese. PubMed and SciELO were selected as they represent premier databases for high-quality peer-reviewed biomedical and Latin

American literature, respectively.

Initially, forty-three records were identified. After applying the inclusion and exclusion criteria (Table 1), the removal of duplicates and initial screening, twenty-five full texts were reviewed and included. Data extraction was performed independently by two reviewers (RFBR and GVOF), extracting variables such as laser type, technical parameters, target population, and clinical outcomes. Because this is a narrative review designed to provide a broad synthesis, no formal methodological quality assessment tool was applied to the included studies (Table 2).

Table 1. Selection criteria for references used.

Inclusion criteria	Exclusion criteria
- Articles in English and Portuguese languages.	- Articles outside the proposed theme; - Articles outside the 2020– 2025 period.

3. Results

3.1 Classification of Leukemias

Leukemia is a malignant hematologic neoplasm arising from mutations in hematopoietic progenitors, leading to accelerated, uncontrolled proliferation of immature leukocytes, disrupted differentiation, and evasion of apoptosis.[13] This pathogenic framework yields accumulation of abnormal cells in bone marrow and peripheral blood, constituting a malignant phenotype.[14] Clinically, leukemia is classified by cell lineage (myeloid vs lymphoid) and disease tempo (acute vs chronic), yielding four principal categories: AML, CML, ALL, and CLL, as widely described in epidemiologic and clinical reviews and consistent with categorizations referenced in the literature.[14-16] This review synthesizes evidence from multiple sources to support the claims and integrate the oral health context during antineoplastic treatment.

Table 2. Characteristics and main quantitative findings of the included clinical studies evaluating laser therapy and OM.

Study (Author, Year)	Design	Population / Sample Size	Intervention (Wavelength, Power, Dose)	Evaluated Outcomes	Key Quantitative & Clinical Findings
Alves et al. (2016)	Pilot Study	21 patients with ALL undergoing chemotherapy	LLLT for 5 consecutive days; Red light (660 nm or 808 nm); 100 mW; 4 J/cm²; 1 J; 10s per point	Incidence of OM, opioid use, hospital readmission	0 cases of grade 4 OM; mucositis lesions resolved rapidly within 4 days; 0 hospital readmissions due to OM
Castro et al. (2013)	Clinical Trial	40 patients with ALL receiving high doses of methotrexate	LLLT (660 nm or 830 nm); 100 mW; 35 J/cm² (prophylactic, 10s) vs 70 J/cm² (therapeutic, 20s)	Efficacy of prophylactic vs. therapeutic laser; Red vs. Infrared comparison	Prophylactic laser yielded better outcomes than no intervention; Red laser (660 nm) was more effective than infrared (830 nm)
Neves et al. (2021)	Clinical Study (Controlled)	24 pediatric patients receiving high-dose methotrexate.	Prophylactic red-light laser (InGaAlP, 660 nm, 100 mW, 1 J, 10s) vs. No laser.	Severity grades of OM (mild vs. severe).	Severe OM (Grades III–IV) occurred predominantly in the no-laser group; mild-to-moderate OM (Grades I–II) was

					more frequent in the treated group.
Cowen et al. (1997)	Randomized double-blind clinical trial	30 patients undergoing chemotherapy prior to bone marrow transplantation.	HeNe laser; 632.8 nm; 60 mW; 1.5 J/cm²; daily for 5 consecutive days.	Prevention and reduction of chemoradiotherapy-induced OM.	Successfully reduced the incidence of high-dose chemoradiotherapy-induced OM.
Andrade et al. (2022)	Comparative randomized trial	Oncologic patients with oral mucositis.	Photobiomodulation vs. curcumin antimicrobial photodynamic therapy.	Antimicrobial effect, analgesic effect, and alteration in OM degree	Demonstrated efficacy as a coadjuvant treatment for OM symptom relief.
Silva et al. (2015)	Clinical Study	Patients undergoing HSCT	LLLT	Impact on OM severity and patient QoL	Confirmed positive biological effects including accelerated tissue repair and improved quality of life metrics.
Curra et al. (2021)	Observational Cohort	Pediatric patients receiving chemotherapy.	N/A (Risk Factor Assessment).	Incidence and risk factors for OM development	Identifies high-dose Methotrexate, cachexia, and nausea as primary risk factors increasing OM incidence in pediatrics

ALL: acute lymphoblastic leukemia; LLLT: low-level laser therapy; InGaAlP: indium gallium aluminum phosphide; OM: Oral mucositis; QoL: Quality of Life; HSCT: hematopoietic stem cell transplantation; HeNe: Helium-Neon; N/A: not applicable.

AML is an aggressive acute leukemia arising from genetic mutations in the myeloid lineage, with rapid marrow-to-blood expansion of myeloblasts; it accounts for a large proportion of adult acute leukemias and has an increased incidence with age. It is described as aggressive with relatively low cure rates, accounting for approximately 80% of acute leukemias in adults, and is more common in older adults (e.g., >45 years) with male predominance in some cohorts. [14,16]

CML is characterized by a specific genetic lesion (notably BCR-ABL1), has a more insidious onset than AML, and a three-phase clinical course (chronic, accelerated, blastic). In the initial phase, altered cells may maintain relatively normal function; thus, diagnosis often occurs incidentally during routine tests, as many patients are asymptomatic. It can present in three phases: chronic (stable for 3–5 years), accelerated (splenomegaly and increased immature cells), and blastic (the most severe stage, with predominance of blasts in blood and bone marrow and aggressive evolution), hampering disease control. [14,15]

ALL is the most frequent malignant neoplasm in childhood, accounting for ~25% of cancers between 0 and 14 years and 85% of childhood leukemias; it is more common in males and Caucasians. Although the etiology is unknown, ALL is believed to arise from uncontrolled proliferation of lymphoblasts – immature precursors of the lymphoid lineage – primarily in the bone marrow, but also in the thymus and lymph nodes. [14,15]

CLL is characterized by progressive proliferation and accumulation of mature but functionally abnormal B lymphocytes in the bone marrow, peripheral blood, lymph nodes, and spleen. A mature B-cell neoplasm, most common in adults, particularly the elderly; characterized by accumulation of clonal CD5+ B cells in blood, marrow, and lymphoid tissues; progression is variable, with prognosis influenced by IGHV mutation status and other molecular features. It predominantly affects older adults; the mean age at diagnosis is ~70 years. Incidence rises significantly after age 50, is rare under 40, and extremely uncommon in pediatrics. [14-16] Across sources, CLL receives

particular emphasis as a prevalent adult leukemia with substantial discussion of prognostic markers (immunoglobulin heavy chain variable [IGHV] mutation status, TP53 status, cytogenetics) and treatment evolution (chemoimmunotherapy, BTK inhibitors, BCL-2 inhibitors). [17]

Oral health is closely linked to systemic balance, and this relationship becomes even more evident during antineoplastic treatments. These therapies can cause several adverse effects in the oral cavity, with oral mucositis being one of the most frequent and debilitating complications. In this scenario, the role of the hospital dentist is essential within the multidisciplinary team for the prevention, diagnosis, and management of oral changes and for promoting the quality of life of oncology patients.

3.2 Oral Mucositis

OM is an inflammation of the oral mucosa induced by antineoplastic treatments, including chemotherapy, head-and-neck radiotherapy, and hematopoietic stem cell transplantation (HSCT). [18,19] Clinically, it begins with erythema and edema and may progress to ulcerated lesions covered by pseudomembranes, usually accompanied by intense pain. It typically appears 7-10 days after the start of chemotherapy. [20] Among acute complications of cancer therapy, OM stands out for its high incidence and as a major dose-limiting factor during treatment.

The oral sites most frequently affected include the floor of the mouth, lateral borders and ventrum of the tongue, soft palate, buccal mucosa, commissures, and labial mucosa. Importantly, keratinized areas such as the hard palate, dorsum of the tongue, and gingiva are generally spared. [21] Ulcerated lesions, often with fissures, serve as portals of entry for oral microbiota, increasing the risk of serious infections such as bacteremia and sepsis, especially in neutropenic patients. [22]

Because advanced OM is challenging to manage, it interferes with basic functions such as eating, swallowing, and oral hygiene. These impairments directly affect communication, nutritional status, and consequently, quality of life. In many cases, a decline in overall condition necessitates temporary interruption of antineoplastic therapy, compromising continuity and effectiveness. [23,24] It is estimated that 9–19% of chemotherapy protocol interruptions are related to OM, directly compromising treatment efficacy. [24]

The World Health Organization (WHO) proposed a standardized OM grading system to facilitate communication among health professionals and to guide treatment choices based on severity. Grade 0: normal mucosa; Grade 1: mild erythema, no ulcers; Grade 2: more intense erythema with ulcers, but the patient can maintain a solid diet; Grade 3: painful, extensive ulcers limiting intake to liquids; Grade 4: the most severe stage, with deep ulcerations, necrosis, and intense pain, making oral feeding impossible. [25]

OM incidence is directly associated with the type of cancer treatment, tumor profile, and therapeutic protocol. Tumor stage, size, and anatomical location influence prognosis. [26] Regarding individual characteristics, women show a higher predisposition to OM with 5-fluorouracil (5-FU) or methotrexate (MTX) protocols. [27] Other antineoplastic agents, such as cytarabine, alkylating agents, and platinum compounds, can also trigger OM. In most cases, the possibility of OM occurrence is described in the package insert, making it essential to consult this document to identify risks associated with each patient's pharmacotherapy. [28]

The pathogenesis of OM can be understood through the five-phase Sonis model: 1. Initiation: chemotherapy generates reactive oxygen species, causing direct DNA damage to basal epithelial cells; 2. Signaling: inflammatory pathways (such as NF-κB) are activated; 3. Amplification: pro-inflammatory cytokines (IL-1, IL-6, TNF-α) are released, accelerating tissue damage; 4. Ulceration: the mucosal integrity is breached, leading to painful ulcers and secondary bacterial colonization; and 5. Healing: epithelial proliferation resumes once the cytotoxic therapy concludes. PBM directly counters this process by inhibiting the signaling and amplification phases through its anti-inflammatory effects and accelerating the healing phase via mitochondrial stimulation.

In pediatric oncology patients, the likelihood of OM is higher in the presence of nausea, cachexia, prior OM history, and elevated anxiety. There is also evidence for genetic susceptibility. [29] High-dose MTX use is associated with increased incidence in children undergoing cancer therapy. [30]

In this context, laser therapy plays a relevant role in OM prevention by promoting biological effects mediated by photophysical and biochemical processes. This approach increases cellular metabolism, thereby accelerating tissue repair and healing. [31]

3.3 Laser Therapy

Photobiomodulation therapy involves using lasers or non-coherent light sources, such as light-emitting diodes (LEDs), to positively modulate cellular metabolism. It is a non-thermal method in which the energy and power levels remain below thresholds that could cause adverse effects from tissue heating or mechanically induced cell damage. [32]

By stimulating mitochondrial activity, laser therapy exerts anti-inflammatory, analgesic, and wound-healing actions on mucosal lesions. Emitted energy is absorbed by both the superficial layer of adjacent tissue and the directly irradiated area, triggering epithelial and fibroblast proliferation, as well as cellular and vascular changes. Among the resulting effects are increased collagen and elastin production, wound contraction, enhanced macrophage phagocytosis, lymphocyte proliferation and activation, and increased tissue tensile strength, all of which accelerate healing. [31]

According to Ferraresi *et al*. (2014), laser irradiation stimulates mitochondrial activity, thereby increasing intracellular ATP production. This favors arachidonic acid synthesis and conversion of prostaglandins to prostacyclin – mechanisms that explain anti-edematous and anti-inflammatory effects.[33] The therapy also raises circulating endorphin levels, contributing to analgesia in inflammatory pain.

Currently, laser therapy is widely used across dental specialties, offering greater patient comfort and clinician safety. LLLT acts in both prevention and treatment of OM, helping the preserve mucosal integrity. It also provides relief of acute and chronic pain with an immediate, temporary analgesic effect. [34]

Clinical protocols primarily utilize red or near-infrared wavelengths (typically between 632 and 830 nm). Red lasers (e.g., 660 nm) are highly absorbed by superficial tissues, making them highly effective for mucosal lesions, while infrared lasers penetrate deeper for musculoskeletal

pain. Power usually ranges from 10 to 100 mW, with energy densities between 1.5 and 4 J/cm² proving most effective for prophylaxis. While traditional diode lasers provide focused, coherent light, LEDs (Light Emitting Diodes) offer non-coherent light that can cover broader tissue areas, though clinical efficacy remains highest with specific diode protocols. Beyond OM, LLLT is highly valuable in oncological dentistry for managing temporomandibular joint pain, accelerating healing post-extraction in stable phases, and treating other forms of oral ulceration.

3.4 Dental Management of the Patient with Leukemia

Managing patients with leukemia requires a multidisciplinary approach that includes not only the teams responsible for antineoplastic treatment but also professionals trained to treat secondary complications. The hospital dentist or oral care professional plays a fundamental role on this team because the oral cavity can be affected directly or indirectly by various pathologies and by the treatments themselves. [35]

Dental evaluation and care should be provided before, during, and after antineoplastic treatments to reduce the risk of oral infections and other related complications. [36] Treatment planning must consider the ongoing antineoplastic therapy to ensure integrated and safe management. As noted, OM is a major complication of leukemia treatment, although other oral alterations may also arise from the disease and the therapeutic procedures employed. [37]

Oral manifestations are often early signs of leukemia and occur more frequently during acute phases. [38] The early oral symptoms may result from direct leukemic cell infiltration and from the reduction of normal bone marrow components, especially during the acute phase. [39] These alterations include gingival bleeding, gingival hyperplasia, opportunistic infections, and bone changes. Leukemic cell infiltration can cause gingival edema – one of the most recurring clinical findings. Gingival hyperplasia is generally diffuse, with varying intensity among cases.

Patients with OM can benefit substantially from therapeutic laser in the management of this condition. In more severe cases, the dentist should prescribe opioid analgesics for adequate pain control. Lubrication of the oral cavity with artificial saliva helps prevent fissures, while topical corticosteroids reduce local inflammation. [22]

4. Discussion

OM is a multifactorial biological process that develops in sequential phases triggered by cytokine-mediated mechanisms. In the first phase, the vascular/inflammatory phase, there is the release of inflammatory cytokines such as interleukin (IL)-1, IL-6, and tumor necrosis factor (TNF)-α, local epithelial injury, increased vascularization, and accumulation of cytotoxic substances.[40,41] The second phase, the epithelial phase, is marked by the death of basal cells, leading to tissue atrophy and ulcer formation; chemotherapy interferes with the division of basal epithelial cells, reducing their renewal capacity. The third phase, the bacterial/ulcerative phase, is associated with neutropenia and bacterial colonization, which exacerbate ulceration. Finally, in the healing phase, tissue regenerates, and the epithelium is restored once cytotoxic agents are withdrawn. [42]

When OM reaches advanced stages, it can cause dysphagia, weight loss, and the need for enteral feeding via tubes. In certain situations, interruption of antineoplastic therapy is necessary, compromising disease control. Thus, preventing progression to critical stages is essential, and prophylactic low-level laser therapy is an effective strategy. [22]

Alves et al.[43] conducted a pilot study involving 21 patients with ALL undergoing chemotherapy. Treatment consisted of LLLT applied for 5 consecutive days starting on the first day of the chemotherapy cycle, with red light (660 nm or 808 nm), at 100 mW, an energy density of 4 J/cm², 1 J of energy, and an exposure time of 10 seconds per point. The study demonstrated a low incidence of OM in both groups, with no cases of grade 4 mucositis, opioid use, treatment interruption, or hospital readmission due to OM. Mucositis lesions resolved rapidly within 4 days, and laser therapy was well tolerated, with no reported side effects.

In another study, 40 patients with ALL who received high doses of methotrexate, were divided 2 groups: A - Preventive Group: preventive laser (red-subgroup A1 or infrared-subgroup A2) for 5 days; and B (Treatment Group): laser treatment only if they developed post-chemotherapy mucositis (red-subgroup B1 or infrared-subgroup B2) – laser of 660 or 830 nm wavelengths with output 100 mW, power density 3.57 W/cm(2), and energy of 1 J, resulting in an energy density of 35 J/cm(2) for 10 sec (prophylactic group), and energy of 2 J resulting in energy density of 70 J/cm(2) for 20 sec (therapeutic group). The authors concluded that prophylactic laser therapy yielded better outcomes than no preventive intervention, and that red laser (660 nm) was more effective than infrared (830 nm) in the prevention and treatment of OM. [44]

Neves et al.[45] evaluated 24 patients at the Barretos Childhood Cancer Hospital (Sao Paulo, Brazil): 12 received prophylactic red-light laser therapy (InGaAlP, 660 nm, 100 mW, 1 J, 10 s) and 12 did not. Severe OM (grades III–IV) occurred predominantly among those who did not receive laser, whereas mild-to-moderate grades (I–II) were more frequent in the treated group – supporting prophylactic laser to reduce MTX infusion-associated severity.

In a randomized double-blind clinical trial involving 30 patients undergoing chemotherapy prior to bone marrow transplantation, Cowen et al.[46] applied a Helium-Neon (HeNe) laser at 632.8 nm, 60 mW, and 1.5 J/cm² daily for 5 consecutive days. It reduced high-dose chemoradiotherapy-induced OM. Peralta-Mamani et al.[47] highlight that although no single ideal wavelength has been established, shorter ranges between 632 and 660 nm yield better results, favoring both tissue healing and pain relief, and are considered appropriate dosimetry parameters.

Al-Rudayni et al. [48] and Chaveli-López & Bagan-Sebastian [49] emphasizes that there is no single standardized protocol for OM management. The literature presents various preventive and therapeutic strategies, but no gold-standard intervention has been defined.

4.1 Critical Analysis and Limitations

While the synthesized studies demonstrate the clear benefits of PBM, critical analysis reveals significant heterogeneity in the implementation of these protocols. Discrepancies in outcomes can largely be attributed to variations in dosimetry parameters (wavelength, energy density, and

application time), the specific chemotherapeutic agents used (e.g., high-dose Methotrexate vs. 5-Fluorouracil), and the timing of the intervention.

A major limitation of the current body of evidence is that a vast majority of the cited studies focus heavily on pediatric populations with ALL and feature relatively small sample sizes. Consequently, there is a distinct lack of robust data regarding adult patients and other leukemia variants, such as AML. Furthermore, the lack of standardization in training and equipment costs remain significant barriers preventing the widespread clinical implementation of laser therapy. Future research must prioritize large-scale, multicenter randomized controlled trials with greater statistical power, focusing on adult cohorts to establish universal, standardized PBM guidelines.

5. Conclusion

Based on the literature reviewed, it is possible to conclude that: (1) the use of laser therapy in leukemia patients undergoing antineoplastic treatments is a relevant strategy for preventing oral mucositis, reducing its occurrence and severity; (2) among the benefits of prophylactic laser therapy, the following can be highlighted: (a) contribution to greater patient survival by preventing progression to advanced OM, avoiding treatment interruptions, and maintaining oral feeding, which is often compromised in severe cases; (b) pain relief and control; (c) reduced length of hospital stay; and (d) improved quality of life; (3) regarding adopted protocols, there is no standardization of preventive strategies, as application may vary among professionals. However, the literature generally agrees that, regardless of technique, LLLT is effective in reducing OM.

Abbreviation	Full Form
AML	Acute myeloid leukemia
CML	Chronic myeloid leukemia
ALL	Acute lymphoblastic leukemia
CLL	chronic lymphoblastic leukemia
OM	Oral mucositis
LLLT	Low-level laser therapy
5-FU	5-fluorouracil
MTX	methotrexate
IL	Interleukin
TNF	tumor necrosis factor
LED	Light emitting diode
WHO	World Health Organization
DNA	deoxyribonucleic acid
ATP	Adenosine triphosphate
IGHV	Immunoglobulin Heavy Chain Variable

Declarations:

Supplementary Materials: Not applicable.

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PR; Data curation: RFBR, JZ, GVOF, FBMS, PR; Writing-original draft preparation: RFBR, JZ, GVOF, FBMS, PR; Writing-review and editing: RFBR, JZ, GVOF, FBMS, PR; Visualization: RFBR, JZ, GVOF, FBMS, PR; Supervision: RFBR; Project administration: RFBR; Funding acquisition: ø. All authors have read and agreed to the published version of the manuscript.

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