



Laser Therapy Approaches in the Treatment of Kaposi's Sarcoma: A Comprehensive Review of Dermatologic Options

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Key message: Laser therapy, particularly pulsed dye and Nd:YAG lasers, offers effective, targeted treatment for Kaposi's sarcoma with minimal adverse effects, addressing both superficial mucosal and deeper nodular vascular lesions.

Keywords: Kaposi's sarcoma, Laser therapy, Pulsed dye laser, PDL, Nd:YAG laser

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ABSTRACT **Introduction:** Kaposi's sarcoma (KS) is a multicentric angioproliferative disease associated with human herpesvirus 8 and often exacerbated by immunosuppression. Effective treatment includes systemic and local therapies, with laser treatments gaining prominence in dermatological practice.

Objectives: This narrative review evaluated the efficacy and safety of laser treatment modalities in the management of cutaneous and mucosal KS lesions to guide dermatological decision-making.

Methods: A comprehensive English-language literature search was conducted using PubMed and Scopus up to November 2024. Search terms included "Kaposi's sarcoma", "laser therapy," and "laser treatment." Case reports and observational studies with defined patient characteristics, standardized laser protocols, and documented outcomes were included.

Results: The Nd:YAG laser emerged as the leading modality for thick, nodular KS lesions, demonstrating deep tissue penetration and high efficacy with minimal adverse effects. The pulsed dye laser (PDL) was highly effective for superficial cutaneous and mucosal lesions, with excellent cosmetic outcomes and minimal recurrence. The CO₂ laser showed promise in ablating small lesions but had limita-

tions due to scarring risks and potential biohazard concerns. Argon lasers were historically important but are now less favored due to lower specificity for vascular targets and higher risk of non-vascular tissue damage.

Conclusions: Laser therapy offers versatile and effective treatment options for KS. Nd:YAG and PDL lasers are particularly promising due to their high efficacy, favorable safety profiles, and low recurrence rates. Further research is needed to refine laser protocols and validate long-term outcomes in different patient populations.

1. Introduction

1.1 Definition and Classification

Kaposi's sarcoma (KS) is a multifocal angioproliferative disorder originating from lymphatic endothelial cells, with intermediate malignant potential. It is classified into four types: classic, endemic, iatrogenic, and epidemic, with an emerging category of KS in HIV-negative men who have sex with men (MSM) [1]. Regardless of the type, KS is caused by human herpesvirus 8 infection, with additional factors such as immunosuppression contributing to disease progression.

1.2 Clinical Presentation

KS primarily affects the skin, but mucosal surfaces, lymph nodes, and visceral organs may also be involved. Lesions appear as violaceous, reddish-blue, and/or dark brown macules, plaques, or nodules predominantly located on the lower limbs. Though typically asymptomatic, large or friction-exposed lesions may ulcerate, bleed, or impair function. Lymphedema, resulting from dermal lymphatic vessel infiltration, may accompany or precede lesions. Classic KS predominantly affects older Mediterranean or Eastern Europe males and generally follows an indolent course [2,3,4]. Epidemic or HIV-associated KS typically affects MSM in the fourth to fifth decades of life and is strongly linked to low CD4 counts [5]. Multiple cutaneous lesions are frequently observed, and the disease can progress rapidly, spreading to lymph nodes and visceral sites. Notably, it can also occur in patients with normal CD4 counts and in the context of immune reconstitution inflammatory syndrome [6,7]. Iatrogenic KS, linked to immunosuppressive therapy, predominantly affects solid organ transplant recipients and generally occurs within the first two years post-transplantation [8]. Iatrogenic KS and KS in HIV-negative MSM closely resemble the clinical presentation and course of classic KS [1,9].

1.3 Diagnosis and Staging

KS diagnosis requires histopathological confirmation, with blood tests and imaging to evaluate extracutaneous involvement. For epidemic KS, the AIDS Clinical Trial Group staging system categorizes patients into good or poor risk

prognostic groups based on tumor extent, immune status, and systemic illness severity [10]. For classic, endemic, and iatrogenic KS, the EDF/EADO/EORTC guidelines stratify patients into three categories: localized non-aggressive KS, locally aggressive KS, and disseminated KS [11].

1.4 Treatment

Given the absence of treatments capable of eradicating human herpesvirus 8 infection, a definitive cure is not achievable. The therapeutic goal focuses on alleviating symptoms, improving function, and preventing disease progression while maintaining the patient's quality of life. Systemic therapies are primarily indicated for advanced forms of KS, with pegylated liposomal doxorubicin as the first-line treatment, achieving response rates exceeding 70% [12]. Paclitaxel, etoposide, interferon-alpha, and immunotherapy are additional therapeutic options that have shown proven efficacy [13,14,15,16,17,18]. In HIV-related KS, the prompt initiation of antiretroviral therapy is mandatory, though complete remission is attained in only 50% of cases [19]. For localized KS, a wide range of local therapies is available. Surgical excision is effective for solitary lesions but unsuitable for extensive disease or multiple lesions, due to the risk of significant scarring and functional impairment [20]. Radiotherapy has complete response rates exceeding 90%, but complications such as fibrosis, ulceration, and a potential long-term risk of squamous cell carcinoma limit its use [21]. Intralesional vinblastine or vincristine shows response rates of 70% and 98%, respectively [22,23]. Nonetheless, multiple injections are required, and the procedure is associated with significant pain. Electrochemotherapy with bleomycin provides complete responses in over 70% of cases [24]. However, it is not universally available across centers, and adverse effects such as pain, ulceration, and infection have been reported [25]. Topical imiquimod 5% cream and alitretinoin 0.1% gel show moderate efficacy, with response rates of 50% and 37%, respectively [26,27]. However, the high cost, local adverse reactions, and limited effectiveness restrict their use. Cryotherapy, commonly employed for small, flat, or thin nodular lesions, achieves response rates up to 80% [28]. Although the procedure is straightforward and time efficient, it

requires multiple sessions and may cause erythema, blistering, scarring, and pigmentary changes. Among the various lesion-directed treatments, laser therapy has been attracting increasing attention in recent years and is gradually being incorporated into clinical practice.

2. Objectives

The aim of this narrative review was to collect existing data on the efficacy and safety of the various laser treatment modalities employed in the management of cutaneous and mucosal lesions in KS. The goal is to provide dermatologists with evidence-based guidance in selecting the most appropriate treatment.

3. Methods

A comprehensive English-language literature search was conducted using PubMed and Scopus throughout October 2024. The search strategy was implemented without any restrictions on the year of publication. Search terms included “Kaposi’s Sarcoma,” “KS,” “laser,” “laser treatment,” and “laser therapy.” Case reports and observational studies in which laser therapy was employed for the treatment of KS lesions in dermatological settings were included. Only studies with well-defined patient characteristics, standardized treatment protocols, and specified outcome assessment criteria were selected (Table 1).

4. Results and Discussion

4.1 Types of Laser Treatment Modalities

4.1.1 Carbon Dioxide Laser

The carbon dioxide (CO₂) laser emits 10,600 nanometers infrared light, absorbed by water molecules, causing tissue vaporization and ablation. Chun et al. treated a 54-year-old male with classic KS presenting with 12 papules and plaques on the glans and penile shaft using a single session of pulsed CO₂ laser. The procedure resulted in minimal intraoperative bleeding, with no scarring or recurrence at 12 months [29]. However, the evidence supporting the use of CO₂ laser for KS remains limited to this single case report identified in our research. The intrinsic limitations of the CO₂ laser may partly explain its restricted use in KS management. The CO₂ laser operates via a photothermal mechanism, targeting water as its primary chromophore. Each pass ablates approximately 20–60 μm of tissue, but thermal damage extends three to four times deeper, creating a residual thermal damage zone of up to 150 μm [30]. This characteristic enables collagen remodeling and wound healing but also increases the risk of scarring and pigmentary changes. In KS, lesions often extend into the

deep dermis and subcutaneous fat, necessitating deeper tissue ablation, which increases the risk of thermal injury to surrounding tissues. The non-selective nature of the CO₂ laser exacerbates this issue, as water content is uniformly present in both diseased and healthy tissue [31]. This is particularly problematic for patients with darker skin phototypes, where melanin absorption further amplifies thermal effects, heightening the risk of post-inflammatory hyperpigmentation or hypopigmentation [32]. Additionally, CO₂ laser treatment of virus-associated lesions such as KS can release viral particles as a result of the vaporization process [33]. This necessitates appropriate protective measures to minimize the risk of viral transmission to medical personnel. Notably, while fractional CO₂ lasers have been widely adopted for other dermatological conditions, including acne scars and photodamage, no study has specifically explored their application in KS [34]. Fractional technology, which spares untreated skin between columns of ablation, could theoretically reduce thermal injury and improve healing, but its efficacy in KS remains unproven.

4.1.2 Argon Laser

Historically, the argon laser was one of the first to be used effectively to treat cutaneous vascular lesions. Its blue-green light at wavelengths of 488 and 514 nanometers allowed for more selective targeting of hemoglobin compared to older non-laser techniques. In 1985, Wheeland et al. treated a 62-year-old male with classic KS presenting with papular and nodular lesions on the lower extremities over a five-year period. Complete regression occurred within 6–8 weeks, with lesion flattening, no scarring, and significant lymphedema improvement. Post-treatment biopsy showed no residual tumor, and no recurrence was reported during follow-up [35]. Although the argon laser targets the oxyhemoglobin absorption spectrum, its wavelengths fall within a suboptimal range, causing unintended thermal damage to non-vascular tissues, leading to scarring, textural changes, and hypopigmentation [36]. Additionally, early continuous wave technology lacked precise control over heat delivery, compounding the risks of thermal injury. The laser’s limited tissue penetration further restricted its efficacy in treating deeper lesions. With the introduction of selective photothermolysis in 1983, advanced laser systems have been developed to precisely target vascular and pigmented lesions while minimizing damage to surrounding tissues [37]. This concept has led to the refinement of pulsed laser technologies, which offer superior selectivity and safety compared to earlier continuous-wave systems. Consequently, the clinical use of argon lasers has declined, with contemporary studies focusing on more effective and less damaging alternatives [38]. Further research is required to determine whether modifications in argon laser technology could address its historical limitations and reestablish its clinical utility in dermatology.

Table 1. Summary and Comparison of Studies Using Laser Therapy to Treat KS.

Study	N	Age (years)/ Sex	KS type	Lesion type/ Localization	Laser type	Laser parameters	Sessions/ Intervals	Outcome	Adverse events	Recurrence	Follow-up period
Chun et al. (1999)	1	54/M	Classic	Papules and plaques on glans and penile shaft (12 lesions)	CO ₂	Pulsed mode; 7 W; 6 mm spot size	1 session	CR	Minimal procedural bleeding	No	12 months
Wheeland et al. (1985)	1	62/M	Classic	Papules and nodules on feet and lower legs	Argon	4 W; 0.5 s pulses; 2 mm spot size; 65 J/cm ²	3-4 month cycle for 2 years	CR; reduction of lymphedema	None	No	5 years
Tappero et al. (1992)	15	26-45/M	Epidemic	Papules on head and neck, trunk and extremities (39 lesions)	PDL	585 nm; 450 µs pulses; 5 mm spot size; 9.0 J/cm ²	Maximum of 4 sessions/ 4 weeks	CR or PR ⁺ in 44% of lesions	None	Yes, all lesions	12 weeks
Marchell et al. (1997)	1	30/M	Epidemic	Plaque on nose	PDL	585 nm; 450 µs pulses; 7 mm spot size; 6.5 J/cm ²	5 sessions/ 6 weeks	CR	None	No	12 months
Bassi et al. (2011)	1	35/M	Epidemic	Plaques on glans penis	PDL	595 nm; 0.5 ms pulses; 10 mm spot size; 6.5 J/cm ²	3 sessions/ 4-6 weeks	CR	None	No	12 months
Özdemir et al. (2017)	7	45-83 M (4) F (3)	Classic	Papulonodules predominantly on lower extremities (49 lesions)	Nd:YAG	1.5 pulse sequencing, 5 ms delay; 4-6 mm spot size; 260 J/cm ²	2-4 sessions/ 4 weeks	Clinical and histological improvement of all lesions; reduction of lymphedema of the lower limb in one patient	Mild atrophic scarring	No	6 months
Nasca et al. (2020)	9	64-86 M (7) F (2)	Classic	Nodules on feet and lower limbs (81 lesions)	Nd:YAG	5 ms triple pulse; 10 ms delay; 5-7 mm spot size; 140-200 J/cm ²	1-2 sessions/ 4 weeks	CR; reduction of lymphedema in one patient	Minimal scarring	No	12 months

Silvestri et al. (2022)	30	33-76 M (28) F (2)	Classic (15) Epidemic (15)	Macules, papules, plaques and nodules on trunk, arms, legs, feet, nose (174 lesions)	Nd:YAG	3 ms double pulse; 20 ms delay; 2.5-5 mm spot size; 120-140 J/cm ²	4 sessions/ 4 weeks	CR in 26.7% of patients; PR [‡] in 53.3% of patients	Mild hypotrophic scarring in 3 patients; post-inflammatory hyperpigmentation in 3 patients	No	12 weeks
Bostanci et al. (2024)	3	57-78 M (2) F (1)	Classic	Nodules on lower and upper extremities (42 lesions)	Nd:YAG	10-20 ms pulses; 3-7 mm spot size; 200-250 J/cm ²	1-2 sessions/ 4 weeks	Clinical and dermoscopic improvement of all lesions; reduction of lymphedema in one patient	Mild atrophic scarring	No	12 months
Junejo et al. (2024)	6	35-91 M (5) F (1)	Classic (2) Epidemic (3) Iatrogenic (1)	Plaques and papules on lower and upper extremities (51 lesions)	PDL Nd:YAG PDL + Nd:YAG	595 nm, 3-40 ms pulses; 5-10 mm spot size, 7-9.5 J/cm ² 20-25 ms pulses; 5 mm spot size; 90-140 mJ 595 nm; 10 ms pulses; 10 mm spot size; 10 J/cm ² ; 20 ms pulses; 5 mm spot size; 90 mJ energy	3-12 sessions/ 6 weeks 4 sessions/ 6 weeks 2 sessions/ 6 weeks	SI* (1) Mod. Imp. [‡] (1) Mild Imp. [‡] & (1) LFU (1) SI CI [#]	None (4) Edema (1) None Edema and blistering cellulitis	No	7-61 months 35 months 27 months

Abbreviations: N: number of patients; M: male; F: female; KS: Kaposi's sarcoma; CO₂: carbon dioxide; ICG: indocyanine green; PDL: pulsed dye laser; Nd:YAG: neodymium-doped yttrium aluminum garnet; CR: complete response; PR: partial response; Mild Imp.: mild improvement; Mod. Imp.: moderate improvement; SI: significant improvement; CI: complete improvement; LFU: lost to follow-up; W: Watts; W/cm²: watts per square centimeter; mJ: millijoules; J/cm²: joules per square centimeter; min: minutes; s: seconds; ms: milliseconds; μs: microseconds; mm: millimeters; nm: nanometers.

CR: total regression and absence of any detectable residual disease

[‡] Defined as 50% or greater decrease in lesion's size ([‡]and/or complete flattening of a nodule/plaque)

[#] Defined as at least two of the following criteria: 50% reduction in lesion's diameter, 50% reduction in the total number of lesions, flattening of representative elevated lesions

^{*} Defined as 76-100% lesion reduction

[‡] Defined as 51-75% reduction in the lesion's color intensity/size/texture

[‡] Defined as 26-50% reduction in the lesion's color intensity/size/texture

[‡] Defined as 1-25% reduction in the lesion's color intensity/size/texture

4.1.3 Pulsed Dye Laser

The pulsed dye laser (PDL) is a non-ablative laser widely used for the treatment of superficial vascular lesions. It operates at wavelengths ranging between 585 and 600 nanometers, targeting hemoglobin as its primary chromophore. A study involving 15 patients with epidemic KS demonstrated that the 585-nm PDL achieved a complete or partial response in 44% of treated papular lesions. However, recurrence occurred in all cases by 12 weeks, indicating the laser's limited penetration into deeper dermal layers. Histopathological analyses confirmed residual tumor cells in treated areas [39]. Marchell et al. reported a case of a 30-year-old HIV-positive male with a nasal KS plaque treated with five 585-nm PDL sessions, leading to complete clearance without scarring, hyperpigmentation, or recurrence at one year follow-up [40]. Another study described a 35-year-old HIV-positive male with disseminated KS, resistant to systemic liposomal doxorubicin, who underwent three 595-nm PDL sessions for purpuric plaques on the glans mucosa. Complete remission was achieved, with no relapse at 12 month follow-up [41]. A recent study by Junejo et al. evaluated the effectiveness of non-ablative laser therapy in six patients with refractory KS; four patients underwent a 595-nm PDL treatment, receiving between three and 12 sessions for plaques and papules on the upper and lower extremities. One patient was lost to follow-up. In the remaining cases, no recurrence was observed over a follow-up period ranging from seven to 61 months, and all patients demonstrated clinical improvement. Transient edema was reported in one patient, while the others reported no adverse event [42]. The PDL demonstrates strong selectivity for hemoglobin, rendering it particularly advantageous for treating KS due to the vascular nature of the disease. The procedure is generally well-tolerated and often does not require anesthesia. Its low incidence of scarring and pigmentary changes supports its safe application in sensitive areas. Additionally, as a non-ablative modality, the PDL minimizes occupational exposure to virus-infected blood or tissue products for healthcare providers. However, despite these benefits, the limited penetration depth of PDL—approximately 1.2 mm—restricts its effectiveness to superficial lesions and thin dermal regions, where it can photocoagulate vessels up to 100 μ m in diameter [43]. The limited penetration depth of PDL significantly restricts its efficacy in treating nodular or deeply infiltrating KS lesions, where the vascular component extends beyond the laser's effective range. In such cases, residual tumor cells often persist, increasing the likelihood of recurrence due to incomplete lesion clearance. Histopathological evidence supports this observation, indicating that deeper lesions are prone to recurrence following PDL therapy as a result of insufficient energy delivery to the lower dermis and subcutaneous tissue [39]. Integrating systemic therapy with PDL treatment may

enhance therapeutic outcomes, particularly in refractory KS cases. While systemic interventions target the broader disease burden, PDL therapy can effectively address localized, superficial cutaneous and mucosal lesions. This multimodal approach has been suggested to improve lesion resolution rates in patients with widespread or treatment-resistant KS [40,41].

4.1.4 Neodymium-Doped Yttrium Aluminum Garnet Laser

The neodymium-doped yttrium aluminum garnet (Nd:YAG) laser, with its 1064-nanometer wavelength, effectively targets hemoglobin, providing deep tissue penetration for treating thick vascular lesions while minimizing epidermal damage [44]. A preliminary study evaluated the long-pulse Nd:YAG laser in seven patients with classic KS, with lesions predominantly on the lower extremities. While small lesions responded well to a single session, larger, clustered, or multicentric lesions required up to four treatments. Clinical and histopathological improvements were observed, with mild atrophic scarring as the only noted complication. Additionally, one patient experienced a consistent reduction in lymphedema. At the six-month follow-up, no recurrence was observed in any of the patients [45]. Nasca et al. introduced a tilted-angle delivery technique in their study of nine patients with classic KS, presenting with a total of 81 nodular lesions on the lower extremities. This technique involves delivering the laser at an oblique angle, ranging from 30° to 60°, directed circumferentially around each lesion. One to two sessions led to complete lesion resolution at 12 months, with minimal scarring, and one patient showed lymphedema improvement [46]. In a retrospective study, Silvestri et al. investigated the efficacy of the Nd:YAG laser in a cohort of 30 patients, comprising 15 with classic KS and 15 with epidemic KS. After four treatment sessions, clinical improvement was observed in 80% of cases. Notably, greater efficacy was observed in the epidemic KS group, where patients demonstrated a median reduction in lesion diameter of 87% compared to 66% in the classic KS group. Additionally, 87.5% of epidemic KS patients showed flattening of elevated lesions, in contrast to 55.6% in the classic KS group. Treatment demonstrated a favorable safety profile, with only mild hypotrophic scarring and post-inflammatory hyperpigmentation observed in few cases [47]. A plausible explanation for the superior therapeutic response observed in epidemic KS is the higher vascular density compared to classic KS, which may enhance greater laser absorption and coagulative effects, leading to more pronounced lesion regression [48]. Additionally, the heightened inflammatory response and increased vascular endothelial growth factor expression in epidemic KS may contribute to improved treatment outcomes by accelerating endothelial remodeling and lesion resolution [49]. A more recent study

assessed the effectiveness of long-pulsed Nd:YAG laser therapy in three patients with advanced-stage classic KS, targeting a total of 42 nodular lesions on the lower and upper extremities. Clinical and dermoscopic evaluations revealed significant improvements, with minimal atrophic scarring as the only noted complication. Additionally, a reduction in lymphedema was observed in one patient. No recurrence was noted during the one-year follow-up [50]. The study by Junejo et al. involved six patients with classic, epidemic, or iatrogenic KS who had previously failed at least four therapies. Four patients received PDL alone, as previously outlined. Of the remaining two patients, one with plaques on the lower extremities was treated with Nd:YAG laser, achieving significant improvement after four sessions, with no adverse event and no recurrence at 35-month follow up. The second patient, presenting with thick, indurated plaques on the lower and upper extremities, received PDL followed by Nd:YAG laser treatment, resulting in an almost complete resolution, with no recurrence observed at 27 months [42]. The Nd:YAG laser demonstrates significant efficacy in the treatment of thicker, deeply seated KS lesions. Its wavelength enables a penetration depth of up to 5 mm, allowing effective treatment of deep, large-caliber vessels [51]. The lower melanin absorption at 1064 nm permits higher fluence applications with minimal epidermal damage, making this modality safe across diverse skin phototypes. The Nd:YAG laser has also demonstrated potential in lymphedema reduction. This effect may be attributed to laser-induced hypoxia, which can elevate vascular endothelial growth factor levels, promoting lymphangiogenesis and improving lymphatic flow [52]. Additionally, the thermal effects of the laser may facilitate the

breakdown of fibrotic tissue, enhancing lymphatic drainage and reducing fluid accumulation [53]. The laser's immunosuppression-free nature, rapid application without bleeding, and potential for long-term remission underscore its therapeutic value in both HIV-positive patients and frail elderly individuals with classic KS. Despite its advantages, Nd:YAG therapy presents potential risks, particularly for immunocompromised patients. Patients in these groups may be at higher risk for delayed wound healing, secondary infections, and potential KS reactivation due to laser-induced inflammatory responses [54]. Therefore, close monitoring and individualized treatment planning are necessary to optimize safety and efficacy.

4.2 Summary and Overview of Different Laser Treatment Modalities

The selection of an appropriate laser modality depends on patient- and lesion-specific factors, including phototype, lesion depth, size, and anatomical location. A comparative summary of the characteristics and applications of different laser treatment modalities is presented in Table 2.

4.3 Limitations

Several limitations must be considered when evaluating laser therapy for KS in dermatological practice. The lack of comparative studies with established skin-directed treatments hinders a clear assessment of relative efficacy, recurrence rates, and long-term outcomes. Cost remains a concern, as laser therapy requires specialized equipment and multiple sessions, limiting accessibility, especially in resource-constrained

Table 2. Comparison between Laser Therapies to Treat KS.

Laser Type	Wavelength (nm)	Technology/Mode of Action	Indicated Lesion Types	Comments
CO ₂	10,600	Ablative/ Photothermal vaporization	Small and superficial papules and plaques	Effective for mucosal lesions; one session may be sufficient; Anesthesia required; risk of recurrence if ablation is incomplete; risk of scarring and dyspigmentation; biohazard risk due to aerosolization; Not recommended in darker skin phototypes
Argon	488-514	Continuous wave/ Non-selective photothermal coagulation	Superficial papules and nodules	Historically effective, now obsolete; Multiple sessions required; high risk of scarring and dyspigmentation due to non-selective photothermal interaction and continuous-wave emission
PDL	585-600	Non-ablative/ selective photocoagulation	Macules and superficial papules and plaques	Suitable for mucosal and sensitive areas; negligible risk of scarring; excellent cosmetic outcome; Multiple sessions required; high recurrence for deeper lesions due to limited penetration
Nd:YAG	1064	Non-ablative/ selective photocoagulation	Deep Papules and Nodules	Very low risk of recurrence; safe across different skin phototypes; effective for lymphedema; Multiple sessions required; risk of mild scarring and hyperpigmentation

CO₂: carbon dioxide; PDL: pulsed dye laser; Nd:YAG: neodymium-doped yttrium aluminum garnet; nm: nanometers.

settings. Cryotherapy and intralesional vinblastine/vincristine are cost-effective, with relatively high response rates, but direct cost comparisons are lacking. Unlike radiotherapy and electrochemotherapy, which require specialized centers, laser therapy can be performed in outpatient settings with minimal adverse effects, potentially reducing healthcare costs. While some reports integrate laser therapy with antiretroviral therapy and systemic chemotherapy, long-term outcomes of such combined approaches remain unstudied [39,41,47]. Systemic therapy reduces overall disease burden, while laser therapy targets refractory cutaneous lesions, suggesting a synergistic potential requiring further investigation. Study heterogeneity, small sample sizes, retrospective designs, and publication bias further limit the generalizability of current findings.

5. Conclusions

Laser therapy represents a versatile, effective treatment option for managing cutaneous and mucosal KS lesions. The CO₂ laser is suitable for small and superficial KS lesions, though deeper penetration increases scarring risk. Viral particles in laser-generated plume pose a biohazard concern. The argon laser, once popular, is now less favored due to its relatively low specificity for hemoglobin. The PDL has demonstrated high efficacy in treating superficial cutaneous and mucosal lesions, providing excellent cosmetic results. However, its limited penetration restricts its effectiveness in deeper lesions. The Nd:YAG laser has emerged as a preferred modality for treating thicker KS lesions and has also demonstrated efficacy in reducing lymphedema. Laser treatments for KS are generally well-tolerated, fast, and can be performed in outpatient settings. The Nd:YAG and PDL lasers, in particular, are emerging as leading options due to their high efficacy, minimal scarring and pigmentation changes, favorable safety profiles, and low recurrence rates. The non-ablative nature of these lasers minimizes bleeding, promotes rapid healing, and reduces occupational exposure risks for healthcare providers handling virus-infected tissues. Despite promising results, high-quality evidence remains limited, and studies directly comparing laser therapy with established skin-directed treatments remain unexplored. Future research should prioritize randomized controlled trials stratifying patients by KS subtype, lesion characteristics, and immune status to establish the precise role of laser therapy in KS management.

References

1. Denis D, Seta V, Regnier-Rosencher E, et al. A fifth subtype of Kaposi's sarcoma, classic Kaposi's sarcoma in men who have sex with men: a cohort study in Paris. *J Eur Acad Dermatol Venereol*. 2018;32(8):1377-1384. DOI:10.1111/jdv.1483. PMID: 29377280
2. Marcoval J, Bonfill-Ortí M, Martínez-Molina L, Valentí-Medina F, Penín RM, Servitje O. Evolution of Kaposi sarcoma in the past

- 30 years in a tertiary hospital of the European Mediterranean basin. *Clin Exp Dermatol*. 2019;44(1):32-39. DOI:10.1111/ced.13605. PMID: 29934954
3. Dal Maso L, Polesel J, Ascoli V, et al. Classic Kaposi's sarcoma in Italy, 1985-1998. *Br J Cancer*. 2005;92(1):188-193. DOI:10.1038/sj.bjc.6602265. PMID: 15570306
4. Stratigos JD, Potouridou I, Katoulis AC, et al. Classic Kaposi's sarcoma in Greece: a clinico-epidemiological profile. *Int J Dermatol*. 1997;36(10):735-740. DOI:10.1046/j.1365-4362.1997.00284.x. PMID: 9372346
5. Lodi S, Guiguet M, Costagliola D, et al. Kaposi sarcoma incidence and survival among HIV-infected homosexual men after HIV seroconversion. *J Natl Cancer Inst*. 2010;102(11):784-792. DOI:10.1093/jnci/djq134. PMID: 20442214
6. Maurer T, Ponte M, Leslie K. HIV-associated Kaposi's sarcoma with a high CD4 count and a low viral load. *N Engl J Med*. 2007;357(13):1352-1353. DOI:10.1056/NEJMc070508. PMID: 17898112
7. Poizot-Martin I, Bréigéon S, Palich R, et al. Immune Reconstitution Inflammatory Syndrome Associated Kaposi Sarcoma. *Cancers (Basel)*. 2022;14(4):986. Published 2022 Feb 16. DOI:10.3390/cancers14040986. PMID: 35205734
8. Euvrard S, Kanitakis J, Claudy A. Skin cancers after organ transplantation. *N Engl J Med*. 2003;348(17):1681-1691. DOI:10.1056/NEJMra022137. PMID: 12711744
9. Moosa MR. Kaposi's sarcoma in kidney transplant recipients: a 23-year experience. *QJM*. 2005;98(3):205-214. DOI:10.1093/qjmed/hci028. PMID: 15728402
10. Krown SE, Metroka C, Wernz JC. Kaposi's sarcoma in the acquired immune deficiency syndrome: a proposal for uniform evaluation, response, and staging criteria. AIDS Clinical Trials Group Oncology Committee. *J Clin Oncol*. 1989;7(9):1201-1207. DOI:10.1200/JCO.1989.7.9.1201. PMID: 2671281
11. Lebbe C, Garbe C, Stratigos AJ, et al. Diagnosis and treatment of Kaposi's sarcoma: European consensus-based interdisciplinary guideline (EDF/EADO/EORTC). *Eur J Cancer*. 2019;114:117-127. DOI:10.1016/j.ejca.2018.12.036. PMID: 31096150
12. Di Lorenzo G, Kreuter A, Di Trolio R, et al. Activity and safety of pegylated liposomal doxorubicin as first-line therapy in the treatment of non-visceral classic Kaposi's sarcoma: a multicenter study. *J Invest Dermatol*. 2008;128(6):1578-1580. DOI:10.1038/sj.jid.5701215. PMID: 18185536
13. Tulpule A, Groopman J, Saville MW, et al. Multicenter trial of low-dose paclitaxel in patients with advanced AIDS-related Kaposi sarcoma. *Cancer*. 2002;95(1):147-154. DOI:10.1002/cncr.10634. PMID: 12115328
14. Paksoy N, Khanmammadov N, Doğan İ, et al. Weekly paclitaxel treatment in the first-line therapy of classic Kaposi sarcoma: A real-life study. *Medicine (Baltimore)*. 2023;102(5):e32866. DOI:10.1097/MD.00000000000032866. PMID: 36749246
15. Brambilla L, Labianca R, Boneschi V, et al. Mediterranean Kaposi's sarcoma in the elderly. A randomized study of oral etoposide versus vinblastine. *Cancer*. 1994;74(10):2873-2878. DOI:10.1002/1097-0142(19941115)74:10<2873::aid-cncr2820741021>3.0.co;2-1. PMID: 7954250
16. Tur E, Brenner S. Classic Kaposi's sarcoma: low-dose interferon alfa treatment. *Dermatology*. 1998;197(1):37-42. DOI:10.1159/000017973. PMID: 9693183
17. Saller J, Walko CM, Millis SZ, Henderson-Jackson E, Makanji R, Brohl AS. Response to Checkpoint Inhibitor Therapy in

- Advanced Classic Kaposi Sarcoma: A Case Report and Immunogenomic Study. *J Natl Compr Canc Netw*. 2018;16(7):797-800. DOI:10.6004/jnccn.2018.7018. PMID: 30006421
18. Galanina N, Goodman AM, Cohen PR, Frampton GM, Kurzrock R. Successful Treatment of HIV-Associated Kaposi Sarcoma with Immune Checkpoint Blockade. *Cancer Immunol Res*. 2018;6(10):1129-1135. DOI:10.1158/2326-6066.CIR-18-0121. PMID: 30194084
 19. Nguyen HQ, Magaret AS, Kitahata MM, Van Rompaey SE, Wald A, Casper C. Persistent Kaposi sarcoma in the era of highly active antiretroviral therapy: characterizing the predictors of clinical response. *AIDS*. 2008;22(8):937-945. DOI:10.1097/QAD.0b013e3282ff6275. PMID: 1853853
 20. Saiag P, Brunet H, Fortier-Beaulieu M. Les traitements locaux dans la maladie de Kaposi associée au SIDA [Local treatments of AIDS-related Kaposi disease]. *Ann Dermatol Venereol*. 1995;122(8):551-557. PMID: 8572501
 21. Caccialanza M, Marca S, Piccinno R, Eulisse G. Radiotherapy of classic and human immunodeficiency virus-related Kaposi's sarcoma: results in 1482 lesions. *J Eur Acad Dermatol Venereol*. 2008;22(3):297-302. DOI:10.1111/j.1468-3083.2007.02405.x. PMID: 18269597
 22. Boudreaux AA, Smith LL, Cosby CD, Bason MM, Tappero JW, Berger TG. Intralesional vinblastine for cutaneous Kaposi's sarcoma associated with acquired immunodeficiency syndrome. A clinical trial to evaluate efficacy and discomfort associated with infection. *J Am Acad Dermatol*. 1993;28(1):61-65. DOI:10.1016/0190-9622(93)70010-q. PMID: 8381146
 23. Brambilla L, Bellinva M, Turlaki A, Scoppio B, Gaiani F, Boneschi V. Intralesional vincristine as first-line therapy for nodular lesions in classic Kaposi sarcoma: a prospective study in 151 patients [published correction appears in *Br J Dermatol*. 2010 Apr;162(4):907-8. Dosage error in article text]. *Br J Dermatol*. 2010;162(4):854-859. DOI:10.1111/j.1365-2133.2009.09601.x. PMID: 19995366
 24. Di Monta G, Caracò C, Benedetto L, et al. Electrochemotherapy as "new standard of care" treatment for cutaneous Kaposi's sarcoma. *Eur J Surg Oncol*. 2014;40(1):61-66. DOI:10.1016/j.ejso.2013.09.002. PMID: 24075826
 25. Ferioli M, Galuppi A, Buwenge M, et al. Electrochemotherapy in Kaposi sarcoma: A systematic review. *Mol Clin Oncol*. 2021;14(4):64. DOI:10.3892/mco.2021.2226. PMID: 33690455
 26. Célestin Schartz NE, Chevret S, Paz C, et al. Imiquimod 5% cream for treatment of HIV-negative Kaposi's sarcoma skin lesions: A phase I to II, open-label trial in 17 patients. *J Am Acad Dermatol*. 2008;58(4):585-591. DOI:10.1016/j.jaad.2007.11.005. PMID: 18068265
 27. Bodsworth NJ, Bloch M, Bower M, Donnell D, Yocum R; International Panretin Gel KS Study Group. Phase III vehicle-controlled, multi-centered study of topical alitretinoin gel 0.1% in cutaneous AIDS-related Kaposi's sarcoma. *Am J Clin Dermatol*. 2001;2(2):77-87. DOI:10.2165/00128071-200102020-00004. PMID: 11705307
 28. Tappero JW, Berger TG, Kaplan LD, Volberding PA, Kahn JO. Cryotherapy for cutaneous Kaposi's sarcoma (KS) associated with acquired immune deficiency syndrome (AIDS): a phase II trial. *J Acquir Immune Defic Syndr (1988)*. 1991;4(9):839-846. PMID: 1895204
 29. Chun YS, Chang SN, Park WH. A case of classical Kaposi's sarcoma of the penis showing a good response to high-energy pulsed carbon dioxide laser therapy. *J Dermatol*. 1999;26(4):240-243. DOI:10.1111/j.1346-8138.1999.tb03464.x. PMID: 10343470
 30. Brightman LA, Brauer JA, Anolik R, et al. Ablative and fractional ablative lasers. *Dermatol Clin*. 2009;27(4):479-vii. DOI:10.1016/j.det.2009.08.009. PMID: 19850197
 31. Omi T, Numano K. The Role of the CO2 Laser and Fractional CO2 Laser in Dermatology. *Laser Ther*. 2014;23(1):49-60. DOI:10.5978/islm.14-RE-01. PMID: 24771971
 32. Bin Dakhil A, Shadid A, Altalhab S. Post-inflammatory hyperpigmentation after carbon dioxide laser: review of prevention and risk factors. *Dermatol Reports*. 2023;15(4):9703. Published 2023 May 19. DOI:10.4081/dr.2023.9703. PMID: 38205425
 33. Baggish MS, Poesz BJ, Joret D, Williamson P, Refai A. Presence of human immunodeficiency virus DNA in laser smoke. *Lasers Surg Med*. 1991;11(3):197-203. DOI:10.1002/lsm.1900110302. PMID: 1907345
 34. Katz B. Efficacy of a new fractional CO2 laser in the treatment of photodamage and acne scarring. *Dermatol Ther*. 2010;23(4):403-406. DOI:10.1111/j.1529-8019.2010.01340.x. PMID: 20666827
 35. Wheeland RG, Bailin PL, Norris MJ. Argon laser photocoagulative therapy of Kaposi's sarcoma: a clinical and histologic evaluation. *J Dermatol Surg Oncol*. 1985;11(12):1180-1185. DOI:10.1111/j.1524-4725.1985.tb03092.x. PMID: 4067053
 36. Wheeland RG. Clinical uses of lasers in dermatology. *Lasers Surg Med*. 1995;16(1):2-23. DOI:10.1002/lsm.1900160103. PMID: 7715398
 37. Nelson JS. Selective photothermolysis and removal of cutaneous vasculopathies and tattoos by pulsed laser. *Plast Reconstr Surg*. 1991;88(4):723-731. DOI:10.1097/00006534-199110000-00028. PMID: 1896548
 38. Rossi A, Iurassich S, Bozzi M, Villano PA, Voza A. Il laser ad argon in dermatologia. Indicazioni suggerite da quattro anni di esperienza [Argon laser in dermatology: indications suggested by a 4-year experience]. *G Ital Dermatol Venereol*. 1990;125(10):439-443. PMID: 2081623
 39. Tappero JW, Grekin RC, Zanelli GA, Berger TG. Pulsed-dye laser therapy for cutaneous Kaposi's sarcoma associated with acquired immunodeficiency syndrome [published correction appears in *J Am Acad Dermatol* 1993 Feb;28(2 Pt 1):188]. *J Am Acad Dermatol*. 1992;27(4):526-530. DOI:10.1016/0190-9622(92)70217-4. PMID: 1401303
 40. Marchell N, Alster TS. Successful treatment of cutaneous Kaposi's sarcoma by the 585-nm pulsed dye laser. *Dermatol Surg*. 1997;23(10):973-975. DOI:10.1111/j.1524-4725.1997.tb00762.x. PMID: 9357509
 41. Bassi A, Bonan P, Cannarozzo G, et al. New successful treatment of genital AIDS-related Kaposi's sarcoma resistant to systemic therapy with 595-nm pulsed dye laser. *G Ital Dermatol Venereol*. 2011;146(6):507-508. PMID: 22095186
 42. Junejo MH, Hibler BP, Lavin L, Menzer C, Aleisa A, Rossi AM. Nonablative laser therapy for cutaneous Kaposi sarcoma: a single centre proof-of-concept study of six patients. *Clin Exp Dermatol*. 2025;50(2):455-457. DOI:10.1093/ced/llae390. PMID: 39318102
 43. Spicer MS, Goldberg DJ. Lasers in dermatology. *J Am Acad Dermatol*. 1996;34(1):1-28. DOI:10.1016/s0190-9622(96)90827-0. PMID: 8543678
 44. Landthaler M, Hohenleutner U. Laser therapy of vascular lesions. *Photodermatol Photoimmunol Photomed*. 2006;22(6):324-332. DOI:10.1111/j.1600-0781.2006.00254.x. PMID: 17100741

45. Özdemir M, Balevi A. Successful Treatment of Classic Kaposi Sarcoma With Long-Pulse Neodymium-Doped Yttrium Aluminum Garnet Laser: A Preliminary Study. *Dermatol Surg.* 2017;43(3):366-370. DOI:10.1097/DSS.0000000000000973. PMID: 28157730
46. Nasca MR, Luppino I, Spurio Catena A, Micali G. Nodular Classic Kaposi's Sarcoma Treated With Neodymium-Doped Yttrium Aluminum Garnet Laser Delivered Through a Tilted Angle: Outcome and 12-Month Follow Up. *Lasers Surg Med.* 2020;52(10):979-983. DOI:10.1002/lsm.23242. PMID: 32275076
47. Silvestri M, Latini A, Lesnoni La Parola I, Messina C, Nisticò SP, Cameli N. Effectiveness and Safety of Treatment with Neodymium:YAG Laser 1064 nm in Patients with Classic and Epidemic Kaposi Sarcoma. *Bioengineering (Basel).* 2022;9(3):106. Published 2022 Mar 5. DOI:10.3390/bioengineering9030106. PMID: 35324795
48. Barillari G, Sgadari C, Palladino C, et al. Inflammatory cytokines synergize with the HIV-1 Tat protein to promote angiogenesis and Kaposi's sarcoma via induction of basic fibroblast growth factor and the alpha v beta 3 integrin. *J Immunol.* 1999;163(4):1929-1935. PMID: 10438928
49. van der Mescht MA, Steel HC, Anderson R, Rossouw TM. Vascular endothelial growth factor A: friend or foe in the pathogenesis of HIV and SARS-CoV-2 infections?. *Front Cell Infect Microbiol.* 2025;14:1458195. Published 2025 Feb 11. DOI:10.3389/fcimb.2024.1458195. PMID: 40008234
50. Bostanci S, Aygun Alizada M, Farabi B, Akay BN. Efficacy of Long-Pulsed Nd:YAG Laser for Classic Kaposi's Sarcoma: A Dermoscopic Study. *Dermatol Pract Concept.* 2024;14(2):e2024150. Published 2024 Apr 1. DOI:10.5826/dpc.1402a150. PMID: 38552137
51. Srinivas CR, Kumaresan M. Lasers for vascular lesions: standard guidelines of care. *Indian J Dermatol Venereol Leprol.* 2011;77(3):349-368. DOI:10.4103/0378-6323.79728. PMID: 21508585
52. Martinez V, Caumes E, Gambotti L, et al. Remission from Kaposi's sarcoma on HAART is associated with suppression of HIV replication and is independent of protease inhibitor therapy. *Br J Cancer.* 2006;94(7):1000-1006. DOI:10.1038/sj.bjc.6603056. PMID: 16570046
53. Palmieri B, Di Cerbo A, Rottigni V, Fistetto G, Iannitti T. The feasibility of a fiber optic laser approach to relieving lymphedematous syndrome: a case report. *Onco Targets Ther.* 2013;6:85-88. DOI:10.2147/OTT.S39420. PMID: 23425774
54. Mesri EA. Inflammatory reactivation and angiogenicity of Kaposi's sarcoma-associated herpesvirus/HHV8: a missing link in the pathogenesis of acquired immunodeficiency syndrome-associated Kaposi's sarcoma. *Blood.* 1999;93(12):4031-4033. PMID: 10361099