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Review Article

Systemic medication as a safety variable in dermatologic laser and energy-based device procedures: a practical risk-stratification framework

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ABSTRACT

Dermatologic laser and energy-based device procedures are increasingly performed in patients receiving systemic medication. Traditional pre-procedure screening often treats selected drugs as binary contraindications, although the relevance of medication exposure depends on device type, wavelength, pulse duration, cooling, treatment endpoint, tissue depth, epidermal barrier disruption, treatment area, skin phenotype, and follow-up capacity. This practical review proposes a structured framework for incorporating systemic medication into laser and energy-based device safety assessment. PubMed-based evidence mapping was organized by medication class and procedure category. Evidence was graded pragmatically as direct laser or energy-based device evidence, indirect procedural evidence, pharmacologic or biologic plausibility, or absent evidence. The strongest medication-specific laser evidence concerns isotretinoin, where historical blanket delays have been reconsidered for many low-injury and fractional procedures. Antithrombotic agents and Bruton tyrosine kinase inhibitors primarily modify bleeding and purpura risk, with most evidence extrapolated from dermatologic surgery. Direct laser-specific evidence remains sparse for biologics, Janus kinase inhibitors, conventional immunosuppressants, systemic corticosteroids, targeted oncologic therapies, and immune checkpoint inhibitors. Photosensitizing and pigment-altering drugs are most relevant for intense pulsed light, photodynamic therapy, pigment lasers, and darker phototypes. Systemic medication should be considered a procedure-dependent safety variable rather than an automatic contraindication. The proposed framework integrates medication class, procedure injury profile, patient phenotype, and evidence grade to support individualized risk assessment and shared decision-making.

Keywords: Laser therapy, Energy-based devices, Systemic medication, Isotretinoin, Anticoagulants, Photosensitivity, Dermatologic surgery

INTRODUCTION

Dermatologic laser and energy-based device procedures are now routine in the management of vascular lesions, pigmentary disorders, unwanted hair, scars, acne sequelae, photodamage, skin laxity, benign lesions, and selected inflammatory or premalignant conditions. General laser complication reviews emphasize that adverse events depend on patient selection, operator technique, wavelength, pulse duration, fluence, cooling, endpoint selection, and aftercare rather than on device category alone.[1,2] These variables become particularly important when patients receive systemic medications that may alter bleeding, inflammation, pigmentation, infection susceptibility, skin fragility, or wound repair.

Medication screening is often performed before laser procedures, but the clinical logic is frequently inconsistent. Some drugs are still listed as contraindications without separating low-injury

procedures from full-field resurfacing or deep dermal injury. Isotretinoin illustrates this problem. Historical blanket delays after isotretinoin have been substantially reconsidered for many nonablative, fractional, and low-injury procedures, while caution remains appropriate for full-field ablative resurfacing and mechanical dermabrasion.[4-6]

Other medication groups are less well studied. Antithrombotic agents and Bruton tyrosine kinase inhibitors primarily modify bruising and bleeding risk, but discontinuation can expose patients to thromboembolic harm.[7-9] Biologics, Janus kinase inhibitors, conventional immunosuppressants, systemic corticosteroids, photosensitizers, pigment-altering drugs, targeted oncologic agents, and metabolic medications each raise different questions. For many of these classes, direct laser-specific evidence is limited or absent. The purpose of this review is to propose a practical, non-dogmatic framework for systemic medication assessment before dermatologic laser and energy-based device procedures. The framework is not a formal guideline or consensus statement. It is intended to support structured clinical reasoning, conservative parameter selection when appropriate, and shared decision-making.

LITERATURE RESEARCH

This manuscript was developed as a narrative review with structured evidence mapping. PubMed-based searches were performed and updated to June 2026. Search terms included combinations of medication classes, for example isotretinoin, acitretin, anticoagulants, antiplatelet agents, Bruton tyrosine kinase inhibitors, biologics, JAK inhibitors, corticosteroids, photosensitizing drugs, EGFR inhibitors, BRAF/MEK inhibitors, immune checkpoint inhibitors, and GLP-1 receptor agonists, with procedure and device terms, for example fractional laser, ablative laser, pulsed dye laser, KTP, Nd:YAG, intense pulsed light, photodynamic therapy, picosecond, laser-induced optical breakdown, radiofrequency microneedling, microneedling, and microfocused ultrasound. Manual reference screening of retrieved articles was performed to identify key consensus documents, reviews, case series, and clinically relevant mechanistic papers.

Procedure blocks included hair removal lasers and intense pulsed light, vascular lasers, pigment lasers, Q-switched and picosecond devices, tattoo removal, nonablative fractional lasers, ablative fractional lasers, full-field ablative resurfacing, radiofrequency microneedling, microneedling without radiofrequency, photodynamic therapy, and microfocused or high-intensity focused ultrasound. Articles were prioritized when they provided direct laser or energy-based device evidence, perioperative or dermatologic surgery evidence relevant by cautious extrapolation, or pharmacologic mechanisms with plausible relevance to pigmentation, coagulation, inflammation, infection, or wound repair. This was not designed as a systematic review, and no pooled quantitative analysis was attempted.

Evidence was classified pragmatically as direct laser or energy-based device evidence, indirect procedural evidence, pharmacologic or biologic plausibility, or absent/not established evidence. Because the evidence base is heterogeneous and direct laser-specific outcome data are sparse for many medication classes, no quantitative meta-analysis was attempted. Recommendations are therefore framed as risk-stratification principles rather than as fixed treatment rules.

CORE CONCEPT: PROCEDURE INJURY PROFILE

A binary model of medication safety is too simplistic for procedural dermatology. The clinical relevance of a drug depends on the procedure injury profile, the patient phenotype, and the strength of supporting evidence. We propose four practical axes: epidermal barrier disruption, thermal coagulation burden, mechanical or acoustic injury, and re-epithelialization demand.

Hair removal and small-area vascular laser treatments generally have low epidermal disruption. Nonablative fractional lasers create controlled thermal zones with moderate repair demand. Radiofrequency microneedling combines needle-mediated penetration with dermal thermal injury. Ablative fractional lasers create microchannels and increase barrier permeability. Full-field ablative resurfacing produces the highest re-epithelialization demand. Picosecond fractional systems can produce laser-induced optical breakdown and cavitation, which differs from classic photothermal injury. Microfocused ultrasound produces deeper thermal coagulation zones with minimal epidermal breach.

Wavelength, pulse duration, fluence, spot size, cooling, density, passes, treatment area, and endpoint selection are active safety variables. Cooling is not only a comfort measure. Contact cooling, cryogen spray, and cold air modify epidermal thermal exposure and may change the margin between epidermal injury and target heating.[3] In medication-exposed patients, cooling and endpoint selection are especially relevant when skin is photosensitive, atrophic, heavily pigmented, or prone to post-inflammatory hyperpigmentation.

Reducing fractional density decreases the treated surface fraction and lowers cumulative bulk thermal accumulation, but it may not alter the microscopic thermal injury around each beam when fluence, pulse duration, and spot characteristics remain unchanged. This distinction is important in patients with corticosteroid-atrophic or otherwise fragile skin, where both focal microthermal injury and aggregate heat load may affect healing. Similarly, changing a vascular laser from a purpuric to a subpurpuric endpoint changes the

relationship between vessel heating and mechanical rupture. This distinction is clinically relevant in patients receiving antithrombotic therapy, Bruton tyrosine kinase inhibitors, or systemic corticosteroids.

PATIENT PHENOTYPE LAYER

Medication risk should be interpreted in the context of patient phenotype. Important modifiers include Fitzpatrick skin type, tanning status, history of post-inflammatory hyperpigmentation, melasma tendency, keloid or hypertrophic scar history, active inflammatory dermatoses, geriatric skin fragility, senile purpura, chronic actinic damage, diabetes, vascular insufficiency, neuropathy, prior ulceration, infection history, herpes simplex virus history, immunosuppression burden, and polypharmacy.

Fitzpatrick skin types IV to VI deserve explicit consideration because pigmentary adverse events may dominate the safety profile. Drug-induced inflammation, photosensitivity, or pigment deposition may be clinically less relevant in a low-risk phototype undergoing a small nonablative procedure but more relevant in a darker phototype undergoing intense pulsed light, melasma treatment, pigment laser treatment, or high-density resurfacing. In these patients, medication-associated phototoxicity or excess procedural inflammation may translate into prolonged post-inflammatory hyperpigmentation rather than only transient erythema. Conservative endpoints, longer intervals between sessions, aggressive epidermal protection with appropriate cooling, strict ultraviolet avoidance, and early topical anti-inflammatory aftercare when clinically appropriate should therefore be considered part of the safety framework rather than optional cosmetic refinements.

MEDICATION CLASSES

Systemic retinoids

Systemic retinoids remain the most debated medication class in procedural dermatology. Modern literature supports reassessment of historical blanket delays after isotretinoin for many low-injury and fractional procedures.[4-6]

However, this does not justify universal clearance for all procedures. Full-field ablative resurfacing, mechanical dermabrasion, and other procedures with high re-epithelialization demand remain higher-caution categories.

Acitretin and alitretinoin should not automatically inherit all isotretinoin-specific conclusions because direct laser-specific evidence is not established; recommendations for these agents should therefore be framed as extrapolated and procedure-dependent.

Antithrombotic agents and Bruton tyrosine kinase inhibitors

Anticoagulants and antiplatelet agents primarily modify bleeding, bruising, purpura, hematoma, and post-procedure oozing rather than classic wound-healing biology. The strongest evidence comes from dermatologic surgery, where interruption of therapy may expose patients to thromboembolic harm.[7,8] Laser-specific extrapolation should be explicit. Hair removal and low-injury nonablative procedures differ from microneedling, RF microneedling, ablative resurfacing, or purpura-generating vascular laser treatments.

Bruton tyrosine kinase inhibitors such as ibrutinib and acalabrutinib are increasingly relevant. A dermatologic surgery case-control study found increased bleeding events in patients receiving ibrutinib, particularly in older men, patients with lower platelet counts, and those on multiple anticoagulants.[9] This does not establish laser-specific complication rates, but it supports treating BTK inhibitors as bleeding-risk modifiers.

For vascular lasers such as 595 nm pulsed dye laser or 532 nm KTP systems, pulse duration and endpoint are central. A subpurpuric endpoint generally relies on controlled thermal coagulation of the vessel, often using longer millisecond pulse durations, for example in the approximate 10 to 20 ms range when clinically suitable, whereas very short pulses, for example sub-1.5 ms purpuric approaches, are more likely to produce mechanical vascular rupture and visible purpura. In patients receiving antithrombotic agents or Bruton tyrosine kinase inhibitors, this distinction is clinically relevant because purpuric endpoints may produce more extensive or prolonged ecchymosis. Medication interruption should not be recommended solely for cosmetic endpoint optimization without considering thrombotic risk and coordination with the prescribing clinician.

Biologics, Janus kinase inhibitors, and conventional immunosuppressants

Perioperative psoriasis guidance suggests that several biologic and conventional immunomodulatory agents can often be continued for low-risk procedures, while intermediate- and high-risk surgery require individualized assessment.[10,11] These recommendations derive from surgical and perioperative evidence, not laser-specific trials. Therefore, translation to energy-based procedures should be based on barrier disruption, wound area, treatment depth, infection history, and concomitant immunosuppression.

Janus kinase inhibitors should be handled separately from biologics. Direct laser-specific evidence is sparse, and recommendations should be framed as provisional. While systemic JAK inhibitors are pharmacologically linked to an elevated risk of herpes zoster reactivation, the clinical

translation to energy-based device safety remains speculative and may vary by molecule, dose, selectivity profile, indication, comorbidity, and concomitant immunosuppression.[12] In particular, highly selective JAK1 inhibitors, broader JAK1/2 inhibitors, and less selective or pan-JAK inhibition should not be assumed to carry identical procedural risk. For high-injury procedures where antiviral prophylaxis is already standard or strongly favored, such as full-field ablative resurfacing in herpes-risk settings, JAK inhibitor exposure reinforces the need for strict compliance rather than mandating a specific laser-parameter change. For moderate-injury procedures, antiviral prophylaxis should be considered individually when additional risk factors are present.

Methotrexate, cyclosporine, azathioprine, and mycophenolate should be considered indirect risk modifiers rather than universal contraindications. The relevance increases with high-wound-burden procedures, large treatment areas, active infection, leukopenia, combined corticosteroid exposure, transplant status, extensive actinic damage, or poor follow-up capacity.

Systemic corticosteroids

Systemic corticosteroids may contribute to skin atrophy, purpura, dermal thinning, delayed healing, infection risk, and impaired collagen remodeling. A perioperative review concluded that acute high-dose systemic corticosteroid exposure may have limited clinical effect on wound healing, whereas chronic systemic steroid use may impair wound healing in susceptible individuals.[13] For laser and energy-based devices, the practical relevance depends on dose, duration, cumulative exposure, age, visible skin phenotype, diabetes, nutritional status, and concomitant immunosuppression. Systemic corticosteroids should therefore be considered dose-, duration-, and phenotype-dependent risk modifiers rather than absolute contraindications.

Diabetes, metabolic risk, and GLP-1 receptor agonists

Diabetes is best considered a systemic wound-healing context rather than a laser-specific contraindication. Diabetic wounds are characterized by impaired angiogenesis, neuropathy, altered inflammation, infection susceptibility, and barrier dysfunction.[14,15] In cutaneous surgery literature, diabetes alone may not consistently be the strongest independent surgical-site infection risk factor, while systemic immunosuppression often demonstrates a clearer clinical association.[14,16] Risk becomes more clinically relevant with poor glycemic control, peripheral vascular disease, neuropathy, prior ulceration, lower-extremity sites, large treatment areas, active infection, or high-injury procedures.

GLP-1 receptor agonists such as semaglutide and tirzepatide should not be framed as routine laser parameter modifiers. Their relevance is mainly procedural and aesthetic: delayed gastric emptying may matter when sedation, deep analgesia, or anesthesia is planned, while rapid weight loss can alter facial volume, subcutaneous fat support, and skin laxity assessment before rejuvenation or tightening procedures.[17,18] This is particularly relevant before tissue-tightening procedures such as MFU-V, HIFU, volumetric radiofrequency, or deep RF microneedling, where apparent laxity may reflect recent soft-tissue deflation rather than intrinsic dermal laxity alone. At present, any wound-healing benefit or harm should not be presented as laser-specific evidence.

Photosensitizing and pigment-altering drugs

Photosensitizing drugs should not be treated as automatic contraindications to all lasers. A review of lasers and photosensitive medication found very limited direct evidence of laser-related harm in patients taking photosensitizing medication, but absence of published harm is not proof of safety.[19] Drug-induced photosensitivity reviews identify amiodarone, doxycycline, hydrochlorothiazide, naproxen, piroxicam, tetracycline, voriconazole, and vemurafenib among consistently implicated drugs.[20,21] The practical question is whether the drug action spectrum overlaps with the device spectrum, especially for intense pulsed light, photodynamic therapy, ultraviolet or blue-light procedures, and pigment-targeting devices.

Amiodarone deserves explicit mention because it may cause UVA photosensitivity and blue-gray pigmentation, often in older patients with cardiovascular comorbidity.[20,21] Tetracyclines are relevant because doxycycline has phototoxic potential and minocycline may produce persistent blue-gray or muddy-brown pigmentation.[22-25] Hydroxychloroquine and chloroquine may also cause drug-induced pigmentation and have photophysical properties relevant to light exposure.[26-28]

For drug-induced pigment deposition, wavelength selection should account for the intended chromophore, pigment depth, epidermal melanin absorption, and phototype. Persistent minocycline pigmentation has been treated with pigment lasers in case-based literature.[25] For deeper dermal pigment deposits, ultrashort-pulsed Q-switched or picosecond systems in the near-infrared range, particularly 755 nm alexandrite or 1064 nm Nd:YAG platforms, are conceptually preferable because they reduce epidermal melanin competition compared with shorter visible wavelengths. Shorter wavelengths such as 532 nm should be used cautiously for deeper pigmentary targets, especially in Fitzpatrick skin types IV to VI, because epidermal absorption and post-inflammatory hyperpigmentation risk may become limiting. These statements should be read as laser-tissue interaction principles rather than fixed treatment recommendations.

Targeted oncologic therapies and immune checkpoint inhibitors

BRAF inhibitors, particularly vemurafenib, may cause clinically important photosensitivity, while BRAF and MEK inhibitor combinations have broader cutaneous adverse-event profiles.[29,30] EGFR inhibitors may produce xerosis, acneiform eruption, fissuring, and barrier intolerance.[31] Immune checkpoint inhibitors may cause lichenoid, psoriasiform, vitiligo-like, eczematous, and bullous immune-related cutaneous adverse events.[32] Direct laser-specific evidence for EGFR inhibitors and immune checkpoint inhibitors is absent/not established. These therapies should therefore be listed as data-limited but clinically relevant categories, especially when active rash, photosensitivity, Koebner-prone dermatoses/elective high-injury cosmetic procedures are being considered.

Procedure-specific considerations

Hair removal lasers and intense pulsed light usually represent low-injury procedures. Medication relevance is generally lower than for resurfacing, but photosensitizers, tanning, retinoids, fragile skin, and pigmentary risk should still be assessed. Intense pulsed light should be separated from narrowband lasers because its broad emission spectrum may increase relevance of photosensitizing drugs.

Vascular lasers are most relevant to antithrombotic therapy, Bruton tyrosine kinase inhibitors, systemic corticosteroids, senile purpura, and fragile skin. The main concern is often excessive purpura, bruising, hematoma, or distorted vascular endpoint rather than delayed epithelial closure. For 532 nm KTP and 595 nm pulsed dye laser procedures, pulse duration, vessel diameter, fluence, cooling, and desired endpoint determine whether treatment remains subpurpuric or deliberately produces vessel rupture.

Pigment lasers and picosecond devices should be separated from ablative resurfacing, but also from each other when optical breakdown or cavitation is intended. Picosecond fractional systems generate distinct mechanical/acoustic injury profile and can induce optical breakdown in epidermal/dermal compartments.[33] This matters when evaluating pigmentary risk, anticoagulant exposure, and inflammatory/Koebner-prone skin.

Tattoo removal requires separate classification because tattoo pigments may produce allergic, granulomatous, phototoxic, toxicologic, or rarely systemic reactions after pigment fragmentation.[34,35] Photodynamic therapy is also distinct because it deliberately combines a photosensitizer, light, and oxygen to generate reactive oxygen species.[36,37] Porphyrias and severe photosensitivity disorders require specific consideration before PDT or phototoxic light exposure.[38]

Nonablative fractional lasers and microneedling occupy moderate injury categories. Radiofrequency microneedling adds dermal thermal injury to mechanical penetration and should be considered separately from microneedling without radiofrequency.[39-41] Ablative fractional lasers and full-field ablative resurfacing are high-injury procedures because they disrupt the epidermal barrier, increase infection relevance, and require organized re-epithelialization. Antiviral prophylaxis is well supported for ablative resurfacing in herpes-risk settings, and unusual bacterial, fungal, viral, or atypical infections must be considered when healing deviates from the expected course.[42-44]

Microfocused ultrasound and high-intensity focused ultrasound occupy a distinct category: low epidermal disruption but deeper focal thermal coagulation. Their medication-specific evidence is limited, but patient selection, coupling, visualization, pain, bruising, dysesthesia, and rare thermal or nerve-related complications should be considered.[45-47]

Table 1: Medication classes and laser-relevant risk domains.

Medication or systemic context	Main risk domains	Highest concern procedures	Evidence type/key references
Isotretinoin	Historical scarring concern, mucocutaneous fragility	Full-field resurfacing, dermabrasion	Direct and consensus-supported for many lower-injury procedures[4-6]
Acitretin/ alitretinoin	Retinoid mucocutaneous effects, theoretical healing effects	Ablative resurfacing	Direct laser-specific evidence not established; extrapolated and procedure-dependent
Anticoagulants/ antiplatelets	Purpura, bruising, hematoma, bleeding	Vascular lasers, microneedling, RF microneedling, ablative procedures	Indirect dermatologic surgery evidence[7,8]
BTK inhibitors	Bleeding, bruising, platelet dysfunction	Microneedling, RF microneedling, ablative procedures	Indirect dermatologic surgery evidence[7,9]
Biologics	Infection, altered inflammatory response	Ablative fractional, full-field resurfacing, deep RF microneedling	Indirect perioperative guidance[10,11]

Continued.

Medication or systemic context	Main risk domains	Highest concern procedures	Evidence type/key references
JAK inhibitors	Infection, herpesvirus risk, immune modulation; risk may vary by dose, indication, and JAK selectivity profile	High-injury procedures	Sparse laser-specific evidence; herpes zoster risk evidence; do not assume equivalent risk across selective JAK1, JAK1/2, and broader JAK inhibition[12]
Conventional immunosuppressants	Infection, repair, actinic damage context	Ablative and large-area procedures	Indirect perioperative/procedural extrapolation[10,11]
Systemic corticosteroids	Atrophy, purpura, delayed healing, infection	Full-field resurfacing, aggressive fractional ablative, deep RF microneedling	Indirect wound-healing evidence[13]
Diabetes / metabolic risk	Impaired healing, neuropathy, vascular insufficiency	Lower legs, ablative procedures, deep RF microneedling, volumetric RF, HIFU/MFU-V	Indirect evidence; in clinically relevant polyneuropathy, visual endpoint monitoring and conservative stepwise energy escalation are mandatory[14-16]
GLP-1 receptor agonists	Sedation/anesthesia planning, rapid facial fat loss, skin laxity assessment	Sedation or volume/laxity assessment, especially MFU-V/HIFU, volumetric RF, deep RF microneedling	Peri-anesthetic and aesthetic-planning relevance; not a routine laser-parameter modifier[17,18]
Photosensitizing drugs	Phototoxicity, erythema, PIH, dyspigmentation	IPL, PDT, UV/blue light, pigment procedures	Direct/indirect photosensitivity evidence[19-21]
Amiodarone	UVA photosensitivity, blue-gray pigmentation	IPL, pigment lasers, phototherapy, PDT	Photosensitivity evidence[20,21]
Minocycline/ tetracyclines	Drug pigmentation, phototoxicity	Pigment lasers, IPL	Phototoxicity and drug-pigmentation evidence[22-25]
Hydroxychloroquine/ chloroquine	Pigmentation, photophysical effects	Pigment lasers, IPL	Pigmentation and photophysical evidence[26-28]
Targeted oncologic therapies	Photosensitivity, rash, barrier effects	IPL, PDT, pigment/light-based procedures	Direct cutaneous toxicity evidence, indirect laser relevance
Immune checkpoint inhibitors	Immune-related dermatoses, Koebnerization	High-injury procedures during active dermatitis	Theoretical / sparse

Table 2: Procedure injury profile by device category.

Procedure category	Epidermal disruption	Thermal burden	Mechanical/ acoustic injury	Overall concern
Hair removal laser/IPL	Low	Low to moderate follicular	Minimal	Low
Vascular laser	Low	Low to moderate vascular	Minimal	Low to moderate
IPL	Low	Variable broad-spectrum	Minimal	Low to moderate
Pigment laser	Low	Variable	Moderate photomechanical	Low to moderate
Picosecond/LIOB	Low to moderate	Low to moderate	Moderate to high focal acoustic	Moderate
Tattoo removal	Low to moderate	Variable	Moderate to high	Moderate
Nonablative fractional laser	Moderate	Moderate	Minimal	Moderate
Microneedling	Puncture injury	Minimal	Mechanical	Moderate
RF microneedling	Puncture injury	Moderate dermal	Mechanical	Moderate
Volumetric RF	Minimal	Moderate bulk heating	Minimal	Low to moderate

Continued.

Procedure category	Epidermal disruption	Thermal burden	Mechanical/ acoustic injury	Overall concern
HIFU/MFU-V	Minimal epidermal	Deep focal thermal	Minimal	Low to moderate, deep-structure specific
Ablative fractional laser	High focal	High focal	Minimal	High
Full-field ablative resurfacing	Very high	High	Minimal	Very high
PDT	Variable	Photochemical	Minimal	Drug/light-specific

Table 3: Practical medication and procedure adjustment matrix.

Medication category	Low-injury procedures	Moderate-injury procedures	High-injury procedures	Practical response
Isotretinoin	Usually not automatic exclusion	Conservative settings, staged treatment	High caution for full-field ablative/ dermabrasion	Avoid blanket prohibition and blanket clearance
Antithrombotics	Usually continue	Expect bruising/purpura	Coordinate if bleeding consequences significant	Do not stop without thrombotic-risk assessment
BTK inhibitors	Individualized	Higher bruising concern	Consider coordination	Treat as bleeding-risk modifier
Biologics	Usually not automatic exclusion	Assess infection history	Higher caution if large area/barrier disruption	Avoid active infection, ensure follow-up
JAK inhibitors	Caution if infection history	Individualized	High caution	Data-sparse category; risk may vary by JAK selectivity and indication; ensure strict antiviral compliance when prophylaxis is indicated
Systemic corticosteroids	Usually possible if low-dose/no atrophy	Caution with fragile skin	High caution	Dose, duration, phenotype, comorbidities matter
Photosensitizers	Device-dependent	Test spot/ conservative endpoint	Avoid aggressive light exposure if high risk	Match drug action spectrum with device
Diabetes	Usually possible if controlled	Caution lower legs/large areas	Optimize before elective high-wound procedures	Assess vascular status, neuropathy, infection. In clinically relevant polyneuropathy, visual endpoint monitoring and conservative stepwise energy escalation are mandatory
GLP-1 receptor agonists	No laser parameter effect	No laser parameter effect	Sedation/anesthesia relevance only	Do not overstate laser relevance; reassess facial volume loss and skin laxity before MFU-V/HIFU, volumetric RF/deep RF microneedling

Table 4: Evidence-language guide.

Evidence category	Permitted claim language	Avoid
Direct laser/EBD evidence	Evidence supports or suggests in specified procedure category	Safe in all patients
Indirect procedural evidence	May be extrapolated cautiously from dermatologic surgery or perioperative literature	Laser-specific proof
Biologic plausibility	Mechanistically plausible and should be considered	Established risk
Absent evidence	Not established; individualized caution warranted	No evidence of harm means safe

CLINICAL USE OF THE FRAMEWORK

The proposed framework is intended to guide a sequence of questions rather than produce automatic yes or no decisions. First, identify the medication-related risk domain: bleeding, photosensitivity, pigment alteration, immune modulation, infection, skin fragility, delayed healing, or anesthesia-related relevance. Second, define the procedure injury profile. Third, integrate patient phenotype, especially Fitzpatrick skin type, PIH history, geriatric skin, diabetes, infection history, active dermatoses, and treatment site. Fourth, assign an evidence grade: direct, indirect, biologically plausible, or absent.

For low-injury procedures, many systemic medications will not require interruption. For moderate-injury procedures, test spots, smaller treatment areas, conservative initial parameters, less aggressive endpoints, and earlier follow-up may be appropriate. For high-injury procedures, especially full-field ablative resurfacing, aggressive ablative fractional treatment, deep RF microneedling, lower-leg procedures, or extensive field treatment, coordination with the prescribing physician and careful shared decision-making may be necessary.

Consent should acknowledge uncertainty. A practical formulation is: Your medication may influence bruising, inflammation, pigmentation, infection risk, or wound healing depending on the procedure. The available evidence is stronger for some medications than for others. Treatment has been selected and adjusted according to procedure intensity, skin type, medical history, and medication profile. Medication changes should be discussed with the prescribing physician because stopping medication can sometimes be more dangerous than continuing it.

LIMITATIONS

The main limitation of this framework is evidence asymmetry. Direct laser-specific evidence is comparatively stronger for isotretinoin and selected photosensitizer questions than for biologics, Janus kinase inhibitors, conventional immunosuppressants, systemic corticosteroids, targeted oncologic therapies, immune checkpoint inhibitors, and GLP-1 receptor agonists. Therefore, many recommendations remain indirect or biologically plausible rather than evidence-proven laser safety rules.

The absence of published adverse events should not be interpreted as proof of safety. Cosmetic and procedural complications are likely affected by underreporting and publication bias. Routine infections, pigmentary events, hypertrophic scars, or prolonged erythema may not be published because they are considered insufficiently novel, medicolegally sensitive, or difficult to attribute to medication exposure.

Device categories are also heterogeneous. Wavelength, pulse duration, fluence, spot size, density, passes, cooling, treatment area, skin preparation, endpoint, and aftercare can change risk substantially.[1-3] The framework is therefore not a substitute for device-specific training or clinical judgment.

FUTURE RESEARCH

Future clinical studies and registries should routinely report systemic medication status, medication dose and duration, indication, concomitant immunosuppression, antithrombotic exposure, Fitzpatrick skin type, tanning status, diabetes and vascular status, infection and herpes simplex virus history, device type, wavelength, fluence, pulse duration, spot size, density, passes, cooling, endpoint, prophylaxis, wound care, and follow-up interval. Priority areas include outcomes in patients receiving direct oral anticoagulants, BTK inhibitors, biologics, JAK inhibitors, systemic corticosteroids, targeted oncologic therapies, immune checkpoint inhibitors, and photosensitizing medications.

CONCLUSION

Systemic medication is an important safety variable in dermatologic laser and energy-based device procedures, but it should rarely be treated as a simple binary contraindication. Clinical relevance depends on the interaction between medication class, procedure injury profile, patient phenotype, and evidence grade. A low-injury hair removal or vascular laser treatment differs fundamentally from full-field ablative resurfacing, deep RF microneedling, photodynamic therapy, or tattoo removal in a high-risk patient.

The proposed framework encourages clinicians to move beyond blanket prohibitions while avoiding unsupported reassurance. Medication status should prompt structured risk assessment, conservative parameter selection when appropriate, staged treatment, test spots, enhanced aftercare, and coordination with the prescribing physician when medication interruption is considered.

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