Immunosuppressive agents in solid organ transplantation: Cutaneous adverse effects and dermatologic considerations

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Solid organ transplant recipients are living longer due to advances in immunosuppressive therapy, but chronic immunosuppression carries a high burden of cutaneous adverse effects. This clinical review provides a practical, agent-specific overview of cutaneous toxicities associated with commonly used immunosuppressants in solid organ transplant recipients, including induction agents, calcineurin inhibitors, antimetabolites, mammalian target of rapamycin inhibitors, corticosteroids, and co-stimulation blockers. Cutaneous adverse effects range from early hypersensitivity reactions, alopecia, and aphthous ulcers to delayed complications such as photosensitivity, impaired wound healing, and skin cancers. Azathioprine and cyclosporine are associated with increased risk of cutaneous squamous cell carcinoma, while mammalian target of rapamycin inhibitors may offer protective benefits. Dermatologists play a central role in screening, prevention, and management of these toxicities. Risk stratification tools such as the skin and ultraviolet neoplasia transplant risk assessment calculator can guide timing of dermatologic referral and surveillance. Close collaboration with transplant teams is essential to adjust immunosuppressive regimens safely and minimize dermatologic morbidity while preserving graft function. This review aims to equip dermatologists with an updated framework for recognizing, managing, and preventing cutaneous adverse effects in this growing patient population. (J Am Acad Dermatol 2026;95:127-37.)

Key words: drug response; immunosuppression; solid-organ transplant; transplant.

INTRODUCTION

Advances in immunosuppressive therapy have substantially improved graft and patient survival in solid organ transplant recipients (SOTRs), leading to a growing population of individuals living years to decades post-transplant.[1] As survival improves, the cumulative burden of immunosuppression is increasingly evident, with dermatologic complications among the most frequent manifestations (Table I).[2]

This review aims to equip dermatologists with a practical understanding of commonly used immunosuppressive agents, their associated cutaneous toxicities, and dermatology-specific management considerations in the care of transplant recipients.

IMMUNOSUPPRESSIVE STRATEGIES IN SOTRs

Immunosuppressive therapy is essential for maintaining graft function in SOTRs. Regimens are generally divided into 2 phases: induction therapy, administered at the time of transplantation to prevent early rejection, and maintenance therapy, continued long term to support graft survival.[3]

Induction therapy is initiated intraoperatively or within the first 24 hours post-transplant and provides potent short-term immunosuppression during the period before maintenance drugs reach therapeutic efficacy.[4,5] Although induction drug administration typically lasts 4 to 14 days, the immunologic effects

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may persist for several weeks depending on the agent.[6] Maintenance therapy is initiated within the first 24 to 48 hours post-transplantation, often overlapping with induction therapy, and is continued indefinitely to prevent rejection.[4] This early initiation is critical, as maintenance agents such as calcineurin inhibitors (CNIs) and antimetabolites require time to reach therapeutic levels. Standard regimens include a CNI, an antimetabolite, and corticosteroids, tailored to patient-specific factors, organ type and rejection risk.[3] Emerging molecular HLA-matching tools and advances in organ preservation such as normothermic regional perfusion may optimize immunosuppression intensity.[7,8] Over time, immunosuppressive regimens may be adjusted to reduce long-term toxicities.

CAPSULE SUMMARY

- This article synthesizes known cutaneous adverse effects of immunosuppressive therapies used in solid organ transplant recipients, emphasizing their timing, presentation, and oncologic risk.
- It encourages dermatologists to proactively screen for and manage these toxicities, coordinate with transplant teams, and tailor surveillance based on agent-specific risks.

DRUG CLASSES AND DERMATOLOGIC CONSIDERATIONS

Induction agents

Induction agents such as antithymocyte globulin (ATG), alemtuzumab, and basiliximab are administered perioperatively to reduce the risk of acute rejection during the critical early post-transplant period by targeting key components of the immune response. While these agents are typically given for a limited duration, dermatologists should recognize that their immunologic effects may persist well beyond administration and give rise to distinct, agent-specific cutaneous adverse events that require early identification and management.

ATG is a polyclonal antibody that binds to multiple T-cell surface antigens, leading to profound T-cell depletion through complement-mediated lysis and antibody-dependent cellular cytotoxicity.[9] ATG is associated with serum sickness in approximately 7% to 27% of renal transplant recipients, and has been reported in liver, pancreas, and cardiac transplant patients.[10-24] This reaction generally occurs between 7 and 14 days after first exposure but can present within 12 to 36 hours in sensitized individuals receiving repeat dosing.[16,18] Dermatologists should maintain a high index of suspicion when evaluating new-onset symmetric urticarial or morbilliform eruptions involving the trunk and proximal extremities during this time frame, as cutaneous findings are often the earliest manifestation (Fig 1).[25] Lesions may evolve over several days and are frequently accompanied by pruritus, pain, fever, arthralgia, lymphadenopathy, or vasculitic features.[17,26] In practice, diagnosis is clinical and biopsy is rarely required. Management consists of prompt discontinuation of ATG and initiation of systemic corticosteroids, most commonly prednisone 0.5 to 1 mg/kg/d with a short taper once symptoms improve. Early recognition is critical, as refractory or severe cases may progress and require therapeutic plasma exchange.[27]

Alemtuzumab is a monoclonal antibody targeting CD52, resulting in broad lymphocyte depletion through cytolytic mechanisms.[28] While cutaneous adverse events are uncommon, immune reconstitution may trigger autoimmune skin conditions such as vitiligo, alopecia areata or chronic urticaria.[29,30] Most cases reported have been mild and self-limited, and management in clinical practice generally involves topical corticosteroids or antihistamines without the need for discontinuation of immunosuppression.

Basiliximab, an IL-2 receptor antagonist, inhibits T-cell activation without depleting lymphocytes and is generally from a dermatologic perspective. Nevertheless, clinicians should recognize that nonspecific pruritic rashes, cysts, herpes simplex, herpes zoster, hypertrichosis, and ulceration have been reported in 3% to 10% of transplant recipients.[31] Immediate hypersensitivity reactions, while rare, may present with urticaria shortly after administration and should prompt discussion with the transplant physician regarding drug discontinuation.[31,32]

Maintenance immunosuppressants

Calcineurin inhibitors. CNIs, including cyclosporine and tacrolimus, suppress T-cell activation by inhibiting calcineurin phosphatase, thereby blocking interleukin-2 transcription and downstream immune activation.[33] Because CNIs are foundational components of long-term maintenance immunosuppression, dermatologists should be aware of both early dermatologic effects and delayed oncologic complications when caring for transplant recipients.

Cyclosporine frequently causes hypertrichosis, which typically develops within the first few weeks of therapy and may improve with dose reduction.[34-39] Gingival hyperplasia is another early-onset effect and should prompt reinforcement of dental hygiene and referral to dental specialists when severe.[35,38,39] Sebaceous hyperplasia and acneiform eruptions may develop with prolonged therapy; these are generally

Abbreviations used:

ATG:	antithymocyte globulin
CF:	cystic fibrosis
CNI:	calcineurin inhibitors
KS:	Kaposi Sarcoma
MCC:	Merkel cell carcinoma
PTLD:	post-transplant lymphoproliferative disorder
SOTR:	solid organ transplant recipients
SUNTRAC:	skin and ultraviolet neoplasia transplant risk assessment calculator

managed conservatively and rarely necessitate changes in immunosuppression.[39,40] Bacterial folliculitis has been reported and typically responds to topical or systemic antibiotics.[34]

Chronic use increases cutaneous squamous cell carcinoma (cSCC) risk by risk by activating PI3K/AKT signaling, which impairs DNA repair, disrupts checkpoints, and activates pro-tumorigenic pathways such as TGF-β/TAK1/NFκB and VEGF-mediated angiogenesis.[41] Given this risk, we recommend strict photoprotection counseling and routine dermatologic surveillance, typically every 6 to 12 months, with closer follow-up for patients with prior skin cancer or extensive actinic damage.[42-45] In patients who develop multiple or aggressive cSCCs, discussion of alternative immunosuppressive strategies, such as transition to mTOR inhibitors, should occur in close collaboration with the transplant team.

Tacrolimus has also been associated with diffuse nonscarring alopecia a resembling telogen effluvium as well as changes in hair texture.[46,47] Management is typically supportive, and dermatologists may consider topical minoxidil or intralesional corticosteroids, recognizing that responses are variable.[47]

Antimetabolites. Mycophenolate mofetil (MMF)[48] Rash has been reported in up to 26% of clinical trial participants receiving MMF, and typically presents as a nonspecific maculopapular eruption within the first few months of therapy.[49-52] In clinical practice, these eruptions are usually mild and can be managed with topical corticosteroids and oral antihistamines without interruption of MMF.

Aphthous ulcers may occur with MMF use and can be painful or interfere with nutrition.[53-56] Dermatologists should recognize MMF-associated aphthae and inititate treatment with dexamethasone rinse (0.5 mg/5 mL, swish and spit twice daily) along with supportive measures (topical lidocaine for analgesia, oral hygiene, dietary modification, and compounded "magic mouthwash" (eg, diphenhydramine, lidocaine, and an antacid)).[57] In patients with refractory symptoms, dose reduction or modification of immunosuppression may be required in coordination with the transplant team.[58]

Diffuse nonscarring alopecia has been reported in patients on MMF.[59-61] When hair loss is clinically significant, dermatologists may consider topical minoxidil 5% and, in cases of patchy involvement, intralesional corticosteroids.

Azathioprine (AZA), a purine analog metabolized to 6-mercaptopurine, is associated with several dermatologic toxivities.[62] Clinicians should be alert to photosensitive dermatitis resembling chronic actinic damage, which reflects azathioprine-induced impairment of UVA-mediated DNA repair.[52,62] Strict photoprotection should be emphasized in all patients receiving AZA, and topical corticosteroids may be used for symptomatic relief. For patients with persistent photosensitivity or extensive photodamage, substitution with an alternative antimetabolite such as MMF should be considered.[62] AZA may also provoke a systemic hypersensitivity syndrome during the first 4 weeks of therapy, characterized by a maculopapular, vesicular, or pustular rash, fever, malaise, arthralgia, and gastrointestinal symptoms (Fig 2).[63,64] Because this reaction is dose independent and may be life-threatening, early recognition and immediate discontinuation of azathioprine are essential, and rechallenge is contraindicated.[65] Systemic corticosteroids are often required for management, particularly when associated with neutrophilic dermatoses such as Sweet syndrome.[65-69]

Long-term AZA use is associated with a markedly increased risk of cSCC, particularly in sun-exposed areas.[70,71] We recommend regular dermatologic surveillance at 6-12-month intervals, reinforced photoprotection counseling, and close monitoring for signs of field cancerization.[72]

mTOR inhibitors. Sirolimus and everolimus are mammalian target of rapamycin inhibitors (mTORi) that suppress T-cell proliferation and angiogenesis through inhibition of the mTOR pathway.[73-75]

Aphthous stomatitis is among the most common dermatologic adverse effects of mTOR inhibitors, occurring in approximately 10% to 20% of patients and typically presenting within the first month of therapy (Fig 3).[53,75,76] These painful oral ulcers, described as ovoid ulcers with erythematous halos, may be small and discrete or large (>1 cm), often classified as major aphthae.[77,78] The incidence is dose-dependent, and serum levels should be monitored to avoid supratherapeutic levels.[79,80] Management includes the use of topical high-potency corticosteroids, specifically dexamethasone oral solution (0.5 mg/5 mL, swish and spit), triamcinolone acetonide oral or dental paste, or clobetasol

Table I. Medication groups, cutaneous adverse effects, and timeline of presentation

Medication group	Cutaneous adverse effect	Time from medication initiation to event
Induction agents		
Antithymocyte globulin	Serum sickness	7-14 d after first exposure
Alemtuzumab	Vitiligo	1 y after initiating therapy
	Alopecia areata	After second infusion
	Chronic urticaria	After second infusion
Basiliximab	Herpes reactivation	Weeks to months
	Hypertrichosis	Weeks to months
	Hypersensitivity	Within minutes to 24 h
Calcineurin inhibitors		
Cyclosporine	Hypertrichosis	Within 1-6 mo, usually within 3 mo
	Gingival hyperplasia	Within first 6 mo
	Sebaceous hyperplasia	With prolonged use (months to years)
	Acneiform eruptions	Within first few weeks-months
	Bacterial folliculitis	Not well defined
	cSCC	Chronic exposure (months to years)
	KS	Chronic exposure (months to years)
Tacrolimus	Diffuse nonscarring alopecia	Highly variable (reported after a mean of 5 y)
Antimetabolites		
Mycophenolate mofetil	Maculopapular rash	Not well defined
	Aphthous ulcers	Months-1 y
	Nonscarring alopecia	Highly variable
Azathioprine	Photosensitivity	Not well defined
	Hypersensitivity syndrome	Days to weeks
	cSCC	Chronic exposure (reported after median of 6.7 y)
mTOR inhibitors		
Sirolimus, Everolimus	Aphthous stomatitis	Within 8 wk
	Wound-healing delay	Perioperative or after trauma
	Peripheral edema	Weeks to months
	Lymphedema	Weeks to months
	Acneiform eruptions	Within first month
Sirolimus	Erosive pustular dermatosis	Weeks to months
	Palmoplantar peeling	Within first few days to weeks
Corticosteroids	Skin atrophy	Weeks to months (dose-dependent)
	Striae	Weeks to months (dose-dependent)
	Bruising	Weeks to months (dose-dependent)
	Delayed wound healing	Perioperative or after trauma
	Acneiform eruptions	Weeks to months (dose-dependent)
	Hirsutism	Weeks to months (dose-dependent)
Co-stimulation blockade		
Belatacept	Cutaneous PTLD	~1 year

cSCC, Cutaneous squamous cell carcinoma; *KS*, Kaposi sarcoma; *PTLD*, post-transplant lymphoproliferative disorder; *mTOR*, mechanistic target of rapamycin.

propionate gel. Antiseptic mouth rinses and viscous lidocaine or magic mouthwash are also helpful for symptom control.[81] In severe or refractory cases, dose reduction or discontinuation of sirolimus may be necessary.[78,81] Persistent, atypical, or nonhealing ulcers should prompt evaluation for opportunistic infection, particularly cytomegalovirus.[82]

Acneiform eruptions develop in 15% to 45% of patients on sirolimus, often within the first month of therapy initiation.[75,83-85] These present as erythematous inflammatory papules and pustules, primarily involving the face and upper trunk.[85] Treatment consists of standard acne therapies.[86] Less commonly, erosive pustular dermatosis of the scalp has been reported and may present with erosions, crusting, and scarring alopecia.[87] Recognition of this entity is critical, as resolution often requires discontinuation of the offending agent.[87]

mTORi are strongly associated with wound-healing complications, occurring in 5% to 47% of patients taking sirolimus and 6% to 40% of those on everolimus.[86,88-92] To reduce the risk of these adverse

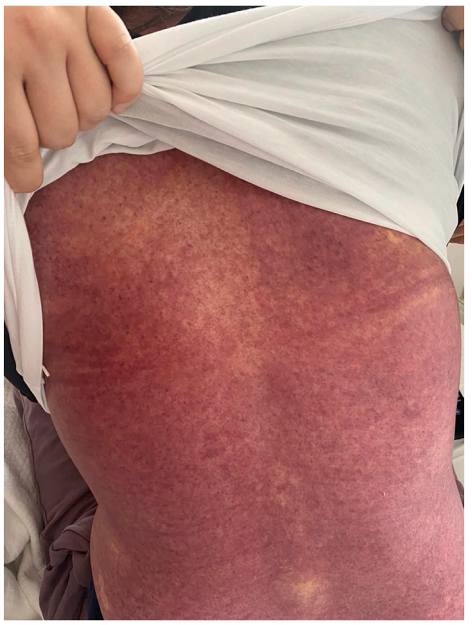

Fig 1. Serum sickness secondary to antithymocyte globulin characterized by a widespread erythematous, morbilliform eruption on the dorsal trunk. Figure adapted from Aydın M.S. et al., *Haydarpaşa Numune Medical Journal* (2024), under CC BY-NC 4.0.

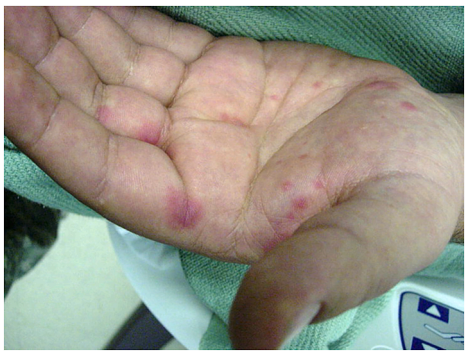

Fig 2. Azathioprine hypersensitivity syndrome presenting with indurated papules on palm of left hand. Reprinted from Bidinger JJ, Sky K, Battafarano DF, Henning JS. The cutaneous and systemic manifestations of azathioprine hypersensitivity syndrome. J Am Acad Dermatol. 2011;65(1):184-191. Copyright © 2011, with permission from Elsevier.

effects, careful dosing is recommended. Sirolimus should be maintained at trough levels between 5-10 ng/mL, and everolimus between 3-8 ng/mL, while avoiding high loading doses altogether.[93]

Peripheral edema occurs in up to 22% to 65% of patients on mTORi and may present with unilateral or bilateral limb swelling.[94-96] Most cases respond to dose reduction and supportive measures such as low-dose diuretics.[96]

Lymphedema, though less common, has been reported with sirolimus and everolimus and may progress to chronic stasis changes if unrecognized.[97-104] Management includes compression garments, lymphatic drainage, and elevation; drug discontinuation is often required in resistant cases.[103,105]

Palmoplantar peeling has also been described following initiation of sirolimus and may cause discomfort with daily activities.[106] While mild cases may resolve spontaneously, persistent or symptomatic desquamation should prompt consideration of dose reduction or discontinuation.[106]

Corticosteroids

Corticosteroids are widely used in solid organ transplantation for induction, maintenance immunosuppression, and treatment of acute rejection.[1] They exert their immunosuppressive effects by inhibiting

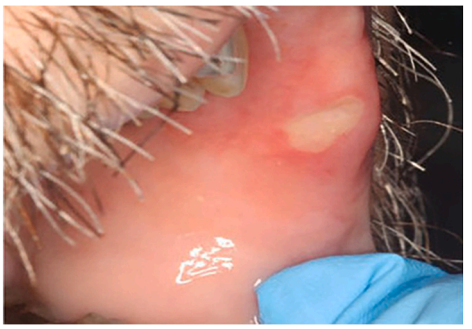

Fig 3. Aphthous ulcer with surrounding erythema on buccal mucosa secondary to mycophenolate mofetil. Reproduced from Sonis ST, Villa A. A New Hypothesis Describing the Pathogenesis of Oral Mucosal Injury Associated with the Mammalian Target of Rapamycin (mTOR) Inhibitors. Cancers. 2023;15(1):57, with permission from the authors under the terms of the Creative Commons CC BY license.

pro-inflammatory cytokines, suppressing T-cell activation and proliferation, and inducing lymphocyte apoptosis. Because corticosteroids are often continued long term, clinicians should anticipate predictable dose- and duration-dependent cutaneous adverse effects.[107] Common manifestations include skin atrophy, striae, bruising, impaired wound healing, hirsutism, and acneiform eruptions.[108] Dose tapering, transitioning to steroid-sparing agents, and patient education are key strategies across multiple complications.

Co-stimulation blockade

Belatacept is a fusion protein that inhibits the CD80/CD86—CD28 co-stimulatory signal required for T-cell activation and is used as a calcineurin inhibitor—sparing maintenance immunosuppressant.[109] Cutaneous adverse events are relatively infrequent but include occasional reports of nonspecific morbilliform eruptions, pruritic eczematous dermatitis, and rare cases of post-transplant lymphoproliferative disorder (PTLD) with cutaneous involvement, particularly in Epstein-Barr virus -seronegative recipients.[110-113] Suspicion for cutaneous PTLD should prompt immediate biopsy and coordination with transplant and oncology teams, as first-line management involves reduction of immunosuppression, with rituximab and tabelecleucel reserved for refractory disease.[114]

While earlier studies suggested reduced NMSC risk when switching from CNIs to belatacept,[115] a recent single-center study found no significant reduction in skin cancer incidence with de novo belatacept use.[116] Clinicians should be cautious in assuming cutaneous benefit in de novo settings and continue routine dermatologic surveillance.

Tegoprubart, an anti-CD40L antibody currently in clinical development, represents another potential co-stimulation pathway therapy, though transplant-specific dermatologic data are not yet available.

SKIN CANCER RISK

SOTRs face increased skin cancer risk from chronic immunosuppression and cumulative ultraviolet exposure (Table II).[117] These include keratinocyte carcinomas such as basal cell carcinoma and cSCC, as well as virus-associated malignancies like Kaposi Sarcoma (KS) and Merkel cell carcinoma. Among these, cSCC is the most common, with incidence rates 65 to 250 times higher than in the general population.[118-122] Given this elevated risk, dermatologists should take a proactive role in early detection, prevention, and longitudinal surveillance of transplant recipients.

Skin cancer risk varies by transplanted organ, immunosuppressive intensity, and patient-specific factors. Heart and lung transplant recipients warrant particular vigilance, as these patients typically require more intensive and prolonged immunosuppression.[118-122] Virus-associated malignancies, including KS and Merkel cell carcinoma (MCC), also occur at increased frequency, and new or atypical vascular or rapidly growing lesions should prompt early biopsy.[123-126] The cornerstone of KS management in SOTRs is reduction or modification of immunosuppression (ie substituting CNIs such as cyclosporine or tacrolimus).[43-45] Systemic chemotherapy is generally reserved for patients with extensive, visceral, or refractory disease.[43]

Risk stratification tools such as the Skin and Ultraviolet Neoplasia Transplant Risk Assessment Calculator (SUNTRAC) integrate readily available clinical variables to estimate post-transplant skin cancer risk.[127,128] Incorporation of validated tools such as SUNTRAC may help guide timing of dermatology referral and surveillance intervals, particularly in

Table II. Skin cancer risk by immunosuppressant

Medication	Skin Cancer Risk	TBSE Recommendation
Cyclosporine	+++	Every 6-12 mo
Azathioprine	+++	Every 6-12 mo
Tacrolimus	++	Annually or per clinical risk
Antithymocyte globulin	+	Annually or per clinical risk
Alemtuzumab	+	Annually or per clinical risk
Mycophenolate mofetil	+	Annually or per clinical risk
Corticosteroids	+	Annually or per clinical risk
Belatacept	+	Annually or per clinical risk
Basiliximab	+/−	Annually or per clinical risk
Sirolimus	+/−	Annually or per clinical risk
Everolimus	+/−	Annually or per clinical risk

+++ indicates high risk; ++ indicates moderate risk; + indicates low risk; +/− indicates no increased risk or potentially protective. Skin cancer risk reflects relative risk for cSCC and other nonmelanoma skin cancers in SOTRs based on current evidence. TBSE recommendations are based on current literature. Skin cancer risk should be evaluated on an individual basis, considering additional factors such as Fitzpatrick skin type, history of prior skin cancer, cumulative immunosuppression, sun exposure, and age. We recommend incorporating validated risk stratification tools such as SUNTRAC to guide surveillance intervals and clinical decision-making.

centers managing large transplant populations. While SUNTRAC has been validated in diverse cohorts, clinical judgment remains essential, especially for patients with skin of color, in whom cancer may present later and at more advanced stages.[127]

Immunosuppressive agents further modify risk. AZA and cyclosporine are linked to increased skin cancer incidence, while methotrexate and mycophenolate appear to confer lower risk.[117,129] In patients with multiple or high-risk cSCCs, modification of immunosuppressive regimens, including conversion from calcineurin inhibitors to mTOR inhibitors, should be considered through shared decision-making with transplant teams, balancing oncologic benefit against rejection risk.[130-133]

Preventive strategies include systemic chemopreventive agents, such as acitretin, capecitabine, and nicotinamide, as well as topical treatments like 5-fluorouracil and photodynamic therapy for field cancerization.[134-137]

PERIOPERATIVE CONSIDERATIONS

Dermatologic surgeons treating SOTRs must consider the complex interplay between immunosuppressive therapies and wound healing. Preoperative planning should include review of the patient's immunosuppressive regimen, as specific agents, particularly mTOR inhibitors, are associated with an increased risk of wound complications. For patients undergoing major surgery, temporary interruption of mTOR inhibition 1-2 weeks preoperatively with low-dose corticosteroid bridging is recommended, with resumption after adequate wound healing is confirmed.[86,93,138-142] For minor cutaneous procedures, continuation of immunosuppression is appropriate provided close postoperative monitoring is ensured. Coordination with transplant teams is essential when perioperative modification of immunosuppression is considered. Dermatologic surgeons should maintain a low threshold for infection surveillance and emphasize meticulous wound care, especially following Mohs micrographic surgery or procedures requiring complex closure.[143,144]

CONCLUSION

Dermatologists play a central role in monitoring, preventing, and managing the cutaneous consequences of immunosuppression in SOTRs. Incorporating dermatologic considerations into immunosuppressive decision-making, particularly in patients at elevated risk for skin cancer or wound-healing complications, can improve clinical outcomes. Close collaboration with transplant teams ensures that immunosuppression is tailored to balance graft preservation with dermatologic risk.

Declaration of generative AI and AI-assisted technologies in the writing process

AI was not used in this manuscript.

Conflicts of interest

None disclosed.

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