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TABLE OF CONTENTS

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Review of Pemphigus Vulgaris Management

Bahar Bahrani, MD, FRCPC

Introduction

Pemphigus Vulgaris (PV) is an autoimmune blistering disease that is characterized by painful erosions and flaccid blisters involving the mucous membranes and skin (**Figure 1**). The production of pathogenic immunoglobulin autoantibodies, mainly IgG4, against the desmosomal cadherins desmoglein 1 (Dsg-1) and desmoglein 3 (Dsg-3), lead to the loss of epidermal keratinocyte adhesion. In the mucosal dominant type, Dsg-3 antibodies are present, while in the mucocutaneous type, antibodies against both desmoglein 1 and 3 are found. Diagnosing PV requires compatible clinical features, histopathological examination of the involved skin, and the detection of autoantibodies by indirect immunofluorescence of non-affected peri-lesional skin. The pemphigus disease area index (PDAI) and the autoimmune bullous skin disorder intensity score (ABSIS) provide a standardized assessment of disease extent and response to treatment.

A treatment algorithm has been proposed for PV (**Figure 2** and **Figure 3**). In addition, supportive management is also important (**Table 1**).

Treatment

Systemic Steroids

Systemic steroids play an important role in the initial treatment of PV due to their high efficacy and ability for achieving rapid control. Prednisone is the systemic steroid therapy of choice, with doses ranging from 0.5–1 mg/kg/day.[1] Typically, mild PV is treated with 0.5 mg/kg/day, while moderate to severe PV is treated with 1 mg/kg/day, with a usual maximum of 60 mg/day. In mild PV <5% of the total body surface is involved, and there are no oral lesions or only mild oral lesions that do not impair food intake or require topical analgesics. In moderate-to-severe PV >5% of the body surface area is involved, with multiple mucosal

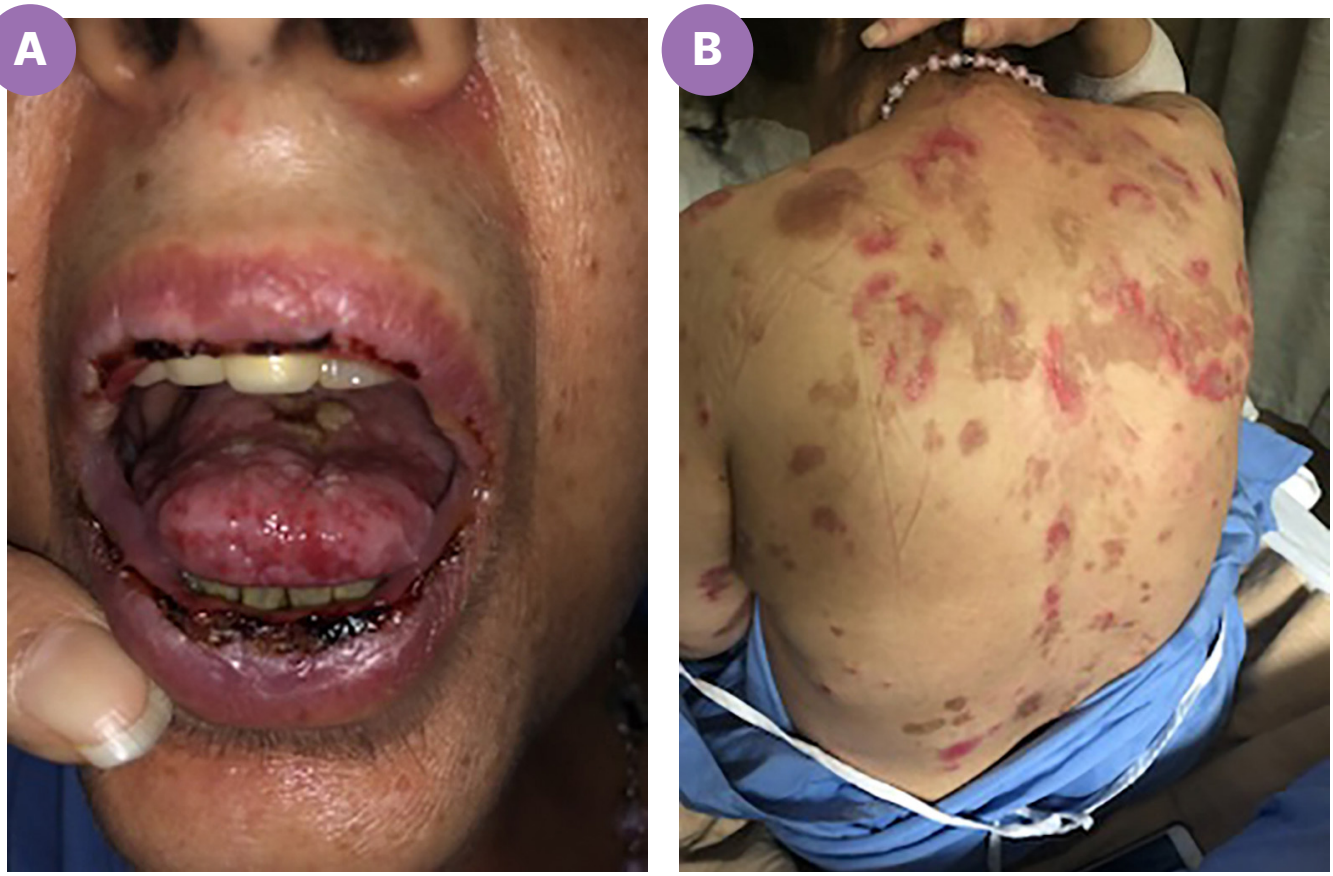

Figure 1. A) Erosions on the tongue and hemorrhagic crust on the lips are observed in a patient with oral pemphigus vulgaris. **B)** Superficial erosions and post-inflammatory hyperpigmentation are observed on the back and arms of a patient with cutaneous pemphigus vulgaris; *courtesy of Bahar Bahrani, M.D., FRCPC.*.

sites, severe oral lesions, dysphagia, weight loss, and significant pain. In the majority of patients, blisters cease within 2–3 weeks and full disease control is achieved within 6–8 weeks.[2] If there is no response to oral steroids after 3–4 weeks, intravenous pulse therapy with 0.5-1 g/day methylprednisolone for 3–5 consecutive days can be added.[3] This treatment requires admission to hospital, as continuous cardiac monitoring is needed.

Steroid therapy can be tapered once there is cessation of new blister formation and established blisters have healed by approximately 80%. The objective is to reduce the steroid dosage to the lowest level that maintains good disease control. Dose reduction should occur in a stepwise fashion, however, there is no standardized approach to this. One proposed regimen is to reduce the dosage from 1 mg/kg to 0.75 mg/kg once no new lesions occur for 1 week, then to 0.5 mg/kg if no new lesions occur for at least 1 week, then to 30 mg if no new lesions are seen for at least 2 weeks. From that point the dosage can be lowered by 2.5–5 mg every 2 weeks until it reaches zero. Another well-described tapering plan is to reduce the dose by 25% every 2 weeks until reaching a dose of 20 mg per day, then decreasing by 2.5 mg per week until reaching 10 mg per day, and decreasing by 1 mg per day thereafter.[4] There are serious potential side effects associated with long-term use of prednisone that include infection, osteoporosis, hyperglycemia, hypertension, and suppression of the adrenal glands.

Rituximab

Rituximab is a monoclonal antibody targeting CD20 on mature B cells, which causes B-cell depletion and reduced antibody production. The preferred initial treatment for PV is a combination of rituximab and prednisone. However, the high

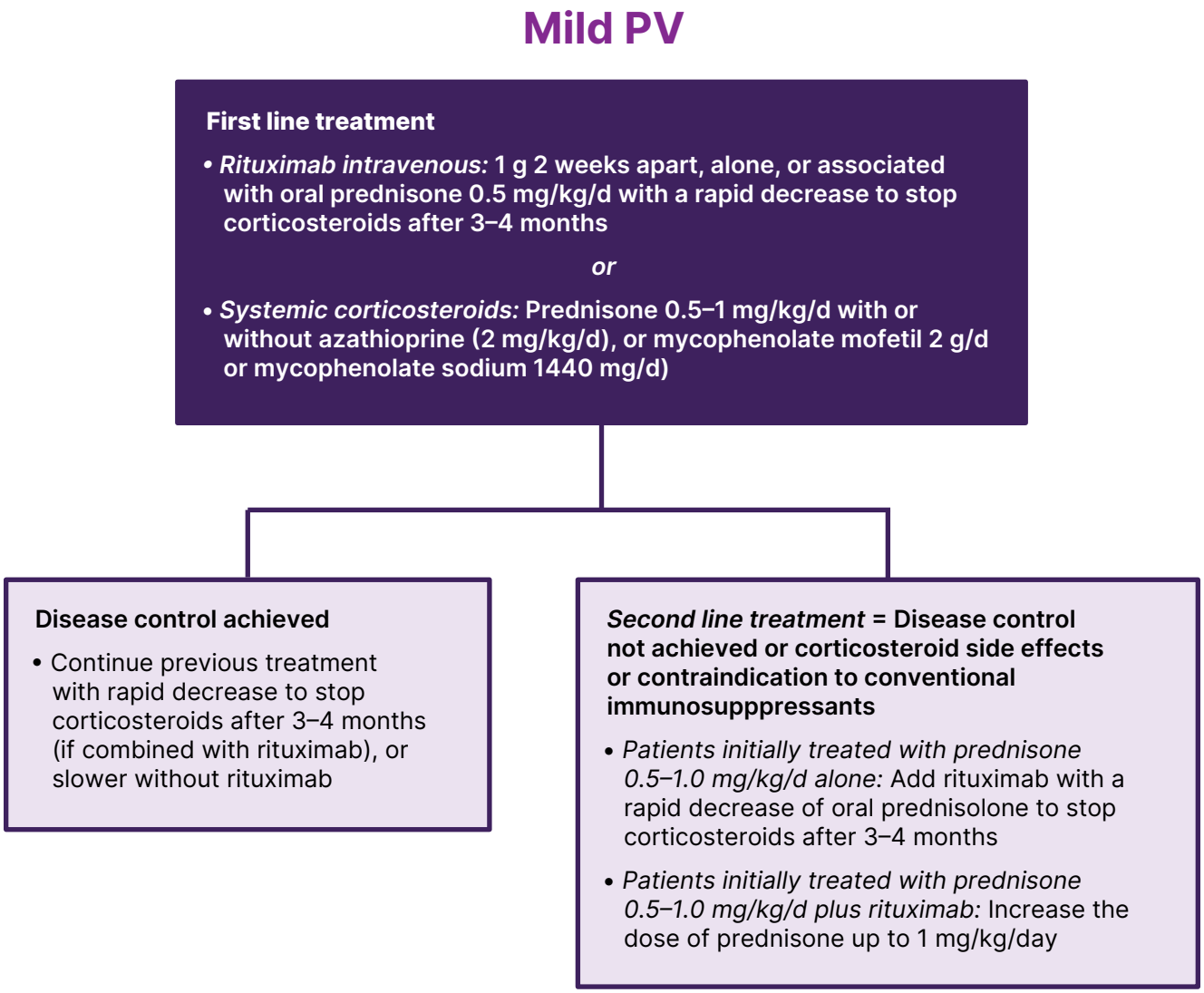

Figure 2. Treatment algorithm for mild pemphigus vulgaris; *adapted from Joly 2020.*[3]

cost and limited availability of rituximab can restrict its use. When rituximab therapy is not possible, prednisone and adjuvant mycophenolate mofetil or azathioprine is recommended.

A study compared 2 treatment regimens with rituximab (1000 mg on days 1 and 14, then 500 mg at 12 and 18 months) plus prednisone (0.5 mg/kg/d for moderate disease and 1 mg/kg for severe disease) tapered over 3–6 months, or prednisone alone (1 mg/kg/day for moderate disease and 1.5 mg/kg/day for severe disease) tapered over 12–18 months. A much larger proportion of patients receiving rituximab and prednisone achieved clinical remission off therapy, reached complete remission much sooner, and experienced less frequent adverse events.[5]

In a randomized trial that compared rituximab plus prednisone versus mycophenolate mofetil plus prednisone, the rituximab/prednisone combination was superior.[6] This study showed a higher likelihood of achieving sustained complete remission, lower dependence on oral steroids, and reduced disease flares in patients with moderate to severe PV in the rituximab/prednisone group. While rituximab has not been directly compared

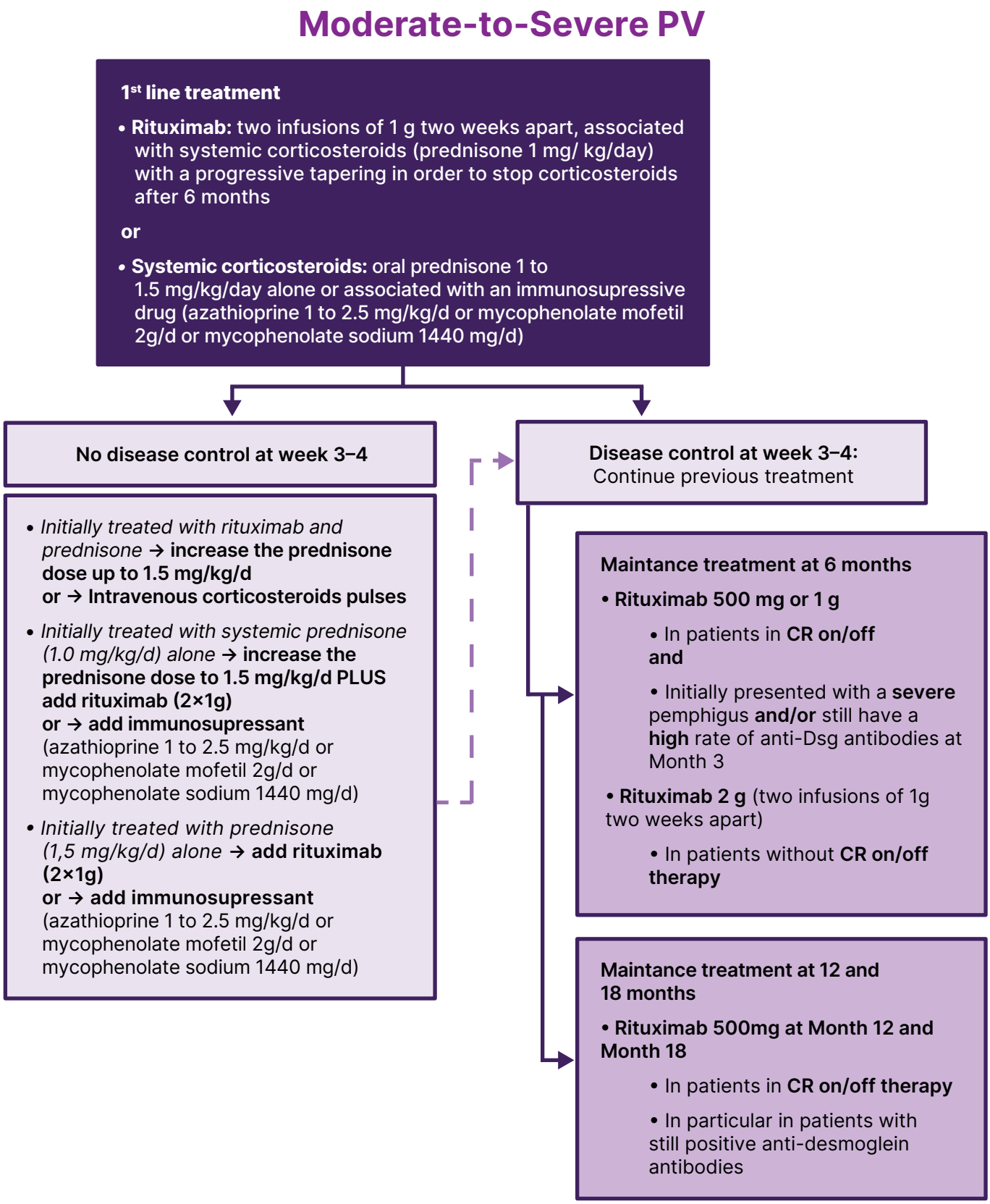

Figure 3. Treatment algorithm for moderate-to-severe pemphigus vulgaris; *adapted from Joly 2020.*[3]

Supportive Care	
Oral symptoms	
Oral hygiene	• Regular brushing with a soft-bristle brush • Bland toothpaste • Daily flossing • Regular professional dental cleaning
Avoidance of certain foods	• Avoid spicy, hot, sharp, or abrasive foods
Topical Anesthetics	• Lidocaine 2% solution or gel as needed
Topical anti-inflammatory therapies	• High-potency topical corticosteroids (fluocinonide or clobetasol in ointment or gel) twice a day as needed • Steroids mouthwash (dexamethasone 0.5 mg/5 ml or prednisolone 5 mg/5 mL) 2–3 times a day to swish and spit as needed • Intralesional triamcinolone acetonide to help with isolated persistent lesions • Tacrolimus 0.1% ointment twice a day as needed
Treatment of Candida	• Nystatin swish and spit or fluconazole
Skin Symptoms	
Wound care	• Erosions should be covered with bland emollient (e.g. petrolatum) +/- non-adhesive wound care dressing
Anti-inflammatory therapies	• High-potency topical corticosteroids (fluocinonide or clobetasol in ointment) twice a day as needed • Intralesional triamcinolone acetonide to help with isolated persistent lesions • Tacrolimus 0.1% ointment twice a day as needed
Treatment of secondary infections	• Treat for secondary infections with herpes simplex virus and bacterial infections

Table 1. Supportive adjunct therapies in pemphigus vulgaris; *courtesy of Bahar Bahrani, MD, FRCPC*.

to azathioprine, a meta-analysis suggests a greater likelihood of disease remission and lower cumulative steroid doses with rituximab.[7]

Rituximab is administered as a 1000 mg intravenous solution, given 2 weeks apart, followed by periodic maintenance doses as needed. Typically, steroid therapy begins before rituximab, as the effects of rituximab may not appear until 8–12 weeks after therapy. The major risks of rituximab are infusion reactions and infections. Progressive multifocal leukoencephalopathy is a rare complication that has been reported in patients treated with rituximab for other indications.

Azathioprine

Azathioprine downregulates purine metabolism and reduces the activity of T and B lymphocytes. Reduced dosing is recommended for patients with low or intermediate

thiopurine-methyltransferase (TPMT) activity and is not advised for those with absent TPMT activity.[3] The usual starting dose is 1 mg/kg (ideal body weight) per day and can increase by increments of 0.5 mg/kg to reach a maximum of 2.5 mg/kg for treating PV. It is important to note that a normal TPMT level does not exclude the possibility of myelotoxicity, thus, regular monitoring of blood counts is critical. Severe adverse effects include pancytopenia and hepatotoxicity.

The findings from studies evaluating the effectiveness of azathioprine have been conflicting. A randomized trial compared the use of prednisolone 2 mg/kg/day (maximum 120 mg per day) alone and in conjunction with azathioprine (2.5 mg/kg). After one year, patients receiving prednisone and azathioprine had significantly lower doses of prednisolone than those receiving prednisolone alone. It should be noted that clinical outcomes with complete remission were similar in both groups.[8] In a similar study, no differences were noted in the mean total dose of prednisolone.[9] A systematic review and analysis showed that azathioprine had superior steroid-sparing effects compared to mycophenolate mofetil (MMF).[10] Although there is more evidence to support the use of azathioprine, MMF is more frequently used in PV due to its more favourable side effect profile and ease of use.

Mycophenolate Mofetil

MMF depletes guanosine nucleotides in T and B lymphocytes and inhibits their proliferation, thereby suppressing cell-mediated immune responses and antibody formation. MMF is used at a dose of 2–3 g/day in PV. The gastrointestinal side effects of MMF may be overcome with the use of enteric-coated mycophenolate sodium. MMF has a similar action to azathioprine, but with less myelosuppression but more gastrointestinal side effects.

The findings from studies on the steroid-sparing effects of MMF in PV have been conflicting. Two randomized trials found a statistically significant reduction in steroid use in patients who received MMF, while one randomized trial failed to show this.[11] A network meta-analysis of randomized trials also did not show a steroid-sparing effect.[7] A meta-analysis compared azathioprine and MMF and confirmed that MMF was inferior in its steroid-sparing effect.[10] Drug adverse effect profiles, comfort, and familiarity of clinicians generally guide selection between these two drugs.

Recalcitrant and Emerging Therapies

Refractory cases of PV require escalation of management with therapies such as intravenous immunoglobulin (IVIG), immunoadsorption, plasmapheresis, and cyclophosphamide. These therapies are usually added to a baseline immunosuppression regimen that includes steroids plus conventional immunosuppressants. However, due to the high costs, availability, and the technical difficulty associated with administering IVIG, plasmapheresis, and immunoadsorption, these treatments are limited to refractory cases only. Cyclophosphamide is considered as a rescue drug and is reserved for recalcitrant cases due to its unfavourable side effect profile, which includes cytopenia, sterility, and bladder cancer. Numerous new emerging therapies are currently being studied for PV (**Table 2**).[12]

IVIG

IVIG, a blood product that is comprised of pooled plasma, is used for its immunomodulatory effects in several inflammatory conditions. Its mechanism of action involves the degradation of immunoglobulins by binding to the neonatal Fc Receptor (FcRn). Considering that IVIG is not immunosuppressive, it can be administered in combination with systemic steroids and other immunosuppressants in cases of recalcitrant PV.[13] Due to its rapid onset of action, IVIG can be administered prior to assessing the response to rituximab in severe cases. A multi-centre randomized, placebo-controlled, double-blind study showed that administering IVIG at 2 g/kg over 5 consecutive days every 4 weeks is a safe and effective treatment option in steroid-resistant PV.[14] Side effects of IVIG include headache, nausea, fevers, tachycardia, aseptic meningitis, acute renal failure, and thromboembolic events. Aseptic meningitis is a serious side effect that requires immediate termination of treatment. The

Target	Category	Approved	Under Trial	Candidates
B-cell	CD20 mAb (First generation)	Rituximab		
	CD20 mAb (Second generation)			veltuzumab ocrelizumab
	CD20 mAb (Third generation)		ofatumumab	obinutuzumab ocaratuzumab
	CD19 mAb			inebilizumab
	BTK inhibitor	Rilzabrutinib[δ]	tirabrutinib	ibrutinib
Dsg3-specific B cells			CAAR-T cell	
T cell and T cell-B-cell interaction	CD25			daclizumab
	PolyTregs		NCT03239470	
Autoimmune cells				Autologous hematopoietic stem cell
Cytokines	TNF-α			etanercept infliximab
	IL-6			tocilizumab
	IL-4			dupilumab
	BAFF		VAY736	atacicept
Other	Fas ligand			PC111
	FcRn	SYNT001[δ]	efgartigimod	

Table 2. Emerging therapeutic agents and their status of clinical trial for pemphigus vulgaris; *adapted from Yuan 2022*.

Abbreviations: BAFF: B-cell-activating factor of the tumour-necrosis-factor family, **BTK:** Bruton tyrosine kinase, **CAAR:** chimeric autoantibody receptor, **Dsg-3:** Desmoglein 3, **FcRn:** neonatal Fc Receptor, **IL:** interleukin, **mAB:** monoclonal antibody

δ Granted Orphan Drug Designation by the United States FDA for PV therapy

absence (not deficiency) of serum IgA needs to be excluded prior to IVIG therapy, as this could lead to anaphylaxis.

Immunoadsorption/Plasmapheresis

Immunoadsorption removes circulating IgG autoantibodies, whereas plasmapheresis non-selectively removes plasma proteins from the circulation. The combination of immunoadsorption and immunosuppressive therapies is considered effective in treating severe PV as it is able to promptly remove the pathogenic autoantibodies.[15] The high cost, lack of availability in most countries, and potential side effects (venous thrombosis and infections) with immunoadsorption limit its use in treating PV. Plasmapheresis has limited efficacy in removing pathogenic autoantibodies and lacks high-quality trials showing its efficacy. As such, plasmapheresis is no longer a mainstay in the treatment of refractory PV.

CAR T-Cell Therapy

Chimeric antigen receptor (CAR) T cell technology has revolutionized cancer therapy and is currently being studied for PV. In this method, T cells from a patient's own blood are genetically modified in the laboratory to express a CAR that is able to recognize a specific target antigen. Engineered chimeric Dsg-3 autoantibody receptor (CAAR) T cells have been created for PV and show an affinity for Dsg-3 B cells leading to their selective elimination.[16] Studies using mouse models have shown that these CAAR T cells improve clinical outcomes in PV and reduce pathogenic IgG antibodies.

Adjunct Therapies

Due to its chronic and relapsing course, it is important to consider prophylactic medications to prevent complications from treatment for PV. The American College of Rheumatology guidelines recommend taking vitamin D (600–800 IU daily) and calcium (1000–1200 mg once daily) supplements to prevent osteoporosis during long-term (≥3 months) steroid therapy.[17] Bisphosphonates such as alendronate or risedronate can be initiated in patients with risk factors (postmenopausal women, men aged >50 years, positive osteoporosis screening score), although some guidelines advocate routine use of bisphosphonates with prednisone >7.5 mg/d for ≥3 months.[3,18] There is insufficient evidence to routinely add proton pump inhibitors for gastric ulcer prevention while on steroids. Thus, the decision can be individualized for each patient depending on their risk factors (concurrent non-steroidal anti-inflammatory use, prior history of gastric ulcers, comorbidities).[3] Prophylaxis for pneumocystis pneumonia (PCP) is not routinely indicated for PV patients, despite prolonged use of immunosuppressive therapies.[19]

Vaccinations

Prior to starting any immunosuppressive medication, a patient's vaccine history should be obtained, and they should be advised to receive any necessary vaccinations. Live vaccinations are contraindicated for patients on high dose steroids (>20 mg/day) and immunosuppressive medications, and should be delayed until at least one month after discontinuation, depending on the medication and its half-life.[20] It is recommended that patients on oral steroids or immunosuppressive therapy receive non-live vaccines such as inactivated influenza, inactivated shingles, COVID-19, and pneumococcal vaccines. For pneumonia vaccination, it is recommended to administer pneumococcal conjugate (PCV13) followed by a dose of the pneumococcal polysaccharide vaccine (PPSV23) at least 8 weeks later in patients on immunosuppressive medications. For vaccines given as a series (i.e. shingles vaccine, hepatitis B vaccine, etc.), the first dose should ideally be administered before starting therapy to ensure the best response. Other standard inactive vaccines (i.e. tetanus, diphtheria, pertussis, polio, among others) should also be up to date. Non-live vaccines should ideally be administered at a minimum 2 weeks prior to starting traditional immunosuppressive therapy (i.e. azathioprine, mycophenolate mofetil) to enhance vaccine immunogenicity. The administration of non-live vaccines should be delayed for a minimum of 2–4 weeks before starting rituximab.[21] Any additional or follow-up vaccines should be delayed until at least 6 months after rituximab therapy. Patients can receive non-live vaccines while on any immunosuppressant to acquire some immunity and can be revaccinated at a later time, especially if disease severity does not allow delays.

Conclusion

The objective of treating PV is to halt disease development and heal existing erosions. Rapid and effective treatment is critical for those with severe disease. Management of PV should be personalized for each patient depending on disease severity, age, comorbidities, and personal preferences. The use of systemic steroids and immunosuppressive agents has improved the prognosis of PV, however there can be significant morbidity associated with complications of this treatment. Larger randomized clinical trials are necessary to evaluate the efficacy of treatment agents in PV.

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Financial Disclosures

None declared.

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Recommendations and Basic Principles of Phototherapy

Tashmeeta Ahad, BM BCh(Oxon), MA(Cantab), MRCP(UK)(Derm)

Phototherapy has long been a cornerstone in dermatology, offered in most dermatology clinics globally. Despite the advent of several new biologic and systemic therapeutics, phototherapy remains a favoured treatment option due to its low side-effect profile and efficacy for treating mild-to-moderate inflammatory dermatoses. It can be used for a variety of skin conditions, including psoriasis, eczema, vitiligo, lichen planus, mycosis fungoides, pityriasis lichenoides, nodular prurigo, pruritus, and morphea.

In this article, we will provide an overview of the basic principles of phototherapy, as well as offering recommendations for managing a phototherapy service. Our focus will be on whole-body phototherapy, without the use of psoralens.

Introduction

Phototherapy, in the form of sunlight, has been used since ancient times, with historical evidence indicating its use in Egyptian and Indian cultures to treat skin diseases. The modern practice of phototherapy began with the pioneering work of Danish physician Niels Finsen, who used ultraviolet (UV) radiation to treat lupus vulgaris. For his contributions, Finsen was awarded the Nobel Prize in 1903.[1,2]

By the 19th century, phototherapy began to become established as a medical treatment. In 1925, Goeckerman combined UV radiation with crude coal tar to treat psoriasis. However, it was not until the 1970s that broadband UVB (BB-UVB) became a widely accepted treatment for various inflammatory skin diseases. A paradigm shift occurred in the 1980s, when Parrish and Jaenicke demonstrated that the action spectrum and most effective therapeutic wavelength for treating psoriasis was 313 nm. Following this, Philips introduced fluorescent narrow-band (NB-UVB) phototherapy lamps that emit light between 310 and 311 nm, which were later adopted by other manufacturers.[1,2]

Phototherapy and Mechanism of Action

Ultraviolet radiation (UVR) exerts its effects on the skin through multiple biological mechanisms, involving both the innate and adaptive immune systems. It has an immunosuppressive effect on T-cell function and induces antigen-specific tolerance. In addition, UVB reduces DNA synthesis, which is helpful for skin conditions such as psoriasis, where there is accelerated DNA synthesis. It induces the production of cytokines such as interleukin (IL)-6, IL-1, and activates pathways including the expression of tumour suppression gene p53, which leads to cell cycle arrest and allows for DNA repair. Over time, phototherapy leads to epidermal hyperplasia and tanning.[3]

Wavelengths associated with each type of phototherapy are shown in **Table 1**. In simple terms, the longer the wavelength, the deeper it penetrates into the skin. UVB mainly penetrates into the epidermis and upper dermis. In contrast, UVA is able to penetrate to the deep layers of the dermis and potentially beyond. When choosing phototherapy wavelengths, it is important to consider the following factors, such as photon energy (inversely proportional to wavelength) and action spectrum (measure of the importance of each wavelength in producing a particular photobiologic response). Nucleic acids, DNA, and chromophores in skin mainly absorb UV photons at approximately 300 nm (UVB). Apart from DNA and nucleic acids, UVR also acts on other chromophores such as urocanic acid and tryptophan found in skin. UVR may also induce apoptosis of keratinocytes forming 'sunburn cells'. The mechanism of DNA damage underpinning photocarcinogenesis includes the creation of dimeric DNA photoproducts, namely **i)** Cyclobutane pyrimidine dimers, and **ii)** 6–4 photoproducts. In contrast to UVB, UVA causes DNA damage via indirect photon absorption, such as type 1 and type 2 photosensitized reactions, and the creation of reactive oxygen species and free radical damage.[1,2,4]

Phototherapy Type	Wavelength Range *(nm)*	Penetration Depth
NB-UVB	311–313	Epidermis
BB-UVB	280–320	Epidermis, upper dermis
Combined UVAB	280–400	Deep dermis
UVA1	340–400	Deep dermis

Table 1. Wavelengths associated with different types of phototherapy; *courtesy of Tashmeeta Ahad, BM BCh(Oxon), MA(Cantab), MRCP(UK)(Derm)*.

Abbreviations: NB-UVB: narrow-band ultraviolet B, **BB-UVB:** broadband ultraviolet B, **UVAB:** ultraviolet A and B, **UVA1:** ultraviolet A1

Phototherapy Modalities

Phototherapy modalities used for whole-body phototherapy are shown in **Table 1**. Narrow-band UVB (NB-UVB) is the most commonly used type of phototherapy, although centres such

as ours in Vancouver, British Columbia offer BB-UVB combined UVA and UVB as well as UVA1 whole-body phototherapy.

Phototherapy is typically administered over several weeks, with patients undergoing treatment 2–3 times a week. Typically, to treat skin conditions such as psoriasis or eczema, patients may require at least 25 treatments to achieve adequate improvement. The UV dose administered is increased over time as photoadaptation occurs, allowing patients to tolerate higher doses without burning. This is due to epidermal hyperplasia and tanning. The objective is to aim for a suberythemogenic dose each time, which is just below the threshold required to cause erythema (redness/sunburn) of the skin, to achieve a photobiologic response.

Phototherapy Equipment

There are various types of phototherapy devices used to treat patients, ranging from full-body units to targeted handheld devices. Most hospital clinics and dermatology offices will incorporate full-cabinet devices, although 3D panels and single panels may be used for home phototherapy. Handheld devices are also available. Phototherapy machines use specialized UV lamps, most commonly fluorescent bulbs. Traditionally, mercury arc lamps were used.[1,2] More recently, LED-based systems are being explored, due to their ability to provide precise wavelengths with energy efficiency.

Dosimetry and Calibration

Accurate dosimetry is critical in phototherapy to ensure patients receive the correct dose. UVB and UVA irradiance must be regularly monitored using calibrated metres. Any changes in lamp performance, such as bulb replacements, can alter the output and must be adjusted to maintain consistent dosages. Dosimeters integrated into the cabinet may be unreliable, although for an office-based practice they may have to be relied upon if access to external calibration checks, such as those provided by medical physics, are unavailable.[1,5] These differences mean that UV dose outputs across different machines and clinics are often not comparable. It is important to periodically verify the irradiance of phototherapy equipment. The following formula can be used for calculating the dose and corresponding exposure times to make calibration adjustments:

Irradiance (mW/cm^2) x Time (seconds) = Dose (mJ/cm^2)

Treatment Protocols

Although published references for treatment protocols, such as by the American Academy of Dermatology,[6] are available, these protocols may need to be modified based on various factors. These include the type of equipment used and its calibration, the skin phototype profile of the patient population being treated, the available clinical supervision in clinics (e.g. nurse/medical office assistant), and ability to manage adverse effects such as erythema. These and other factors may influence the 'aggressiveness' of a treatment protocol.

There are 3 main components to consider when determining a phototherapy treatment protocol.

- ***Starting Dose:*** Determined based on the patient's Fitzpatrick skin type or Minimal Erythema Dose (MED).
- ***Incremental Doses:*** Increased gradually over time to maximize efficacy while avoiding erythema. Typical increments tend to be 10% or 20%.
- ***Frequency:*** Treatment frequency typically starts at 2–3 times per week.

Starting Dose

Different individuals are likely to tolerate varying initial doses of UV. Factors influencing this include skin pigmentation, phenotype, medications which may cause drug induced photosensitivity,[8] and underlying photosensitivity disorders.[9] The starting dose for phototherapy can be chosen based on an individual's MED, or empirically chosen based on skin pigmentation properties, such as the Fitzpatrick skin phototype[7] (**Table 2**). The MED is defined as the minimum UV dose

Fitzpatrick Skin Phototype	
I	Always burns, does not tan
II	Burns easily, tans poorly
III	Tans after initial burn
IV	Burns minimally, tans easily
V	Rarely burns, tans darkly easily
VI	Never burns, always tans darkly

Table 2. Fitzpatrick Skin Phototype.[7]

Skin Phototype	NB-UVB Starting Dose (mJ/cm^2)	BB-UVB Starting Dose (mJ/cm^2)
I	130	20
II	220	25
III	260	30
IV	330	40
V	350	50
VI	400	60

Table 3. Example starting dose for UVB (in mJ/cm^2); *courtesy of Psoriasis and Phototherapy Clinic, Vancouver General Hospital.*

Abbreviations: NB-UVB: narrow-band ultraviolet B, **BB-UVB:** broadband ultraviolet B

that induces erythema within 24 hours. In Europe and the UK, a patient's MED may be used as the primary method of selecting a starting dose for phototherapy. However, due to time and labour constraints, most North American clinics tend to use empirical dosing based on the Fitzpatrick skin phototype.

The starting dose for phototherapy is typically set at 70% of a patient's MED or is based on empirical guidelines tailored to the Fitzpatrick skin phototype (**Table 3**).

Incremental dose

Typically, incremental dosing of 10%–20% may be used, with treatment administered 2–3 times a week. The maximum doses can reach approximately 3000 mJ/cm^2, as tolerated. Incremental dosing may need to be adjusted depending on side effects, most commonly erythema (**Table 4**) and if treatments are missed (**Table 5**).

Adverse Events and Safety Procedures

Patients should be informed about both acute and chronic side effects and provide consent. It is advisable to incorporate a patient consent form and provide written patient information before starting phototherapy. A nursing education session or similar briefing, prior to starting a treatment course, which outlines what phototherapy entails, safety procedures, expectations, and potential adverse events will help mitigate medicolegal risks. Patients should be advised on the importance of protective equipment such as eye goggles and of wearing the same clothing to avoid non-photoadapted skin from becoming exposed or burned. The doses administered and side effects observed should be documented in detail following each visit.[5]

Acute Side Effects

- Erythema/burning
- Tanning
- Reactivation of herpes virus infection (cold sore)
- Itching
- Activation of photosensitivity disorder

Chronic Side Effects

- Photoageing
- Skin cancer risk is theoretical with UVB phototherapy, because existing literature has shown no evidence of increased skin cancer risk with UVB phototherapy.[10,11] However, guidelines recommend offering routine skin cancer screening to patients who have had more than 500 UVB exposures or may have other risk factors.[5]

Grade of Erythema	Reaction to Previous Exposure	Dose Increment
0	No erythema or pain	10–20%
1	Mild erythema without pain	5–10%
2	Mild erythema with minimal pain/discomfort lasting <24 hours	0% Keep same fluence
3	Moderate erythema with pain/discomfort lasting >24 hours	-10%
4	Severe erythema with symptoms e.g., blistering/tenderness	Hold treatment for at least 1 week

Table 4. Adjustment to UV incremental dosing based on erythema/adverse events. (Approximate values; variations possible); *courtesy of Psoriasis and Phototherapy Clinic, Vancouver General Hospital.*

Time Since Last Treatment	Decrease in Fluence
1 week	25%
2 weeks	50%
3 weeks	75%
4 weeks	Baseline fluence

Table 5. Adjustment to UV incremental dosing for missed treatments. (Approximate values; variations possible); *courtesy of Tashmeeta Ahad, BM BCh(Oxon), MA(Cantab), MRCP(UK)(Derm).*

Conclusion

Phototherapy remains a fundamental treatment in dermatology for inflammatory skin diseases. By understanding the underlying mechanisms, using equipment appropriately, and individualized treatment protocols for each patient, dermatologists can provide effective and safe phototherapy services.

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None declared.

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Clascoterone Cream 1% in Acne Management: Case Series and Real-World Canadian Experience

Wei Jing Loo, BSc, MBBS, MRCP, FRCP

Acne vulgaris is a globally prevalent dermatological condition associated with a substantial physical and psychological burden.

This case series includes 10 patients with acne vulgaris who received treatment with clascoterone cream 1% from August 2023 to May 2024. Treatment with clascoterone cream 1% was effective and well tolerated regardless of acne severity, age, gender, and ethnicity. Clascoterone led to clinical improvement when used as monotherapy, as an adjunctive treatment in combination with other topical or systemic agents or laser therapy, and as maintenance therapy to prevent acne relapse. Moreover, clascoterone helped address other concerns in several patients, including hirsutism, hidradenitis suppurativa, retinoid-induced dermatitis, androgenetic alopecia, folliculitis, and laser-induced acne flares. This recent real-world clinical experience supports the effectiveness, tolerability, and versatility of clascoterone cream 1% for patients with acne vulgaris across a variety of clinical and demographic characteristics.

Introduction

Acne vulgaris primarily affects sebaceous regions such as the face, chest, and back, driven largely by androgens that stimulate sebum production and inflammation. Treatment strategies vary based on severity, with topical agents (i.e., retinoids, benzoyl peroxide, clindamycin) used for mild acne, and systemic therapies (i.e., oral antibiotics, antiandrogens, isotretinoin) for moderate-to-severe cases.[1] The challenges with side effects and tolerability underscore the need for safer and more effective alternatives.

Clascoterone cream 1%, a topical androgen receptor inhibitor, is a first-in-class therapy approved for acne vulgaris in male and female patients ≥12 years old. It was first approved in the US in 2020 and became available in Canada in June 2023.[2,3] In Phase 3 clinical trials for moderate-to-severe acne vulgaris, clascoterone cream 1% resulted in significantly higher treatment success rates and a greater reduction in lesion counts compared with the vehicle.[4] In this case series, we share our real-world Canadian experience with clascoterone cream 1% for managing acne vulgaris.

Materials and methods

Ten patients with acne vulgaris attending a private clinic in London, ON, Canada between August 2023 and May 2024 were included in this case series (**Table 1**). The information presented was obtained from a retrospective chart review of the patients' medical records. This case series did not require informed consent because it was a retrospective chart review with anonymized data.

Case 1

A 25-year-old African American female with hormonal acne, intolerant of oral contraceptives because of migraines, was initially treated with spironolactone 50 mg daily along with topical dapsone 5% and adapalene 0.3%/benzoyl peroxide 2.5%. After minimal improvement was observed, the spironolactone dose was increased to 100 mg daily which led to better control of her acne but was accompanied by bothersome side effects such as dizziness and nocturia. Introducing clascoterone cream 1% twice daily allowed for a dose reduction and gradual tapering of spironolactone over 6 months. Her acne remained well controlled with clascoterone monotherapy.

Case 2

A 34-year-old White female with polycystic ovary syndrome experienced persistent acne despite being treated with cyproterone acetate, clindamycin 1%/benzoyl peroxide gel 5%, and tretinoin gel microsphere 0.1%. The addition of clascoterone cream 1% twice daily to her existing topical regimen resulted in excellent control of her acne. The patient also reported an improvement in facial hirsutism, noting a decrease in the appearance of coarse dark hairs on her chin.

Case 3

A 47-year-old Hispanic female with metastatic breast cancer presented with concurrent acne and mild hidradenitis suppurativa (HS). The patient's condition was previously managed with oral doxycycline 100 mg daily, benzoyl peroxide 5% acne wash, and fusidic acid 2% cream, which yielded only partial improvement. Adalimumab was deemed unsuitable due to concerns regarding immunosuppression in the context of malignancy. She was advised to apply clascoterone cream 1% twice daily to both her facial acne and HS lesions. A remarkable resolution of both conditions was observed following the addition of clascoterone at the 1-year follow-up.

Case 4

A 21-year-old White transgender individual who recently underwent female-to-male gender reassignment surgery and was receiving testosterone therapy presented with acne breakouts. They were initially treated with minocycline, clindamycin 1%/benzoyl peroxide gel 5%, and a salicylic acid cleanser, which provided minimal improvement. They declined oral isotretinoin due to concerns regarding the potential side effects. Adding clascoterone cream 1% twice daily and tazarotene lotion 0.045% every other night to their doxycycline 100 mg

Case	Age (*years*)	Gender	Race or ethnicity	Acne severity	Clinical presentation, lesion subtypes	Relevant comorbidities	Concomitant treatments	Duration of clascoterone treatment* (*months*)
1	25	Female	African American	Moderate	Acne tarda Papules and nodules on lower cheek and jawline	n/a	Spironolactone, topical dapsone	6
2	34	Female	White	Mild	Acne tarda Papules and nodules on chin and jawline	PCOS, Hirsutism	Cyproterone acetate (for PCOS), topical antibiotics, BP, topical retinoids	11
3	47	Female	Hispanic	Mild-to-moderate	Papules, pustules, and comedones on cheeks	HS	Oral antibiotics, BP, fusidic acid cream	12
4	21	Transgender	White	Moderate-to-severe	Papules, pustules, nodules, and a few cysts	n/a	Topical retinoids	9
5	35	Male	Asian	Mild	Papules, pustules, and open and closed comedones	Androgenetic alopecia	Topical retinoids	9
6	14	Female	Middle Eastern	Mild	Papules and open and closed comedones on the nose and forehead	Excessive sebum production	Topical retinoids, salicylic acid, BP	7
7	18	Male	White	Severe	Nodulocystic acne and macular erythema on the face	Retinoid-induced dermatitis	Oral isotretinoin	6
8	22	Female	White	Severe	Nodulocystic acne on the face, shoulders, and upper back	n/a	Topical retinoids	11
9	31	Female	African American	Moderate	Papules and pustules on the shoulders and back, macular hyperpigmentation	Folliculitis, PIH	Fusidic acid cream, trifarotene, BP wash	12
10	18	Male	Asian	Moderate	Papules, pustules, comedones, and a few nodules	n/a	Laser therapy	6

Table 1. Demographics and clinical characteristics of patients included in the case series; *courtesy of Wei Jing Loo, BSc, MBBS, MRCP, FRCP.*

*All patients applied clascoterone cream 1% to acne-affected areas twice daily.

Abbreviations: BP: benzoyl peroxide, **HS:** hidradenitis suppurativa, **n/a:** not applicable, **PCOS:** polycystic ovary syndrome, **PIH:** postinflammatory hyperpigmentation

daily regimen resulted in marked improvement in their acne.

Case 5

A 35-year-old Asian male with mild facial acne was prescribed trifarotene cream 0.005%, which resulted in skin dryness and irritation. In response to these adverse effects, the frequency of trifarotene application was reduced to 3 times per week, and clascoterone cream 1% twice daily was added to the regimen. This adjustment led to effective control of his acne, along with resolution of dryness and irritation. He also applied clascoterone to his scalp for androgenetic alopecia without medical direction and reported stabilization of hair loss and some evidence of hair regrowth.

Case 6

A 14-year-old Middle Eastern female with acne vulgaris complained of "oily skin". Her skincare regimen included a 5% salicylic acid cleanser and adapalene 0.3%/benzoyl peroxide 2.5%. Despite these efforts, her acne persisted, and she continued to struggle with oily skin. The patient experienced a notable improvement in her acne and decreased sebum production following the addition of clascoterone cream 1%.

Case 7

An 18-year-old White male with severe nodulocystic acne was treated with isotretinoin 40 mg daily. He experienced prominent macular erythema, irritation, and dryness. The addition of clascoterone cream 1% twice daily alleviated the side effects associated with isotretinoin therapy. After completing a 6-month course of isotretinoin, he maintained clear skin with topical clascoterone monotherapy.

Case 8

A 22-year-old White female with a history of acne conglobata had failed to respond to oral contraceptive pills, systemic antibiotics, and various topical prescription creams. Following 6 months of treatment with isotretinoin 50 mg daily, she experienced substantial clearance of her acne lesions. However, due to concerns about potential relapse, she was hesitant to discontinue isotretinoin. To address her concerns, she was prescribed clascoterone cream 1% and tazarotene lotion 0.045%. Eleven months after discontinuing isotretinoin, her acne remained well controlled.

Case 9

A 31-year-old African American female presented with a combination of acne and folliculitis on her shoulders and back. She reported inadequate control of papules and pustules on her back despite using fusidic acid cream, trifarotene, and benzoyl peroxide wash. Adding clascoterone cream 1% to her existing regimen resulted in a marked improvement in her acne and folliculitis, as well as a notable reduction in macular hyperpigmentation.

Case 10

An 18-year-old Asian male with moderate acne vulgaris on his face underwent AviClear® laser therapy but experienced a severe acne flare, consistent with "purging," after the first treatment session. Adding clascoterone cream 1% to his regimen mitigated the initial flares, and subsequent sessions were better tolerated. After completing 3 sessions of laser therapy with adjunctive clascoterone cream 1%, he achieved clear skin.

Discussion

This study underscores clascoterone's efficacy and tolerability across diverse clinical presentations, genders, and ethnicities. It can complement both topical and systemic acne therapies, reduce side effects, and function as maintenance therapy to prevent relapse.

Acne is a common adverse effect observed in transgender individuals receiving masculinizing hormone therapy.[5,6] Although clinical trials for clascoterone did not specifically include transgender patients, the use of topical antiandrogens such as clascoterone appears to be a safe option due to its lack of systemic antiandrogen activity.[4,6,7]

The American Academy of Dermatology guidelines recommend a multimodal approach that

incorporates agents with multiple mechanisms of action to address the multifactorial pathogenesis of acne.[1] In clinical studies, clascoterone cream 1% was evaluated as a monotherapy.[4] However, limited data have been published on the efficacy and safety of concomitant usage of clascoterone cream 1% with other acne treatments.[8] The findings from this case series demonstrate that clascoterone may also work well as an adjunct to other topical and systemic therapies, as well as laser treatments, or as maintenance therapy to prevent relapse. The efficacy of clascoterone in preventing acne relapse following isotretinoin therapy underscores the importance of individualized maintenance therapy strategies for patients who have completed isotretinoin treatment, particularly those with a history of severe acne or concerns about relapse.

There is growing interest in the potential applications of clascoterone for other dermatological conditions.[9,13] Previous reports have shown that clascoterone cream 1% led to substantial reductions in the number and severity of lesions in patients with HS.[9,11] Clascoterone may also benefit patients with androgenetic alopecia by competitively inhibiting dihydrotestosterone, a known pathogenic trigger in androgenetic alopecia, based on evidence from Phase 1 and Phase 2 studies.[12,13] Phase 3 studies are currently recruiting participants to evaluate the efficacy and safety of clascoterone solution for androgenetic alopecia. **Cases 2**, **3**, **5**, **6**, and **9** highlighted the potential versatility of clascoterone cream 1% in addressing multiple dermatologic concerns including hirsutism, HS, retinoid-induced dermatitis, androgenetic alopecia, and folliculitis.

Conclusion

This case series presents Canadian real-world evidence demonstrating the efficacy, safety, and tolerability of clascoterone cream 1% for managing acne vulgaris across all patients, regardless of acne severity, age, gender, or ethnicity. In clinical practice, clascoterone is highly versatile for use as monotherapy, adjunctive therapy with other topicals, systemic agents, and laser devices, as well as maintenance therapy to prevent acne relapse.

As with any case study, the results should not be interpreted as a guarantee or warranty of similar results. Individual results may vary depending on the patient's circumstances and condition.

Patient data courtesy of Wei Jing Loo, BSc, MBBS, MRCP, FRCP.

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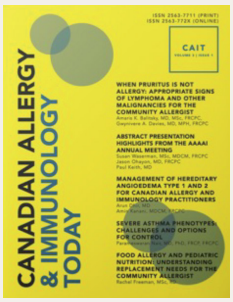

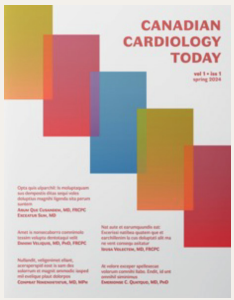

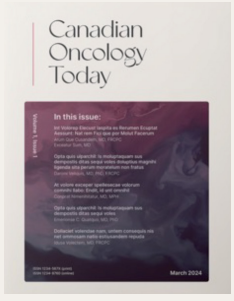

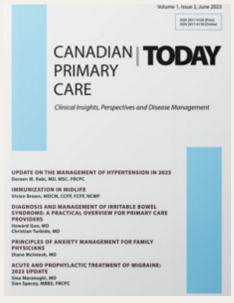

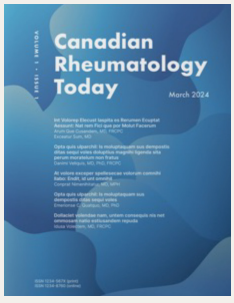

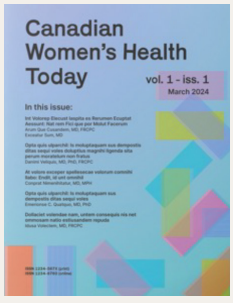

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Tattoo Regret? Principles and Pearls to Optimize Laser Tattoo Removal

Vincent Richer, MD

Introduction

Tattooing of the skin is an age-old practice that involves delivering pigments to the dermis.[1] Tattoos can be a mark of honour or status in specific cultures, a long-lasting reminder of a moment in time, or more simply a decorative feature of self-expression. Occasionally, tattoos can become painful reminders of moments past, such as a radiation tattoo years after remission from cancer, a lover's name from a failed relationship or a tattoo from a gang affiliation/imprisonment/human trafficking. On a more mundane note, a tattoo can become out of fashion or individual taste may change over the years. "Tattoo regret" is very common and many patients seek out options to remove tattoos. Though historically, surgical techniques were required to remove tattoos, pigment-targeting lasers have become the first-line treatment. Successful and safe laser tattoo removal requires a thorough understanding of treatment principles, appropriate laser wavelength selection, relevant biological endpoints, and prognostic factors that may guide the expected number of treatment sessions needed for a satisfactory outcome.

Process of Tattooing and Tattoo Composition

Tattoos are the result of ink insertion in the dermis, where it is subsequently taken up by macrophages, mast cells, and fibroblasts. Tattoos may be professional (tattoo artists using vibrating hollow needles), amateur (most often with a solid needle using the "stick-and-poke" technique), cosmetic (such as permanent makeup for eyebrows, eyeliner, or lip liner), traumatic (pencil or gravel, often in the context of an accident) or medical (for port placement or radiotherapy fields). Though historically, specific metals were used for their colour (such as cobalt for blue or mercury sulfide for red), nowadays most tattoos are made with carbon-based dyes such as azo dyes.

Scientific Principles Behind Laser Tattoo Removal

The principle of selective photothermolysis[2] explains and guides the process of laser tattoo removal. Tattoo pigment is the chromophore that absorbs laser photons, therefore, a wavelength that has affinity for a specific pigment must be selected (**Table 1**). Longer wavelengths have deeper optical penetration within the skin, allowing them to reach deeper pigment layers. Extreme care must be exercised when using shorter wavelengths, such as a quality-switched (QS) 532 nm laser, as penetration to the dermo-epidermal junction may target endogenous melanin in patients with Fitzpatrick IV–VI skin types and cause dyspigmentation. At a given wavelength, a larger spot size can increase dermal depth of penetration if needed. Lastly, because tattoo pigment is a very small structure with a short thermal relaxation time, the pulse duration of the device must also be very short (in the nanosecond or picosecond range) to produce thermal confinement and avoid collateral damage to the surrounding dermis. Picosecond lasers, with their extremely short pulses, are believed to exert their effects through acoustic (photomechanical, shattering the particle) effects, rather than heat (photothermal, heating the particle) effects. In general, millisecond pulse duration range pigment-targeting lasers and intense pulsed light are not considered appropriate to remove tattoos.

Pigment-targeting lasers are the most commonly used for tattoo removal. In certain scenarios (see accompanying patient photos), fractional ablative resurfacing or fully ablative resurfacing may be considered. These devices target water, and so are less specific for tattoo pigment and significantly increase the risk of side-effects such as prolonged healing, hypopigmentation, and scarring.

Predicting the Number of Treatments for Laser Tattoo Removal

The factors that affect the number of treatments required to minimize the appearance of a tattoo have been studied, and a scoring scale has been published. The Kirby-Desai scale[3] (**Table 2**) is particularly useful during clinical consultations, because it provides patients with an estimated number of treatments based on their individual situation. The tattoo removal process can be lengthy and costly, thus, patients appreciate knowing the expected number of treatment sessions before starting the process. Managing patient expectation is key; the author prefers to discuss the objective of making the tattoo unrecognizable rather than fully invisible, as full removal may not always be possible.

Let us apply a patient clinical scenario to the Kirby-Desai scale. A 27-year-old female patient of Middle-Eastern origin asks about the removal of a tattoo she got in university. You score her as Fitzpatrick III, locate the tattoo on the left lateral chest, determine the amount of ink is moderate, note the absence of layering or scarring, and confirm it is all black ink with magnification. After tallying her points, you discuss that she can expect 8 treatments to be satisfied with the appearance of the tattoo. The outcome of her treatments is shown in **Figure 1**.

Counselling Regarding Expected Recovery and Risks with Laser Tattoo Removal

After treatment, crusting/blistering is expected. A bland ointment, such as petrolatum jelly, can be applied to the area. A non-stick dressing will usually be applied. Depending on the site of the tattoo, recovery may take 1–3 weeks. Sun avoidance at the treatment site is recommended to minimize the risk of dyspigmentation. Both postinflammatory hyperpigmentation and hypopigmentation are possible complications of the procedure. Scarring, though uncommon, is possible. Patients can return for treatment as frequently as every 4–6 weeks, however longer intervals between treatments do not sacrifice their long-term outcomes if treatment pauses are needed. Some experts advocate even longer intervals between treatments, as ongoing improvement may be observed in some instances.

A thorough medical history is recommended prior to starting laser tattoo removal. Though less common nowadays, patients who have been treated with systemic gold at any point during

Tattoo Colour	1064 nm	755 nm	694 nm	532 nm	Other
Black	X	X	X		
Blue	X	X	X		
Green		X	X		
Purple		X	X		
Red, orange, yellow				X	
Brown, white					Risk of immediate pigment darkening: consider spot test, ablative laser or observing

Table 1. Laser wavelength selection for tattoo colours; *courtesy of Vincent Richer, MD.*

Points	FST	Location	Ink	Layering	Scarring	Colour
0				None	No scar	
1	I	Head/neck/face	Amateur		Minimal	Black only
2	II	Upper trunk/shoulder	Minimal	Layering		Mostly black, some red
3	III	Lower trunk/upper leg	Moderate		Moderate	Mostly black/red, other colours
4	IV	Proximal extremity	Significant			Multiple colours
5	V	Distal extremity			Significant	
6	VI					

Table 2. Kirby-Desai scale.[3]

Abbreviations: FST: Fitzpatrick Skin Type

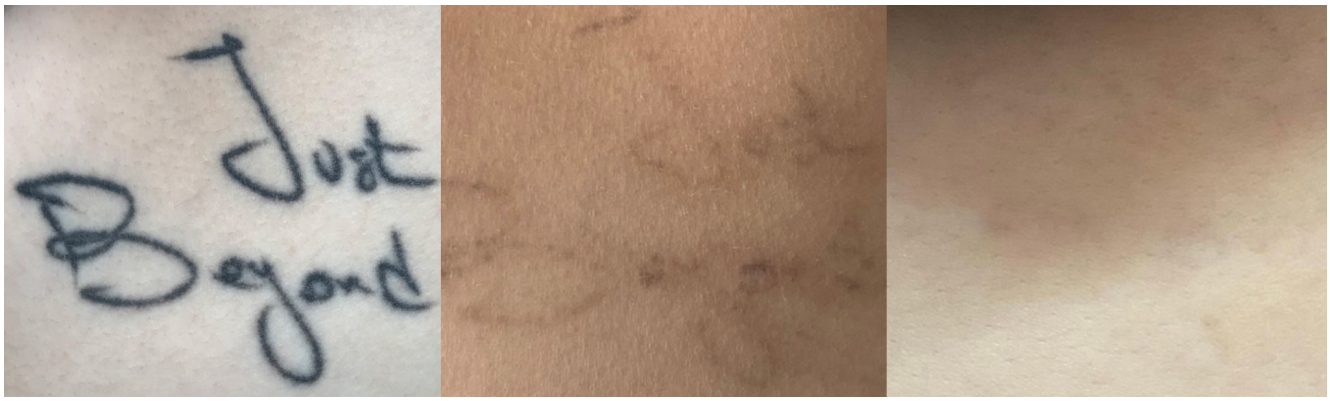

Figure 1. Black ink tattoo on the left chest of a Fitzpatrick III patient before treatment (**left**). Significant lightening was observed after 6 sessions of treatment with a Q-switched neodymium-doped yttrium aluminum garnet (NdYAG) laser and picosecond alexandrite laser (**centre**). The tattoo was rendered nearly invisible after 9 treatment sessions (**right**). Reaching this outcome required one extra treatment session beyond the Kirby-Desai scale estimation; *courtesy of Vincent Richer, MD.*

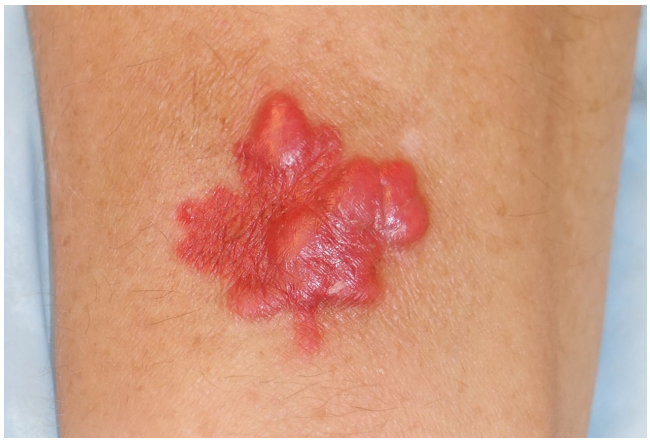

Figure 2. Biopsy-proven allergic contact dermatitis to red tattoo ink. This was associated with extreme pruritus. Intralesional triamcinolone acetonide/5-fluorouracil treated the itch and flattened the lesion, however recurrence occurred within weeks despite several treatments. Eventually ablative laser surgery was performed, as this patient preferred a scar over enduring the severe pruritus; *courtesy of Vincent Richer, MD.*

Figure 3. Blue-black eyeliner tattoo treated with 4 sessions of picosecond alexandrite laser. Metal corneal shields were placed prior to each treatment; *courtesy of Vincent Richer, MD.*

their lives are at risk of laser-induced chrysiasis[4] when using nanosecond or picosecond lasers. The blue-gray pigmentation that develops at sites of laser exposure in patients who have been treated with gold is very difficult to treat. This complication is best avoided by a thorough history and avoiding treatment with a pigment-targeting laser for these patients.

A thorough physical examination of the tattoo is also needed. Nanosecond and picosecond lasers are thought to "reduce" (the opposite of oxidize) ferrous oxide and zinc oxide particles, turning them black, in a process called immediate pigment darkening. This is of particular concern in scenarios of cosmetic tattooing, such as brown eyebrow liner or lip liner. The author unfortunately caused this complication in a patient who had cosmetic tattooing for solar lentigines years ago.[5] The skin-coloured tattoo was not noticed, and the patient did not recall getting a tattoo there until after the complication developed. This was later corrected using an ablative laser. It is prudent to perform a focal test spot prior to treating a brown or white tattoo with a laser.[6]

Photoallergic reactions (most commonly with yellow ink) or allergic contact dermatitis (most frequently seen with red ink) to tattoo ink are possible. These may be unmasked by the process of laser tattoo removal. In general, treating these reactions with nanosecond or picosecond lasers is not recommended, as it may lead to more systemic exposure and an associated systemic allergic contact dermatitis. Intralesional triamcinolone acetonide/5-fluorouracil or laser-assisted drug delivery of triamcinolone acetonide +/- 5-fluorouracil can be considered for this difficult-to-treat reaction.

Lastly, ensuring appropriate eye protection for the patient and laser operator is paramount when performing laser tattoo removal. This is a particularly sensitive matter when removing cosmetic tattoos, such as tattooed eyeliner (**Figure 3**). A metal corneal shield must be placed prior to using a device within the orbit to prevent injury to the iris or retina.[7]

Treatment Delivery and Techniques to Optimize the Laser Tattoo Removal Process

The skin should be cleansed with chlorhexidine prior to treatment. Laser exposure of tattoos is painful, therefore, anesthesia is recommended. In the author's practice, local anesthesia is injected prior to tattoo removal which far outperforms topical anesthesia or the use of cold air/ice.

Upon laser exposure, an immediate whitening reaction of the tattoo ink should be observed, and it should spare nearby healthy skin if it is partially within the beam's surface. This indicates selective lysosome cavitation under the skin, which dissipates within minutes. This biological endpoint allows for titration to a therapeutic fluence, without overtreating. Epidermolysis or immediate pinpoint bleeding may be signs that the fluence is too high.

Though picosecond lasers were developed with the exciting promise of speeding up the process of laser tattoo removal, they have not fully obviated the use of nanosecond-range lasers. Some studies have favoured picosecond lasers, others nanosecond lasers, and some have found no difference in clearance. It is likely that other factors, such as determining an appropriate biological endpoint and fluences used, affect the outcome significantly.

Combination treatments with multiple devices are common when treating tattoos, especially those with multiple colours requiring different laser wavelengths. Fractional ablative resurfacing at low density can also be performed at the same visit, which has the added benefit of reducing post-procedure blistering by ablating tiny perforations in the skin.[1]

Multi-pass treatment is another approach to accelerate the process of laser tattoo removal. Performing a second pass immediately after laser exposure is not usually effective, because the immediate whitening reaction forms an optical barrier to further treatment. The R20 method suggests waiting 20 minutes before retreating and repeating this process several times.[8] However, the practical implementation of this is difficult, due to the very long clinic visits required. In the author's practice, a truncated version

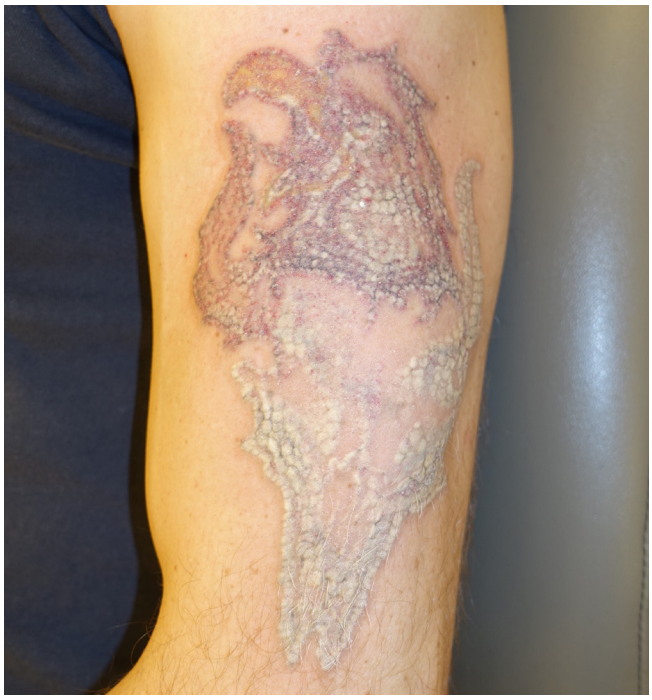

Figure 4. Immediate whitening reaction immediately after laser exposure. A nanosecond alexandrite laser was used to treat the blue and black ink, while a nanosecond 532 nm laser was used to treat yellow ink; *courtesy of Vincent Richer, MD.*

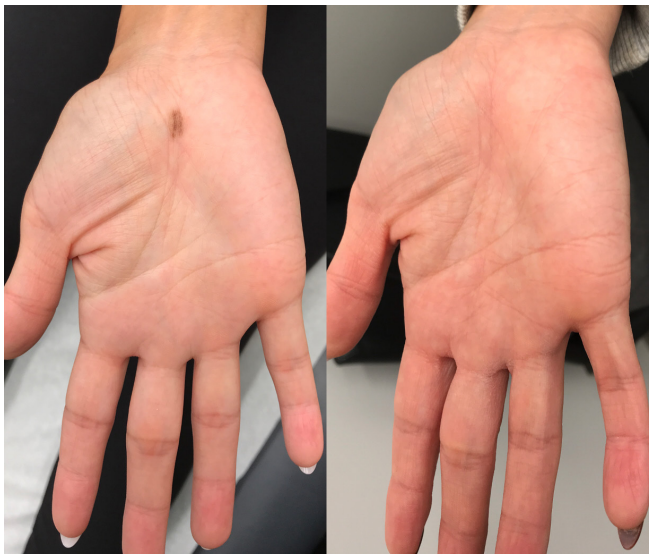

Figure 5. Traumatic tattoo on the right palm years after falling on an outstretched hand on gravel. One session of nanosecond NdYAG laser combined with a picosecond alexandrite laser yielded this improvement. In general, traumatic tattoos can be removed much more readily than professional tattoos; *courtesy of Vincent Richer, MD.*

where 5 minutes is lapsed before a second pass is performed has been helpful. A perfluorodecalin patch can also be applied to the skin prior to laser exposure.[9] This transparent patch reduces the whitening reaction and allows for faster retreatment. An adjunctive rapid acoustic pulse device has also been reported to accelerate clearance of tattoo ink.[10]

Conclusion

Laser tattoo removal can be a very rewarding process for patients and physicians alike (**Figure 5**), but expectations must be well managed. Conducting a thorough history and physical examination, understanding the laser physics, choosing the appropriate laser parameters, appropriately estimating the number of sessions to make the tattoo unrecognizable, and proactively avoiding complications can all lead to improved patient outcomes.

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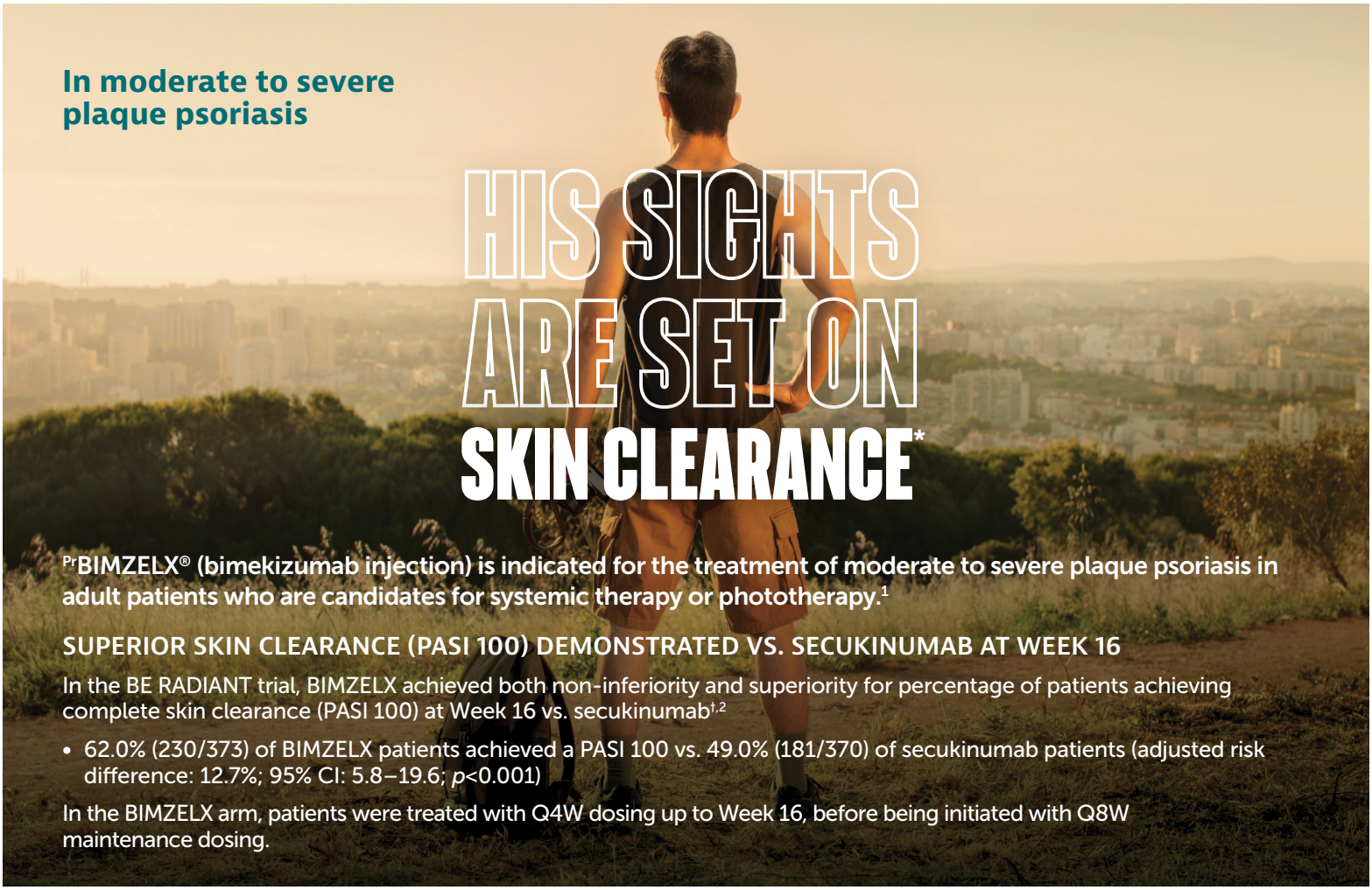

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Management of Androgenetic Alopecia in Men in 2025: A Focused Review

Hanieh Zargham, MD, FRCPC

Androgenetic alopecia (AGA) affects a significant portion of the male population, with studies estimating that approximately 50% of men will experience some degree of AGA by the age of 50.[1] This condition can lead to significant psychological distress and a reduced quality of life.

Recent advancements in understanding the multifactorial etiology of AGA have led to the development of new treatment strategies. This review provides an overview of the currently available treatments for AGA in men.

Topical Formulations

Topical Minoxidil

Topical minoxidil remains a cornerstone in the management of androgenetic alopecia (AGA). To date, topical minoxidil is the only Food and Drug Administration (FDA)-approved treatment for hair loss in both men and women. Minoxidil achieves its therapeutic effect through its vasodilatory, anti-inflammatory, and anti androgen properties, as well as by inducing the Wnt/B-catenin pathway.

When comparing its efficacy by concentration, 5% topical minoxidil applied twice daily was found to be superior compared to the application of 1%, 2%, and 10% solutions twice daily. When comparing the vehicles employed for delivery of topical minoxidil, the gel was found to be equivalent to the solution, while the foam resulted in significantly lower rates of local intolerance, such as pruritus and dandruff. This is likely due to the absence of propylene glycol in the foam formulation.[2-7]

Topical Finasteride

A recent randomized controlled trial examined the efficacy and safety profile of topical finasteride compared to both the oral version and a placebo. The study's findings demonstrated significantly increased hair densities after 24 weeks of 0.25% topical finasteride compared to the placebo, with no significant differences compared to 1 mg of oral finasteride. Moreover, topical finasteride had fewer treatment-related sexual adverse events than the oral formulation.[8]

Compounded Formulations

Many studies have also looked at compounded formulations that combine minoxidil with other ingredients thought to improve hair growth. The addition of 0.01% tretinoin or 1% pyrithione zinc shampoo did not show increased efficacy when compared to minoxidil monotherapy.[9,10] However, the combination of topical finasteride (0.25%) with minoxidil (5%) provided superior efficacy in treating AGA compared to using either topical minoxidil or topical finasteride alone.[11]

Topical Ketoconazole

In addition to its anti-fungal effects, ketoconazole has anti-inflammatory and anti-androgenetic effects which may help in the treatment of AGA. A systematic review looking at topical ketoconazole for the treatment of AGA identified 2 animal studies and 5 human studies, for a total of 318 participants. The murine studies demonstrated a significant increase in hair regrowth in the ketoconazole treatment groups compared to controls, while the human studies reported an increase in hair shaft diameter following ketoconazole use.[12] One study of 100 male patients with AGA included 4 treatment groups: **1)** 1 mg oral finasteride daily, **2)** 1 mg oral finasteride daily with minoxidil 2% solution twice daily, **3)** 2% minoxidil twice daily, and **4)** 1 mg oral finasteride daily with 2% ketoconazole shampoo 3 times weekly. Ten patients were treated with ketoconazole. The patients were assessed every 3 months for 1 year. The highest mean score of hair growth was observed when finasteride was combined with either minoxidil or ketoconazole, with no significant difference between these 2 groups. No significant side effects were reported. Although further studies are needed, overall, ketoconazole seems to be a low-risk addition to the treatment regimen for any patient with AGA.[13]

Oral Formulations

Oral Minoxidil

A 2022 *New York Times* article on oral minoxidil sparked significant interest in this treatment option. In fact, a study published in The Journal of the American Medical Association (JAMA) Network Open observed a notable rise in prescriptions for oral minoxidil, with a significant increase recorded 8 weeks after the article's release.[14]

A 2024 study published in JAMA Dermatology compared the effects of 5 mg oral minoxidil with 5% topical minoxidil applied twice daily for 24 weeks in 90 male participants with AGA. The study confirmed that oral minoxidil was not inferior to the topical solution, with both treatments showing a similar safety profile and well-tolerated side effects. While oral minoxidil showed a trend toward greater improvement, with photographic analysis indicating that it was superior to topical minoxidil on the vertex but not on the frontal scalp, the difference was not statistically significant, and superiority could not be established. The most common side effects in the oral minoxidil group were hypertrichosis (49% of patients) and headaches (14% of patients).[15]

Although rare, pericardial effusion has been identified as a potential side effect of oral minoxidil, regularly causing many patients to reconsider taking the medication. This condition is believed to be caused by fluid retention and altered hemodynamics, with the risk being higher at doses between 10–40 mg, particularly in patients with pre-existing cardiovascular conditions. However, a recent study published in the Journal of Drugs in Dermatology (JDD) involving 100 participants, 51 of whom were using low-dose oral minoxidil, found no significant difference in the prevalence of small, asymptomatic pericardial effusions compared to the control group. This finding is reassuring

that low-dose oral minoxidil has a low side effect profile.[16]

Sublingual minoxidil (SM) has emerged as an alternative to oral minoxidil. This formulation bypasses first-pass metabolism, potentially reducing systemic side effects, as hepatic sulfation enhances this drug's cardiovascular effects. Furthermore, SM might achieve therapeutic effects at lower doses compared to oral minoxidil. A Phase 1B trial evaluated the efficacy of 0.45, 1.35, and 4.05 mg daily doses of SM in 40 participants (male and female) with AGA over 24 weeks. The results showed a significant increase in hair density and terminal hair count in both the frontal and vertex scalp regions compared to placebo, with mild side effects such as dizziness and postural hypotension.[17] A more recent double-blind, randomized, clinical trial compared 5 mg of SM per day versus 5 mg of oral minoxidil daily for 24 weeks in 110 males with AGA. The study found that 5 mg of SM per day did not demonstrate superiority over 5 mg of oral minoxidil per day in treating male AGA. Both treatments were well tolerated, with less frequent palpitations in the SM group.[18]

Oral Dutasteride and Finasteride

The conversion of testosterone to dihydrotestosterone (DHT) by the enzyme 5-alpha-reductase plays a critical role in the development of AGA. Both dutasteride and finasteride function as anti androgens by blocking 5-alpha-reductase, however, dutasteride inhibits both the type I and type II isoforms, while finasteride targets only the type II isoform.

In 1997, finasteride received approval to treat male pattern hair loss at a reduced dose of 1 mg and continues to be the only FDA approved oral treatment for this condition. However, clinicians have been increasingly using oral dutasteride off-label for hair loss. Recently, Japan and South Korea have approved oral dutasteride (0.5 mg/day) for male AGA. Given its broader inhibition of both 5-alpha-reductase isoforms, some have suggested that dutasteride may be more effective than finasteride for treating hair loss.[19]

A meta-analysis evaluating the efficacy and safety of dutasteride and finasteride in treating men with AGA over a 24-week treatment cycle concluded that dutasteride seems to provide a better efficacy compared with finasteride in treating AGA and that the 2 drugs appear to show similar rates of adverse reactions, especially regarding sexual dysfunction.[20]

There is a theoretical concern that using 5-alpha-reductase inhibitors may hinder the early detection of prostate cancer, as these medications reduce the levels of prostate cancer markers. Therefore, the author recommends a baseline prostate specific antigen (PSA) level in patients prior to starting this class of medications. Moreover, the long term effects of anti-androgens in men, beyond sexual side effects, are poorly understood. Although there is a lack of strong evidence, many studies caution regarding risk of metabolic and bone health risks with long term use.[21]

In-office Physical Modalities

Platelet-Rich Plasma (PRP) Therapy

PRP therapy has gained popularity as a regenerative treatment for AGA. **Figure 1** shows a PRP system. The procedure involves collecting a patient's own blood, processing it to concentrate platelets, and injecting this plasma into the scalp. The platelets release growth factors and cytokines that promote cell proliferation, differentiation, and angiogenesis, which are essential for hair follicle regeneration. Furthermore, PRP contains insulin-like growth factor 1 (IGF-1), which can reduce the inhibitory effects of DHT on hair growth.

A systematic review and meta-analysis of 9 randomized controlled trials with 238 participants investigated the effects of PRP on hair density and hair diameter in AGA. The analysis found that PRP significantly increased hair density at 3 and 6 months compared to placebo injections ($P < 0.05$). However, while PRP also improved hair diameter compared to baseline, there were no significant differences compared to the placebo ($P > 0.05$). No serious side effects were observed.[22]

However, PRP monotherapy is likely not the most effective approach for managing AGA. A randomized, double-blind, placebo-controlled trial involving 80 male patients with AGA found

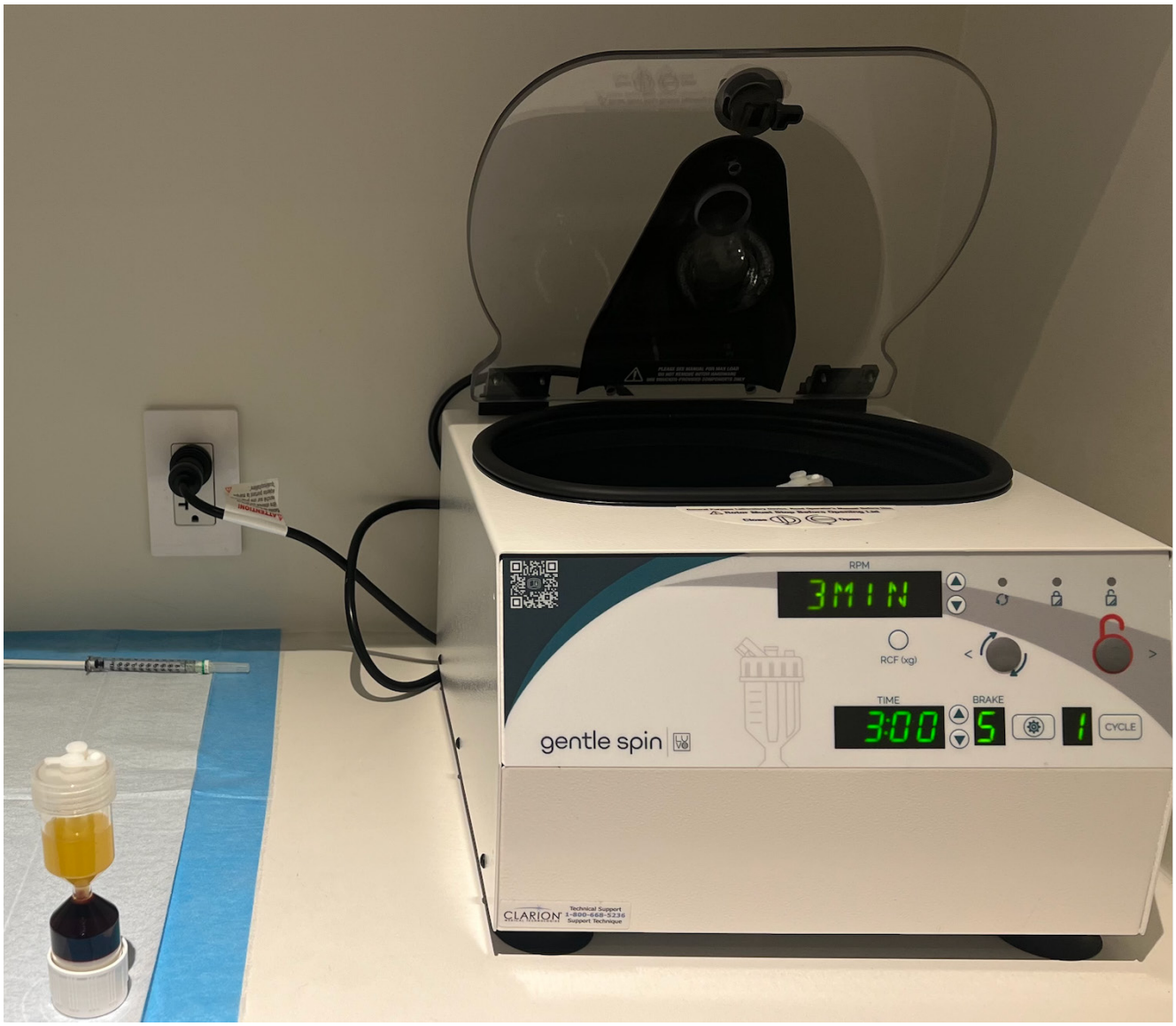

Figure 1. A platelet-rich plasma (PRP) therapy setup: Multiple systems are available. The author is currently using the WorldPRP© system by Clarion Medical Technologies, which yields approximately 6 mL of 3.5X concentrated PRP per 23 mL blood draw; *courtesy of Hanieh Zargham, MD, FRCPC.*

the greatest improvement in hair density when PRP was combined with twice-daily application of 5% topical minoxidil. This was followed by PRP alone, with minoxidil alone showing the least improvement.[23]

An issue regarding the use of PRP for AGA is the lack of a standard dose or protocol. Further studies and trials are needed to establish these parameters. Currently, PRP injections are typically administered once a month for 3–5 consecutive months, then every 4–6 months for ongoing maintenance to sustain results.

Dutasteride Mesotherapy

Dutasteride mesotherapy has also emerged as a potential treatment for AGA, offering an alternative to oral dutasteride or finasteride by delivering the drug directly into the scalp. This localized treatment approach enhances the efficacy of the drug while minimizing systemic side effects.

A multicenter retrospective study involving 541 patients with AGA assessed the safety and effectiveness of 0.01% dutasteride mesotherapy. Patients received intradermal injections of dutasteride every 3 months for a year. After 1 year, 38.4% of the 86 patients evaluated showed marked clinical improvement. The most common side effect was mild, transient pain at the injection site, with no serious or sexual side effects reported.[24]

A clinical trial involving 90 men with AGA compared the efficacy of 0.005% dutasteride alone, 0.05% dutasteride combined with dexpanthenol, biotin, and pyridoxine, and a control group that received physiological saline. The group treated with 0.05% dutasteride combined with the vitamin solution showed a significant increase in hair follicles in the anagen phase, although the contribution of the additional vitamins could not be excluded.[25]

Conclusion

Given the widespread prevalence of AGA and its significant impact on patients' quality of life, it is crucial for dermatologists to have access to a diverse range of effective, evidence-based treatment options. While this review provides an overview of various treatment modalities—ranging from topical and oral therapies to in-office physical procedures—it is not exhaustive. Although we currently have a variety of promising treatments, continued research is essential to refine existing therapies and expand our options for managing AGA.

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The initial request is authorized for a maximum period of four months. When requesting continuation of treatment, the prescriber must provide information making it possible to establish the beneficial effects of the treatment, specifically: An improvement of at least 75% in the PASI score compared to the baseline value; or an improvement of at least 50% in the PASI score and at least 50% in the body surface area affected, compared to the baseline values; or a significant improvement in lesions on the face, palms or soles or in the genital area compared to the pretreatment assessment.

Requests for continuation of treatment are authorized for a maximum period of 12 months.

Authorizations for tildrakizumab are given for 100 mg at weeks 0 and 4, then every 12 weeks thereafter.

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