

Oral Lichen Planus: Treatment Options

Estimates of the percentage of patients with cutaneous lichen planus (LP) who also have oral LP vary from somewhere between a third and a half¹⁻³, to as high as 70%⁴ and even higher when the cutaneous lesions are of long duration.⁴ Some 25¹–85%²⁻⁴ of patients present with only oral LP. Although about 65% of patients with cutaneous LP go into spontaneous remission after one year, such remissions have been estimated to occur in no more than 3% of patients with oral LP.⁵

The underlying mechanism causing LP is thought to be a T-cell mediated immune response against foreign or autogenous antigens.⁶ At least two thirds of the patients with LP are between the ages of 30–60 and the disease is uncommon in the very young and in the elderly.⁷ Oral lichen planus (LP), if erosive or disseminated can be very resistant to treatment. Oral LP has many clinical presentations, with some lesions requiring no treatment and others needing management for decades.

Treatment rationale

Topical corticosteroids should be considered the treatment of choice unless the disease is very extensive.^{1,2} Systemic therapy is reserved for those with severe, refractory disease.³

Oral hygiene¹⁻³ and corrective dentistry¹⁻⁴ play a major role in the management of LP and consultation with a dentist or oral medicine specialist may be helpful.⁶

Acitretin, combined with topical corticosteroid, can be effective, but should be reserved for patients who have not responded to corticosteroids alone. The retinoid should be

used for several months and then tapered as patients improve.³ If acitretin is ineffective, other agents such as antimalarials, azathioprine or cyclosporine¹ have been used.

Dental treatment

Indifferent oral hygiene leading to the formation of plaque and calculus exacerbates gingival LP, which may lead to severe gingivitis and periodontal disease.³ An optimal oral hygiene regimen should be instituted in all patients with oral LP, especially those with gingival involvement. Medical therapy should accompany oral hygiene measures.³ Certain oral clenching and sucking habits can make LP erosive or ulcerative, and habit splints have helped to modify these habits and reduce the inflammation.⁴ Oral trauma from ragged broken teeth and sharp prostheses are provocative.¹ There is some evidence that the presence of gold and mercury amalgam fillings may provoke oral lichenoid reactions. *Only a very small percentage of patients will respond to improved oral hygiene and corrective dentistry without further intervention.^{1,2}*

Lichen planus and hepatic disease

According to European reports hepatic disease does play a role in LP, its role seems to be less important in North America.^{1,2} Nevertheless, it is reasonable to obtain pertinent laboratory evidence on newly diagnosed patients, especially those with erosive disease.^{1,3}

Practice points

- 1% of patients with oral LP will develop oral squamous cell carcinoma.²
- The relative importance of reversible causes of lichenoid eruptions, such as exposure to causative drugs (most commonly diuretics and non-steroidal anti-inflammatory agents), or hypersensitivity reactions to dental restorations

has not been determined but a proper history should be obtained prior to instituting therapy.³

- Secondary candidiasis should be suspected when acute exacerbations develop in patients being treated with chronic topical or systemic steroids or other forms of immunosuppression.³
- There is increasing evidence that many women have concomitant lichen planus *vulvar involvement*, which either they are unaware of or decline to mention to their dermatologists. Female patients should be examined for vulvar involvement, or at least asked about symptoms.¹ Penile lesions are common.
- There are significant *histologic differences between idiopathic lichen planus and a lichenoid drug eruption*. It's important to do a baseline biopsy to distinguish between these two entities and to have these biopsies read by a dermatopathologist.

- Patients who consume *alcoholic beverages which contain flakes of gold* (Goldschlagger[®], Gold Rush[®], Gold Strike[®]) are at increased risk of developing generalized lichen planus. These drinks are more popular in Western Europe, especially with younger individuals, so in such patients inquiring about their patterns of alcohol consumption is prudent.¹

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Therapy for oral lichen planus⁵

First line	<i>topical corticosteroids</i>	Good safety & efficacy, low cost ⁴ used on almost all patients. ^{3,4}
	<i>topical retinoids</i>	Of value when combined with topical corticosteroids in conditions such as LP of the gingiva. ³
Second line	<i>acitretin</i>	May be first choice in severe, resistant disease. ⁸
Other	<i>dapsone, hydroxychloroquine</i>	No large, well designed trials. ⁴ Hydroxychloroquin is very effective when topical therapy fails but many months of treatment are required to realize its benefits. ³
	<i>oral corticosteroids and immunosuppressives</i>	Use oral corticosteroids with caution for a short term. Azathioprine has also been used as a steroid-sparing agent. Cyclosporin does not appear to be better than topical corticosteroids ⁴ and is very expensive. ^{3,4}
Investigational (results need confirmation and these two new treatment approaches need further study)	<i>Extracorporeal photochemotherapy</i>	All seven patients in an open, prospective trial had complete remission of their chronic, erosive, oral LP, after 12 sessions over 1.5 months on average. ⁹
	<i>Enoxaparin (a low molecular weight heparin)</i>	Low doses given to 10 patients with intensely pruritic LP produced complete remission of non-oral skin lesions in eight patients and marked improvement in one; oral lesions improved in one out of four patients with oral LP. ¹⁰

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Nail Psoriasis: Treatment Options

Approximately 50% of patients^{1,2} with psoriasis have nail psoriasis but the life time chance of nail changes must be much higher.³ Nail changes in psoriasis include pitting, thickening, onycholysis, discoloration, oily spots, splinter hemorrhages and paronychia.⁴ Treatment of choice depends on the form the psoriasis takes.⁵ Unfortunately, whatever the treatment used, failure and recurrence are common.⁶

Treatment options⁵

- *Moderate periungual psoriasis with nail ridging* will respond to potent topical corticosteroid applied to the affected nail under polyethylene occlusion, used in combination with practical measures to protect the hands such as gloves and avoidance of mechanical or chemical trauma. Steroid needs to be continued for several months and ideally is only stopped when the cuticle is reformed. The need for hand and nail protection has to be emphasized to the patient.⁵
- *Severe nail psoriasis* **Intralesional corticosteroid injection is still the longest lasting and most effective treatment when the nail dystrophy is of the appropriate form, the patient is well motivated and the clinician has the time and expertise to perform the procedure.**
- *Other treatments* Keeping the nails short and avoiding manicure will minimize the isomorphic response in the nail unit, where trauma can provoke psoriasis.⁴ Nail psoriasis may improve during effective therapy of psoriasis at other sites, particularly when systemic agents such as methotrexate and cyclosporine are used. Whole body⁷ and local⁸ photochemotherapy, and topical cyclosporin⁹ are reported to be useful. In acrodermatitis continua, topical 5-fluorouracil¹⁰ has been used and systemic retinoids are often of benefit in all types of psoriasis affecting the nail.¹¹

Local application of corticosteroid for psoriatic onycholysis

The management of psoriatic onycholysis is often exacerbated by trauma, either from leverage at the nail tip caused by long nails or by attempts to clean beneath the nail with a sharp instrument. Steroid injections are usually not successful in such cases. It is often better to use local therapy after cutting the nail back to the point of attachment with the nail bed. This may be to the level of the lunula and

requires a scalpel in a steady hand, but anesthetic is not usually needed. If the onycholysis is very extensive, it may be more satisfactory to call upon the services of a dermatologic surgeon. The exposed nail bed is very amenable to topical therapy with potent topical corticosteroid.⁵ On occasion it might be necessary to add an antimicrobial topical preparation (fusidic acid or mupirocin S. Maddin, Editor) if secondary invasion of the onycholytic space is suspected.⁶ The alternative treatment approach of advising the patient to apply corticosteroid solution between nail and nail bed has not proven to be successful.⁵

Intralesional injection of corticosteroid for severe nail psoriasis

The use of intralesional corticosteroids is the mainstay treatment for dystrophic changes in psoriatic nails.¹² William Gerstein of Montreal, while training with P. Samman in London, was the first to publish the results of steroid injected into the digit for nail psoriasis.¹³ Triamcinolone acetonide is the most readily available corticosteroid, the usual concentration is 2.5 mg/ml, or sometimes 5 mg/ml, injected into the proximal nail fold every three to four weeks for a total of four to six injections. It is practical to inject a volume of 0.1–0.2 ml into most sites. The injection can be given using a small syringe (such as an insulin syringe) with a 28–30 gauge needle firmly locked so that it won't disengage.⁵ Subsequent injections have been given when necessitated by the frequency and extent of recurrences.¹²

A practical new approach to treatment by Drs. de Berker and Lawrence requires the injection of 10mg/ml into both the nail fold and the nail bed at three monthly intervals.¹⁴ For more specific details of this technique, see de Berker DAR, Lawrence CM. A simplified protocol of steroid injection for psoriatic nail dystrophy. *Brit J Dermatol* 1998; 138: 90–95.¹⁴ Pitting, ridging and nail thickening (hyperkeratosis) require proximal injection of steroid. Pitting in isolation may respond to superficial proximal nail fold injection but the latter two benefit from deeper matrix injections. Nail bed hyperkeratosis improves most if the nail bed is injected, and this always requires local anaesthetic.⁵

Use of anesthesia

Injected steroid does not always require local anesthetic. The injection site should be matched with the focus of the pathology.^{5,15} Small injections given with a fine needle into

the proximal nail fold of non-tender fingers can be tolerated by most patients. It is very important not to push deeper and end up in the matrix, as this will cause much more pain and require anaesthetic.⁵ A proximal ring block is best, as when combined with corticosteroid, distal blocks end up producing a turgid finger tip, temporary tamponade and patients who are more likely to have paresthesia or pain.⁵ If the anesthetic is given proximally, there is seldom any subsequent pain.

To gauge response, in most instances the proximal nail fold is a good place to start. This is always the case for pitting, and often true when ridging and nail plate thickening are associated with inflammation of the proximal nail fold. More generalized dystrophy warrants deeper injection into the matrix, and nail bed injection is helpful for subungual hyperkeratosis and some forms of nail thickening.⁵

Response to intralesional injection

Even after injecting both the nail bed and the nail fold, improvement rather than complete resolution is the norm. In de Berker and Lawrence's study, subungual hyperkeratosis, ridging and thickening responded well, with benefit sustained for at least nine months; onycholysis and pitting, the most common signs, responded less well.¹⁴

Side effects of corticosteroid injection

Significant problems are rare. Injection into the matrix, as distinct from the proximal nail fold, often provokes a small subungual hemorrhage which is incorporated into the nail plate as it grows out. Although Port-o-jet and Dermojet jet guns have been used to deliver corticosteroid into the proximal nail fold, there are several reports of infection and nail plate damage associated with their use and there is the possibility of spreading infection between patient and doctor because of the blood and steroid that splashes back from the skin surface.⁵ Atrophy from steroid injection although uncommon, has been reported in patients being treated for nail psoriasis.^{5,13} The risk of damage to the extensor tendon of the interphalangeal joint remains theoretical.⁵

Summary

In treating nail psoriasis, excluding onycholysis and pustular forms, the proper use of intralesional corticosteroid provides a 60–80% chance of improvement, if the injections have been directed to the appropriate part of the nail apparatus. *This benefit will be sustained for up to nine months and sometimes longer.*

Drs. de Berker and Lawrence^{5,14}

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Dr. S. Maddin, Editor

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Nail Psoriasis: Response to intralesional injection of corticosteroid¹⁴

Nail Changes	Nail fold injection (from 3 published studies ¹⁴)	Nail fold & nail matrix injection ¹⁴
pitting and ridging	beneficial	beneficial
thickening	limited benefit in one study reporting	beneficial
hyperkeratinization	not reported	beneficial
onycholysis	beneficial	beneficial

Sunscreen Controversies

Sunscreens are often applied carelessly and in insufficient amounts.

Although sunscreens are in common use and widely promoted, their use is less than ideal even in situations of high sunburn risk.¹ Two recent studies using fluorescent markers^{2,3} confirm our suspicion that sunscreens are poorly applied. In both studies sunscreen was usually misapplied or incompletely applied, with the temples, ears and the back of the neck being most prone to neglect. *Liberal amounts of sunscreen need to be applied systematically to avoid missing areas.*^{2,3} *The fingertips should be used to cover areas with variable contours.*² Squeezing the sunscreen onto the palms and rubbing the hands together before beginning application leaves much of the screen on the hands.² Gaughan and Padilla², and Azurdia, Pagliaro and Rhodes³ suggest that sunscreens containing fluorescent dyes could be an effective teaching tool leading to significant improvement in sunscreen application^{2,3}, in the same way that teeth staining tablets are used to show faults in brushing technique.²

UV protection given by clothing varies

*Most people do not use sunscreens on the parts of their body covered by clothes, but are not aware that choice of fabrics and color of clothing affects the level of UV protection.*¹ A plain white, single thickness, cotton T shirt has a Sun Protection Factor (SPF) of only seven⁴, two layers 19, green cotton 10 and dark blue denim 1700. Stretch and wetness reduce a fabrics ultra-violet protective factor⁵ (UPF) markedly. A recent article reviews these and other factors affecting the sun-protective qualities of textiles and suggests standards for sun-protective clothing.⁶ Wearing a hat with a wide brim provides a high degree of protection to the scalp and hair as well as the upper face.

Sunscreens and the elderly The former concern that sunscreen use in the elderly might lead to vitamin D deficiency has been laid to rest to a certain extent⁷, but because of age related decrease in vitamin D production, some sun exposure for the elderly is probably to be recommended.⁸

Skin reactions to sunscreens

Although minor irritant reactions are common, *allergic reactions are rare and the incidence of such reactions, even in photomedicine centers, is significantly overstated.*⁹⁻¹¹

Sunscreens and photoaging

*There is substantial evidence that high SPF sunscreens with broad spectrum protection can reduce the stigmata of photoaging.*¹² Because UVA penetrates more deeply into the skin than UVB, a significant proportion of chronic photodamage may be secondary to UVA effects.¹²

Sunscreens and immunosuppression

*Immunosuppression caused by ultraviolet light appears to be a significantly underappreciated phenomenon. There is growing evidence that such immunosuppression is of biologic significance.*¹ Even suberythemal doses of UVB can induce immunosuppression.¹³

There is good evidence that appropriate broad spectrum sunscreens can prevent some aspects of photoaging and can prevent actinic keratosis and, perhaps by inference, squamous cell carcinoma. Sunscreens can prevent sunburn. There is no balance of evidence that would suggest that sunscreens directly prevent basal cell carcinoma or melanoma.¹

D.I. McLean and R. Gallagher¹

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Update on Drugs

Class	Name/Company	Approval Dates and Comments
Anti-HIV	Famciclovir Famvir® SmithKline Beecham	Approved by the US FDA June 1998 for the treatment of recurrent herpes simplex virus infections in HIV-infected patients. Famciclovir was previously approved for the treatment or suppression of recurrent genital herpes and the treatment of herpes zoster in immunocompetent patients.
Antiherpes	Acyclovir sodium Zovirax® Glaxo Wellcome	Approved by the US FDA June 1998 for the supplemental indication of treatment of herpes simplex virus infections in neonatal patients
Antipsoriatic	Calcipotriol Dovonex® Leo	Canadian approval was granted recently for the new indication of topical treatment of mild to moderate psoriasis in children aged two years and older.
Erythema nodosum leprosum	Thalidomide 50 mg Thalomid® Celgene	Approved by the US FDA July, 1998 to treat ENL. No one can prescribe it or take it without participating in a safety program. Women of childbearing age must take repeated pregnancy tests and use two forms of contraception during treatment. Recommended dosage is 100 to 300 mg/day once daily. Reviewed in <i>Skin Therapy Letter, Volume 3, Number 3</i> .
Male-pattern hair-loss	Finasteride 1mg tablets Propecia® Merck	Finasteride has been approved in Canada for the treatment of male-pattern hair loss. It was approved for this indication by the US FDA in December, 1997.
Aids vaccine to enter phase III clinical trial		
Aids vaccine Aidsvax® VaxGen Inc.	Given approval by the US FDA July, 1998 to undergo phase III evaluation for use in the prevention of AIDS. The trial will take three years to complete and will recruit 5000 US and 2500 Thai patients.	
Drug Warning		
Indinavir Crixivan® Merck	Four per cent of HIV-1 patients (42 affected) being treated with indinavir presented with paronychia of the great toes, suggesting a possible link between hypertrophic paronychia and treatment with protease inhibitors. Bouscarat F, Bouchard C, Bouhour D. <i>New Eng J Med</i> 1998; 338: 1998.	
Calcipotriene ointment is unstable with some other topical preparations		
Calcipotriene 0.005% Dovonex® ointment Bristol-Myers Squibb	Calcipotriene ointment degraded when mixed with hydrocortisone-17-valerate ointment 0.2%, ammonium lactate lotion and salicylic acid 6%, but was compatible with halobetasol proprionate ointment and cream 0.05% and a tar gel. Patel B, Siskin S, Krazmien R <i>et al.</i> Compatibility of calcipotriene with other topical medications. <i>J Am Acad Dermatol</i> 1998; 38: 1010-1.	

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