

# Lichen planus

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## INTRODUCTION

Lichen planus is an uncommon disorder of unknown cause that most commonly affects middle-aged adults. Lichen planus may affect the skin (cutaneous lichen planus), oral cavity (oral lichen planus), genitalia (penile or vulvar lichen planus), scalp (lichen planopilaris), nails, or esophagus.

The diagnosis and management of lichen planus, with a focus on cutaneous lichen planus, will be reviewed here. Oral lichen planus, vulvar lichen planus, lichen planopilaris, nail lichen planus, and lichenoid drug eruption (drug-induced lichen planus) are discussed in greater detail separately. (See ["Oral lichen planus: Pathogenesis, clinical features, and diagnosis"](#) and ["Oral lichen planus: Management and prognosis"](#) and ["Lichen planopilaris"](#) and ["Vulvar lichen planus"](#) and ["Overview of nail disorders"](#), section on 'Lichen planus' and ["Lichenoid drug eruption \(drug-induced lichen planus\)"](#).)

## EPIDEMIOLOGY

The epidemiology of lichen planus is not well-defined. Based upon limited data, cutaneous lichen planus is estimated to occur in less than 1 percent of the population [1].

Cutaneous lichen planus most frequently develops between the ages of 30 and 60 years [1-3]. Childhood cutaneous lichen planus occurs, but is uncommon [4]. There does not appear to be a strong sex or racial predilection for cutaneous lichen planus [1,2].

Epidemiologic data on other forms of lichen planus are reviewed separately. (See ["Oral lichen planus: Pathogenesis, clinical features, and diagnosis"](#), section on 'Epidemiology' and ["Vulvar lichen planus"](#), section on 'Epidemiology' and ["Lichen planopilaris"](#), section on 'Epidemiology' and ["Lichenoid drug eruption \(drug-induced lichen planus\)"](#), section on 'Epidemiology'.)

## ETIOLOGY

The etiology of lichen planus is not known. An immune-mediated mechanism involving activated T cells, particularly CD8+ T cells, directed against basal keratinocytes has been proposed [5].

Upregulation of intercellular adhesion molecule-1 (ICAM-1) and cytokines associated with a Th1 immune response, such as interferon (IFN)-gamma, tumor necrosis factor (TNF)-alpha, interleukin (IL)-1 alpha, IL-6, and IL-8, may also play a role in the pathogenesis of lichen planus [5-8].

**Hepatitis C virus** — The association of hepatitis C virus (HCV) with lichen planus is controversial, and a cause and effect relationship is uncertain. A meta-analysis of primarily case-control studies conducted in a number of countries found a statistically significant association between HCV and lichen planus. Compared with control patients, the prevalence of HCV exposure was greater among patients with lichen planus (OR 5.4, 95% CI 3.5-8.3) [9]. A systematic review also identified an increase in the proportion of lichen planus patients that were HCV positive compared with controls (OR 4.80, 95% CI 3.25-7.09) [10]. However, subgroup analysis in both studies revealed that the strength of this association varied geographically, and was not statistically significant in all locations. Estimates of the prevalence of HCV infection among patients with oral lichen planus vary widely; studies have reported prevalence rates from 0 to 62 percent [11].

Clinicians should have a high suspicion for lichen planus in patients with hepatitis C who present with clinical features suggestive of the diagnosis. The meta-analysis cited above found that patients with HCV had an increased prevalence of lichen planus compared to controls (OR 2.5, 95% CI, 2.0-3.1) [9]. In a study from Italy, among 178 adults with HCV antibodies, five (2.8 percent) had oral lichen planus [12]. There are also reports of the development or exacerbation of lichen planus during interferon treatment for chronic HCV; the lesions improved when interferon was stopped [13].

**Drugs** — Clinical manifestations that resemble idiopathic lichen planus can occur as a result of drug exposure ( [table 1](#)). Lichenoid drug eruptions (also known as drug-induced lichen planus) are reviewed separately. (See "[Lichenoid drug eruption \(drug-induced lichen planus\)](#)".)

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## CLINICAL FEATURES

Lichen planus may affect the skin, mucous membranes (especially the oral mucosa), scalp, nails, and genitalia [1].

**Cutaneous lichen planus** — The classic presentation of cutaneous lichen planus is a papulosquamous eruption characterized by the development of flat-topped, violaceous papules on the skin ( [picture 1A-D](#)). Often, the clinical manifestations are described as the four “P’s”:

- Pruritic
- Purple (actually a slight violaceous hue)
- Polygonal
- Papules or plaques

Individual papules are usually a few millimeters in diameter, but may coalesce to form larger plaques [2]. With close inspection, fine white lines may be visible on the surface of papules or plaques of cutaneous lichen planus ( [picture 2](#)). These lines are described by the term “Wickham’s striae” [14].

The extremities, particularly the ankles and the volar surface of the wrists, are common sites for cutaneous involvement [5]. Involvement of the trunk or generalized involvement also can occur ( [picture 3](#)). Rare Blaschkoid ( [picture 4](#) and [figure 1](#)) [15-17], zosteriform [18-21], and inverse (intertriginous) [2] distributions of cutaneous lichen planus have been observed.

Patients with lichen planus may exhibit the Koebner reaction (the development of skin lesions in sites of trauma). The Koebner reaction most often occurs as a result of scratching ( [picture 5](#)).

The pruritus associated with cutaneous lichen planus often is intense. Asymptomatic eruptions are rare. Lesions often heal with significant postinflammatory hyperpigmentation.

**Cutaneous variants** — In addition to the classic presentation of cutaneous lichen planus, multiple other clinical presentations of cutaneous disease have been described. Shared histologic findings support the classification of these disorders as variants of cutaneous lichen planus.

Examples of variants of cutaneous lichen planus include [2]:

- **Hypertrophic lichen planus** – Hypertrophic lichen planus is characterized by the development of intensely pruritic, flat-topped plaques ( [picture 6](#)). The typical site of involvement is the anterior lower legs. Of note, the occasional development of cutaneous squamous cell carcinoma has been reported in patients with longstanding hypertrophic lichen planus lesions [22].
- **Annular lichen planus** – Annular lichen planus is characterized by the development of violaceous plaques with central clearing ( [picture 7A-C](#)). Although the penis, scrotum, and intertriginous areas are common sites of involvement, annular lesions may occur in other areas [23]. Central atrophy may be present.
- **Bullous lichen planus** – Patients with bullous lichen planus develop vesicles or bullae within the sites of existing cutaneous lichen planus lesions. The legs are a common site of lesion development [2].
- **Actinic lichen planus** – Actinic lichen planus (also known as lichen planus tropicus) presents with a photodistributed eruption of hyperpigmented macules, annular papules, or plaques [2]. This variant is most commonly seen in the Middle East, India, and east Africa [5].
- **Lichen planus pigmentosus** – Lichen planus pigmentosus presents with gray-brown or dark brown macules or patches that are most commonly found in sun-exposed or flexural areas [24]. Pruritus is minimal or absent. The term “lichen planus pigmentosus-inversus” is used to describe patients with primarily flexural involvement [25].
- **Inverse lichen planus** – Inverse lichen planus is characterized by erythematous to violaceous papules and plaques in intertriginous sites, such as the axillae, inguinal creases, inframammary area, or limb flexures ( [picture 8](#)) [2]. Associated hyperpigmentation is common. Scale and erosions may be present.
- **Atrophic lichen planus** – Atrophic lichen planus presents with violaceous, round or oval, atrophic plaques. The legs are a common site of involvement [2], and lesions often clinically resemble

extragenital lichen sclerosus. A rare annular atrophic variant of lichen planus characterized by violaceous papules that enlarge peripherally leaving an atrophic center that demonstrates complete loss of elastic fibers on pathology has also been reported [26-28].

- **Lichen planopilaris (follicular lichen planus)** – The scalp is the classic site for lichen planopilaris. However, follicular involvement manifesting as follicular papules may be observed in other body sites, particularly in patients with the Graham-Little-Piccardi-Lasseur syndrome. (See "[Lichen planopilaris](#)", [section on 'Introduction'](#) and "[Lichen planopilaris](#)", [section on 'Graham-Little-Piccardi-Lasseur syndrome'](#).)

Additional variants include palmoplantar lichen planus (a variant that may demonstrate ulceration) [29-31] and perforating [32,33] lichen planus.

**Overlap syndromes** — Lichen planus pemphigoides and lichen planus-lupus erythematosus overlap syndrome are disorders that are characterized by the presence of features of cutaneous lichen planus and a second disease.

- **Lichen planus pemphigoides** – Lichen planus pemphigoides presents with overlapping features of lichen planus and bullous pemphigoid. The onset of lichen planus usually precedes the onset of bullous lesions [34].

Patients develop bullae in sites of previously normal appearing skin and on top of lesions of lichen planus [34]. This contrasts with bullous lichen planus, which presents with bullae that are limited to longstanding lichen planus lesions. Similar to bullous pemphigoid, direct immunofluorescence studies of lichen planus pemphigoides demonstrate linear deposition of IgG and C3 at the dermal-epidermal junction. (See "[Clinical features and diagnosis of bullous pemphigoid and mucous membrane pemphigoid](#)", [section on 'Direct immunofluorescence'](#).)

- **Lichen planus-lupus erythematosus overlap syndrome** – The term lichen planus-lupus erythematosus overlap syndrome refers to a rare condition in which patients develop skin lesions with clinical, histologic, and/or immunopathologic features of both diseases. Clinically, patients often present with blue-red atrophic plaques or upper extremity verrucous papules or nodules [35].

**Other forms of lichen planus** — Other manifestations of lichen planus may be present in patients with cutaneous lesions.

- **Nail lichen planus** – When nails are involved, the disease spectrum varies from minor dystrophy to total nail loss ( [picture 9A-D](#)). The disease process in lichen planus of the nails primarily occurs in the nail matrix. (See "[Overview of nail disorders](#)", [section on 'Lichen planus'](#).)
- **Lichen planopilaris** – Lichen planopilaris is the term used to describe lichen planus that occurs on the scalp. Patients present with areas of hair loss with keratotic follicular papules that, left untreated, can progress to scarring alopecia ( [picture 10](#)). Hair regrowth does not occur once follicles are destroyed. (See "[Lichen planopilaris](#)".)

- **Oral lichen planus** – Lichen planus of the mucous membranes can occur in conjunction with cutaneous disease or independently. Mucous membrane disease may consist solely of lacelike Wickham's striae that are particularly evident on the buccal mucosa or can include papular, atrophic, or erosive lesions ( [picture 11A-D](#)). Erosive mucous membrane disease is frequently painful and may lead to secondary complications including superficial candidal infection. Patients frequently report a persistent loss of appetite due to pain associated with eating. (See "[Oral lichen planus: Pathogenesis, clinical features, and diagnosis](#)", [section on 'Clinical manifestations'](#) and "[Vulvar lichen planus](#)", [section on 'Clinical manifestations'](#).)
- **Genital lichen planus** – Genital lichen planus in men presents with violaceous papules on the glans penis ( [picture 7B, 7D](#)), while in women lesions typically occur on the vulva. The vulvo-vaginal-gingival syndrome is an erosive form of lichen planus that involves the epithelium of the vulva, vestibule, vagina, and mouth; it is particularly resistant to treatment [36,37]. Although all three areas can be affected, the lesions may not be concurrent. The gingival epithelium is usually involved, but erosions, white plaques, or a whitish and lace-like reticular pattern may occur on the buccal mucosa, tongue, and palate ( [picture 12](#)). (See "[Vulvar lichen planus](#)".)
- **Esophageal lichen planus** – Lichen planus may involve the esophagus, presenting with or without symptoms such as dysphagia or odynophagia [38]. Possible endoscopic findings include pseudomembranes, friable and inflamed mucosa, submucosal papules, lacy white plaques, erosions, strictures, and other abnormalities ( [picture 13](#)). Concomitant oral, genital, or cutaneous lichen planus is frequently present [38]. The prevalence of esophageal lichen planus is unknown.
- **Otic lichen planus** – The stratified squamous epithelia of the external auditory canals and tympanic membranes are potential sites for lichen planus. Common clinical features of otic lichen planus include erythema, induration, and stenosis of the external auditory canal; thickening of the tympanic membranes; otorrhea; and hearing loss [39]. Lichen planus of other body sites may or may not be present. Further study is necessary to determine the prevalence of this disease manifestation.

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## HISTOPATHOLOGY

Lichen planus is associated with a set of characteristic pathologic findings that are seen in cutaneous lichen planus and to a variable extent in lichen planus of other body sites. These include ( [picture 14A-B](#)) [40,41]:

- Hyperkeratosis without parakeratosis
- Vacuolization of the basal layer
- Civatte bodies (apoptotic keratinocytes) in the lower epidermis
- Wedge-shaped hypergranulosis, "saw-tooth" shaped rete ridges

- Small clefts at the dermal-epidermal junction (Max-Joseph spaces)
- Band-like lymphocytic infiltrate at the dermal-epidermal junction
- Eosinophilic colloid bodies (apoptotic keratinocytes) in the papillary dermis
- Pigment incontinence (most prominent in dark-skinned individuals)

The pathologic findings of lichen planus in other body sites and lichenoid drug eruptions are reviewed separately. (See ["Oral lichen planus: Pathogenesis, clinical features, and diagnosis"](#), [section on 'Biopsy'](#) and ["Vulvar lichen planus"](#), [section on 'Diagnosis'](#) and ["Lichen planopilaris"](#), [section on 'Biopsy'](#) and ["Overview of nail disorders"](#), [section on 'Lichen planus'](#) and ["Lichenoid drug eruption \(drug-induced lichen planus\)"](#), [section on 'Pathology'](#).)

## DIAGNOSIS

In many cases, the diagnosis of cutaneous lichen planus can be made based upon the recognition of consistent clinical findings. In cases in which the diagnosis is uncertain, a skin biopsy is useful for confirming the diagnosis. The diagnosis of the other forms of lichen planus is reviewed in greater detail separately. (See ["Oral lichen planus: Pathogenesis, clinical features, and diagnosis"](#), [section on 'Diagnosis'](#) and ["Vulvar lichen planus"](#), [section on 'Diagnosis'](#) and ["Lichen planopilaris"](#), [section on 'Diagnosis'](#) and ["Lichenoid drug eruption \(drug-induced lichen planus\)"](#), [section on 'Diagnosis'](#).)

**Clinical evaluation** — The clinical evaluation of a patient with suspected cutaneous lichen planus consists of a patient interview and a physical examination. Patients should be questioned about medications (drugs that may induce lichen planus ( [table 1](#))), pruritus (a common symptom of cutaneous lichen planus), oral or genital erosions or pain (findings suggestive of concomitant mucosal lichen planus), and dysphagia or odynophagia (findings suggestive of esophageal disease). We also typically inquire about risk factors for hepatitis C. (See ['Additional testing'](#) below.)

The physical examination should include an examination of the entire cutaneous surface, including the scalp, as well as examination of the oral cavity and external genitalia. Performing a full examination aids in the assessment of the extent of cutaneous lesions and allows for the recognition of additional sites of involvement.

**Biopsy** — A skin biopsy can be used to confirm a diagnosis of cutaneous lichen planus. A punch biopsy or shave biopsy that reaches the depth of the mid-dermis is usually sufficient. (See ["Skin biopsy techniques"](#), [section on 'Punch biopsy'](#) and ["Skin biopsy techniques"](#), [section on 'Shave biopsy'](#).)

Immunofluorescence studies are not routinely needed. Direct immunofluorescence may be performed in patients with bullous lesions to differentiate the condition from an autoimmune blistering disease. If performed, direct immunofluorescence often demonstrates colloid bodies in the papillary dermis that stain for complement and immunoglobulins (especially IgM) and irregular fibrin deposition along the dermal-epidermal junction [\[40\]](#).

**Dermoscopy** — Wickham's striae often can be visualized during dermoscopic examination of cutaneous lesions of lichen planus [42-44]. (See "[Overview of dermoscopy](#)".)

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## ADDITIONAL TESTING

Given the evidence that suggests an association between lichen planus and hepatitis C virus (HCV) infection, we routinely test for HCV infection in patients with lichen planus. However, there are conflicting opinions regarding the value of such testing, and there is disagreement on whether routine testing is essential in all locations. (See '[Hepatitis C virus](#)' above and "[Screening and diagnosis of chronic hepatitis C virus infection](#)", section on 'Whom to test'.)

In a United States study in which 195 patients with oral lichen planus were prospectively screened for liver abnormalities, no liver abnormalities or antibodies to hepatitis B or C were detected [45]. This report suggested that routine testing for HCV infection is not necessary. Others have suggested testing for HCV infection in patients diagnosed with lichen planus, particularly those with risk factors for HCV infection, due to the risks of morbidity and transmission that exist with HCV infection and the evidence suggesting an association between lichen planus and HCV [9,10,46,47].

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## DIFFERENTIAL DIAGNOSIS

Key disorders in the differential diagnosis for classic presentations of cutaneous lichen planus include the following:

- **Lichenoid drug eruption** – Lichenoid drug eruptions (drug-induced lichen planus) should always be considered so that the offending agent can be withdrawn when possible ( [table 1](#)). The cutaneous manifestations closely resemble idiopathic lichen planus. The patient's history of drug exposure and a skin biopsy can aid in distinguishing lichenoid drug eruptions from idiopathic lichen planus. Lichenoid drug eruptions usually develop insidiously and can affect any area of the body surface ( [picture 15A-D](#)). (See "[Lichenoid drug eruption \(drug-induced lichen planus\)](#)".)
- **Chronic graft-versus-host disease** – Chronic graft-versus-host disease can produce a lichenoid eruption with clinical and histologic findings similar to lichen planus. The history of preceding hematopoietic cell transplant is helpful for diagnosis. (See "[Clinical manifestations, diagnosis, and grading of chronic graft-versus-host disease](#)", section on 'Mucocutaneous manifestations'.)

Other papulosquamous and dermatitic disorders, such as psoriasis, atopic dermatitis, lichen simplex chronicus, subacute cutaneous lupus erythematosus, discoid lupus erythematosus, pityriasis rosea, secondary syphilis, and prurigo nodularis can usually be distinguished from lichen planus through a careful evaluation for clinical findings that are more consistent with these disorders. Lesions suspicious for bullous lichen planus should be distinguished from autoimmune blistering diseases through a biopsy and immunofluorescence studies. Of note, skin involvement in paraneoplastic pemphigus/paraneoplastic autoimmune multiorgan syndrome (PAMS) often presents as lichenoid skin lesions. (See "[Approach to the patient with cutaneous blisters](#)", section on 'Diagnostic tests' and "[Paraneoplastic pemphigus](#)".)

## NATURAL HISTORY

The natural history of most cases of cutaneous lichen planus is to remit within one to two years [48]. Oral, genital, scalp, and nail lichen planus tend to be more persistent. The clinical course of these types of lichen planus and lichenoid drug eruptions are viewed in greater detail separately. (See "[Oral lichen planus: Management and prognosis](#)", section on 'Prognosis and follow-up' and "[Vulvar lichen planus](#)", section on 'Natural history' and "[Lichen planopilaris](#)", section on 'Clinical course' and "[Overview of nail disorders](#)", section on 'Lichen planus' and "[Lichenoid drug eruption \(drug-induced lichen planus\)](#)", section on 'Clinical course'.)

**Comorbid disease** — The results of two case-control studies suggest that the prevalence of dyslipidemia may be greater among patients with lichen planus than among individuals without this disease [49,50]. Additional studies are necessary to confirm an association between dyslipidemia and lichen planus and to explore the clinical relevance of this finding. Further study is also necessary to clarify whether there is an association between oral lichen planus and thyroid disease [51,52].

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## TREATMENT

There are few data to support evidence-based recommendations for the treatment of lichen planus [53-55]. Only a few randomized trials have been performed, most of which were small and subject to methodologic error [56].

The treatment of cutaneous lichen planus is reviewed below. Detailed information on the management of other forms of lichen planus can be found separately. (See "[Oral lichen planus: Management and prognosis](#)" and "[Vulvar lichen planus](#)", section on 'Management' and "[Lichen planopilaris](#)", section on 'Treatment' and "[Overview of nail disorders](#)", section on 'Lichen planus' and "[Lichenoid drug eruption \(drug-induced lichen planus\)](#)", section on 'Treatment'.)

**Cutaneous lichen planus** — Cutaneous lichen planus usually is a self-limited disorder. Thus, treatment is focused on accelerating resolution and managing pruritus [1]. (See '[Natural history](#)' above.)

**First-line therapy** — Topical corticosteroids are commonly used as first-line treatment for localized cutaneous lichen planus. For patients with generalized disease, in whom monotherapy with topical corticosteroids is less practical, topical corticosteroids are often used as an adjunct to systemic therapy or phototherapy. (See '[Second-line therapy](#)' below.)

**Topical corticosteroids** — Although topical corticosteroids are the mainstay of treatment for patients with localized cutaneous lichen planus, the efficacy of these agents has not been evaluated in clinical studies. Recommendations based upon clinical experience and the relative safety of this mode of treatment support the use of topical corticosteroids as first-line therapy [1,5].

We typically treat localized cutaneous lichen planus on the trunk and extremities with a high potency or super high potency topical corticosteroid (eg, 0.05% [betamethasone](#) dipropionate, 0.05%



[diflorasone](#) diacetate) cream or ointment twice daily ( [table 2](#)). Because topical corticosteroid-induced cutaneous atrophy is most likely to occur in intertriginous or facial skin, we prefer to use mid-potency or low-potency corticosteroid creams or ointments when treating these areas. Efficacy should be assessed after two to three weeks.

Patients should be cautioned about the risk for cutaneous atrophy and should be followed closely for this side effect. The adverse effects of topical corticosteroid therapy are reviewed separately. (See "[Topical corticosteroids: Use and adverse effects](#)".)

**Intralesional corticosteroids** — The thick lesions of hypertrophic lichen planus may be less likely than classic cutaneous lesions to respond well to a topical corticosteroid. Clinical experience suggests that intralesional corticosteroid therapy can be beneficial for the treatment of hypertrophic lichen planus [5].

We typically administer [triamcinolone](#) acetonide in a concentration of 2.5 to 10 mg/mL. The quantity administered should blanch or at least infiltrate hypertrophic lesions. Extension of the corticosteroid into the surrounding normal skin should be minimized. We limit the total dose of triamcinolone to no more than 40 mg per treatment session. Injections may be repeated after four to six weeks.

Cutaneous atrophy and hypopigmentation may occur as a result of intralesional corticosteroid therapy. Side effects of this treatment are reviewed separately. (See "[Intralesional corticosteroid injection](#)", [section on 'Side effects, complications, and pitfalls'](#).)

**Second-line therapy** — Patients with cutaneous lichen planus who cannot be treated adequately with local corticosteroids (eg, generalized disease or local corticosteroid-refractory disease) may benefit from other treatments, such as oral glucocorticoids, phototherapy, and oral [acitretin](#). Only acitretin has been evaluated in a blinded placebo-controlled randomized trial. The limited availability of data and the potential for lichen planus to spontaneously resolve contributes to uncertainty about the efficacy of treatments. The risks and benefits of treatment should be considered carefully prior to treatment.

**Systemic glucocorticoids** — Oral glucocorticoid therapy may be beneficial for cutaneous lichen planus based upon clinical experience and a few small uncontrolled studies in which improvements in the signs or symptoms of cutaneous lichen planus occurred during oral glucocorticoid therapy [53,57,58]. We consider prescribing a short course of an oral glucocorticoid when acute control of cutaneous lichen planus is necessary for patients with extensive disease. Continued, long-term treatment with systemic glucocorticoids is less favorable due to the serious side effects associated with long-term therapy. (See "[Major side effects of systemic glucocorticoids](#)".)

The optimal dose and duration of systemic glucocorticoid therapy is not known. Our preference is to begin with 30 to 60 mg daily for four to six weeks followed by a taper of the dose to discontinuation over the next four to six weeks. Lower doses and shorter courses have also been proposed in the literature [53].

**Phototherapy** — Ultraviolet B (UVB) and psoralen plus ultraviolet A (PUVA) phototherapy are utilized for the treatment of cutaneous lichen planus. Narrowband UVB is the most common modality

used [3,5]. Similar to other treatments for cutaneous lichen planus, data on the efficacy of phototherapy are limited. Placebo-controlled randomized trials have not been performed. (See "[UVB therapy \(broadband and narrowband\)](#)" and "[Psoralen plus ultraviolet A \(PUVA\) photochemotherapy](#)".)

Non-placebo-controlled studies evaluating narrowband UVB for cutaneous lichen planus have generally yielded favorable findings. A small, unblinded, randomized trial of 46 patients with generalized lichen planus that compared treatment with narrowband UVB (three times per week for six weeks) to oral [prednisone](#) (0.3 mg/kg per day for six weeks) found a significantly better response in the patients who received phototherapy [59]. Complete responses were attained by 12 of 23 patients in the phototherapy group (52 percent) versus 3 of 23 patients in the prednisone group (13 percent).

Several other studies suggest benefit of UVB phototherapy [60-64]. As an example, a prospective uncontrolled study of 16 patients with generalized cutaneous lichen planus found that 11 patients achieved at least a 90 percent reduction in papules after treatment with narrowband UVB (three times weekly for 40 sessions). Itching tended to improve early in the course of therapy. Additionally, a retrospective study in which 50 patients with generalized cutaneous lichen planus were treated with either narrowband UVB (n = 43) or broadband UVB (n = 7) three times per week found documentation of complete remissions in 70 percent of patients [61]. Among those who achieved a complete remission, 85 percent remained in remission after a median follow-up period of 34.7 months.

PUVA photochemotherapy, which requires oral or bath administration of 8-methoxypsoralen, also seems to be efficacious for some patients with cutaneous lichen planus based upon retrospective studies and case series [65-70]. Although the findings of a retrospective analysis of 28 patients with generalized lichen planus raised the question of whether PUVA is more likely than narrowband UVB to induce an initial response [65], the study failed to find a significant difference in the long term benefit of these treatments. Additional studies are needed to explore the comparative efficacy of PUVA and narrowband UVB phototherapy.

Given the lack of data confirming superiority of a particular mode of phototherapy, narrowband UVB therapy (initially given three times per week) is our preferred mode of phototherapy for cutaneous lichen planus due to the ease with which this treatment can be administered. In contrast to narrowband UVB, PUVA requires administration of an oral or topical photosensitizer. Additionally, oral PUVA requires a period of ocular photoprotection following treatment. We taper the frequency of narrowband UVB treatment once an adequate response is achieved. If no response is observed after three to four months, we discontinue treatment.

Burning, blistering, and pruritus are potential side effects of phototherapy. Short- and long-term side effects of phototherapy are reviewed in detail separately. (See "[UVB therapy \(broadband and narrowband\)](#)", [section on 'Short- and long-term adverse effects'](#) and "[Psoralen plus ultraviolet A \(PUVA\) photochemotherapy](#)", [section on 'Adverse effects'](#).)

**Oral retinoids** — The best support for the use of [acitretin](#), an oral retinoid, for lichen planus stems from a placebo-controlled randomized trial of 65 patients with cutaneous lichen planus [71,72]. At the end of eight weeks, patients treated with 30 mg per day of acitretin (n = 32) were significantly more likely to have achieved remission or marked improvement than the 33 patients in the placebo

group (64 versus 13 percent achieved this level of response). Improvement during treatment with acitretin and other oral retinoids has also been documented in small, open-label studies or case reports [73-75].

Although the randomized trial strongly supports a beneficial effect of [acitretin](#) on cutaneous lichen planus, the potential adverse effects of acitretin account for our categorization of acitretin as a second-line treatment rather than a first-line treatment for cutaneous lichen planus. Xerosis, hair loss, hypertriglyceridemia, visual changes, myalgias, pseudotumor cerebri, and skeletal abnormalities are some examples of retinoid side effects. We primarily reserve the use of acitretin for patients with disease that cannot be managed with local corticosteroids or phototherapy.

Oral retinoids should be administered by or in consultation with clinicians familiar with the use and side effects of this therapy (eg, a dermatologist). [Acitretin](#) is teratogenic, and is contraindicated for women who are pregnant or intend to become pregnant. Pregnancy is contraindicated for three years after discontinuation of acitretin.

**Other treatments** — Oral antihistamines (eg, [hydroxyzine](#) hydrochloride 10 to 50 mg four times a day, as necessary) may be helpful in controlling pruritus. Other medications that have been reported to be of benefit in some patients with cutaneous lichen planus include [methotrexate](#) [56], [thalidomide](#) [76,77], low molecular weight heparin [78,79], [griseofulvin](#) [80], [cyclosporine](#) [53], [dapsons](#) [4,81], [sulfasalazine](#) [82], [metronidazole](#) [83,84], and [hydroxychloroquine](#) [85]. [Mycophenolate](#) mofetil has been used successfully in the treatment of at least two patients with extensive generalized lichen planus and one patient with resistant hypertrophic and bullous lichen planus [86,87]. Further study is necessary to confirm the efficacy of all of these agents.

The results of a small uncontrolled study suggest that [apremilast](#), a phosphodiesterase inhibitor, may be beneficial for lichen planus [88]. Treatment with apremilast (20 mg twice daily for 12 weeks) was associated with clinical improvement in all 10 patients, including three patients who achieved at least a two-grade improvement in the physician global assessment score. Additional studies are necessary to explore the efficacy and safety of apremilast for lichen planus.

## Other types of lichen planus

**Genital lichen planus** — Topical corticosteroids or topical calcineurin inhibitors are often used in the treatment of genital lichen planus. The treatment of vulvar lichen planus is reviewed in detail elsewhere. (See "[Vulvar lichen planus](#)".)

**Lichen planopilaris** — The treatment of lichen planopilaris (LPP) can be difficult. Topical corticosteroids or intralesional corticosteroids are often used as first-line therapies ( [table 2](#)) [89-92]. The treatment of LPP is discussed in greater detail separately. (See "[Lichen planopilaris](#)", [section on 'Treatment'](#).)

**Oral lichen planus** — Topical corticosteroids are first-line therapy for oral lichen planus. The treatment of oral lichen planus is reviewed separately. (See "[Oral lichen planus: Management and prognosis](#)".)

**Nail lichen planus** — The treatment of lichen planus involving the nails is reviewed separately. (See ["Overview of nail disorders", section on 'Lichen planus'.](#))

**Lichenoid drug eruption** — The management of drug-induced lichen planus is reviewed separately. (See ["Lichenoid drug eruption \(drug-induced lichen planus\)", section on 'Treatment'.](#))

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## SOCIETY GUIDELINE LINKS

Links to society and government-sponsored guidelines from selected countries and regions around the world are provided separately. (See ["Society guideline links: Lichen planus"](#).)

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## INFORMATION FOR PATIENTS

UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5<sup>th</sup> to 6<sup>th</sup> grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10<sup>th</sup> to 12<sup>th</sup> grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

- Basics topic (see ["Patient education: Lichen planus \(The Basics\)"](#))

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## SUMMARY AND RECOMMENDATIONS

- Lichen planus is a mucocutaneous disorder that most commonly affects middle-aged adults. Clinicians should be aware of the potential for drug-induced forms of lichen planus. (See ['Introduction'](#) above and ['Etiology'](#) above.)
- Studies have suggested an association of lichen planus with hepatitis C virus (HCV), although the strength of this association varies geographically. Further studies are necessary to determine if HCV screening should be performed in patients with lichen planus. (See ['Hepatitis C virus'](#) above.)
- Lichen planus may affect the skin, nails, or mucous membranes. Cutaneous lichen planus typically presents as pruritic, polygonal, violaceous papules and/or plaques with an overlying white, lacelike pattern (Wickham's striae) ( [picture 1A-D](#)). Significant post-inflammatory hyperpigmentation is common. Cutaneous lichen planus may also present with hypertrophic or vesicobullous lesions. (See ['Clinical features'](#) above.)

- Lichen planopilaris is a form of lichen planus that affects the scalp and can lead to scarring alopecia. When lichen planus involves the oral cavity, it may present with only lacelike Wickham's striae or may include papular, atrophic, or erosive lesions. Genital areas can also be affected by lichen planus. (See '[Clinical features](#)' above and "[Lichen planopilaris](#)" and "[Oral lichen planus: Pathogenesis, clinical features, and diagnosis](#)" and "[Vulvar lichen planus](#)".)
- Cutaneous lichen planus often spontaneously resolves after one to two years. Oral, genital, scalp, and nail lichen planus tend to be more chronic. (See '[Natural history](#)' above.)
- We suggest high potency or super high potency topical corticosteroids as initial treatment of localized cutaneous lichen planus on the trunk or extremities based upon clinical experience and the relative safety of this therapy ([Grade 2C](#)). Intralesional corticosteroids can be useful in patients with hypertrophic lichen planus. Patients with widespread cutaneous disease may benefit from phototherapy, [acitretin](#), or a short course of systemic glucocorticoid therapy. (See '[Treatment](#)' above.)

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## ACKNOWLEDGMENT

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**Drugs causing lichenoid eruptions (drug-induced lichen planus)**

<b>Group of drug</b>	
Antimicrobial substances	Aminosalicylate sodium, ethambutol, griseofulvin, ketoconazole, streptomycin, tetracycline, trovafloxacin, isoniazid
Antihistamines (H <sub>2</sub> -blocker)	Ranitidine*, roxatidine
Antihypertensives/antiarrhythmics	<b>ACE inhibitors</b> (captopril, enalapril), doxazosin, <b>beta blockers</b> (propranolol, labetalol, sotalol), <b>methyldopa</b> , prazosin, nifedipine, <b>quinidine</b>
Antimalarial drugs	<b>Chloroquine</b> , <b>hydroxychloroquine</b> , quinine
Antidepressives/antianxiety drugs/antipsychotics/anticonvulsants	Amitriptyline, carbamazepine, chlorpromazine, levomepromazine, methopromazine, imipramine, lorazepam, phenytoin
Diuretics	<b>Thiazide diuretics</b> (chlorothiazide and hydrochlorothiazide), furosemide, spironolactone
Antidiabetics	Sulfonylureas (chlorpropamide, glimepiride, tolazamide, tolbutamide, glyburide)
Metals	<b>Gold salts</b> , arsenic, bismuth, mercury, palladium, lithium
Nonsteroidal anti-inflammatory drugs	Acetylsalicylic acid, benoxaprofen, diflunisal, fenclofenac, flurbiprofen, ibuprofen, indomethacin, naproxen, sulindac
Proton pump inhibitors	Omeprazole, lansoprazole, pantoprazole
Lipid lowering drugs	Pravastatin, simvastatin, gemfibrozil
Tumor necrosis factor-alpha antagonists	Infliximab, adalimumab, etanercept, lenercept
Checkpoint inhibitors	Nivolumab, pembrolizumab, atezolizumab, ipilimumab
Miscellanea	Allopurinol, bleomycin, cinnarizine, cyanamide, dapsone, hydroxyurea, hepatitis B vaccine, imatinib, immunoglobulins, interferon alfa, l-thyroxin, levamisole, mesalamine, methycran, <b>penicillamine</b> , procainamide, pyrimethamine, pyriethoxine, <b>quinacrine</b> , sildenafil, sulfasalazine, terbinafine, trihexyphenidyl, ursodeoxycholic acid

The **bolded** drugs are the ones most frequently implicated.

\* Ranitidine has been withdrawn from the United States market.

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*Original figure modified for this publication. Shiohara T, Kano Y. Lichen planus and lichenoid dermatoses. In: Dermatology, 3rd Edition, Bologna JL, Jorizzo JL, Schaffer JV (Eds), Elsevier, London 2012. Table used with the permission of Elsevier Inc. All rights reserved.*

## Lichen planus



Violaceous and hyperpigmented, polygonal papules are present on ankles and ventral wrists.

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Graphic 63590 Version 6.0

## Lichen planus



Violaceous, polygonal papules are present on the ventral wrists.

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Graphic 76383 Version 6.0

## Lichen planus

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Note discrete polygonal scaling erythematous papules on the wrist.

*Courtesy of Beth G Goldstein, MD and Adam O Goldstein, MD.*

Graphic 50579 Version 1.0

## Lichen planus



Lichen planus. Flat-topped, violaceous, polygonal papules on the flexor wrists are present. There are active and resolving lesions. Note the postinflammatory hyperpigmentation.

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Graphic 53839 Version 2.0

## Wickham striae

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Note the thick, violaceous, hyperkeratotic plaque with a white, lacelike pattern on the surface (Wickham striae).

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*Courtesy of Beth G Goldstein, MD, and Adam O Goldstein, MD.*

Graphic 61523 Version 4.0



## Generalized lichen planus

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Violaceous and hyperpigmented polygonal papules are present in a widespread distribution in this patient with generalized lichen planus.

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Graphic 50632 Version 1.0

## Lichen planus following the lines of Blaschko



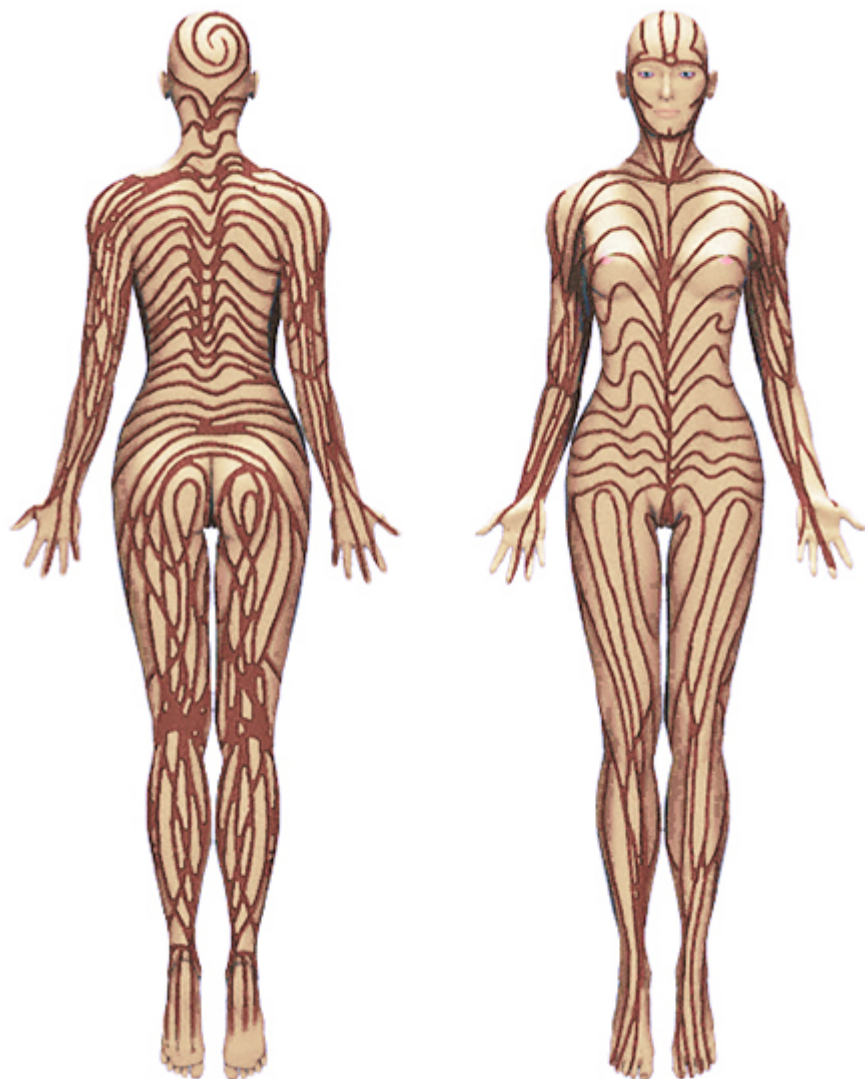
Violaceous plaque with fine scale following the lines of Blaschko.

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Graphic 89626 Version 2.0

## Lines of Blaschko

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The pattern assumed by many different nevoid and acquired skin diseases on the human skin and mucosae. The cause of the pattern of Blaschko lines is unknown; they do not follow nerves, vessels, or lymphatics. The lines described by these conditions not only did not correspond to any known anatomical basis, but were remarkably consistent both from patient to patient and even from one disease to another. The lines may represent a clinical expression of a genetically programmed clone of altered cells, perhaps first expressed during embryogenesis.

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Graphic 57219 Version 3.0

## Koebner reaction in lichen planus



Patients with lichen planus can exhibit the Koebner reaction, in which lesions develop in areas of trauma. In this patient, the Koebner reaction resulted from scratching.

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Graphic 68060 Version 1.0

## Hypertrophic lichen planus

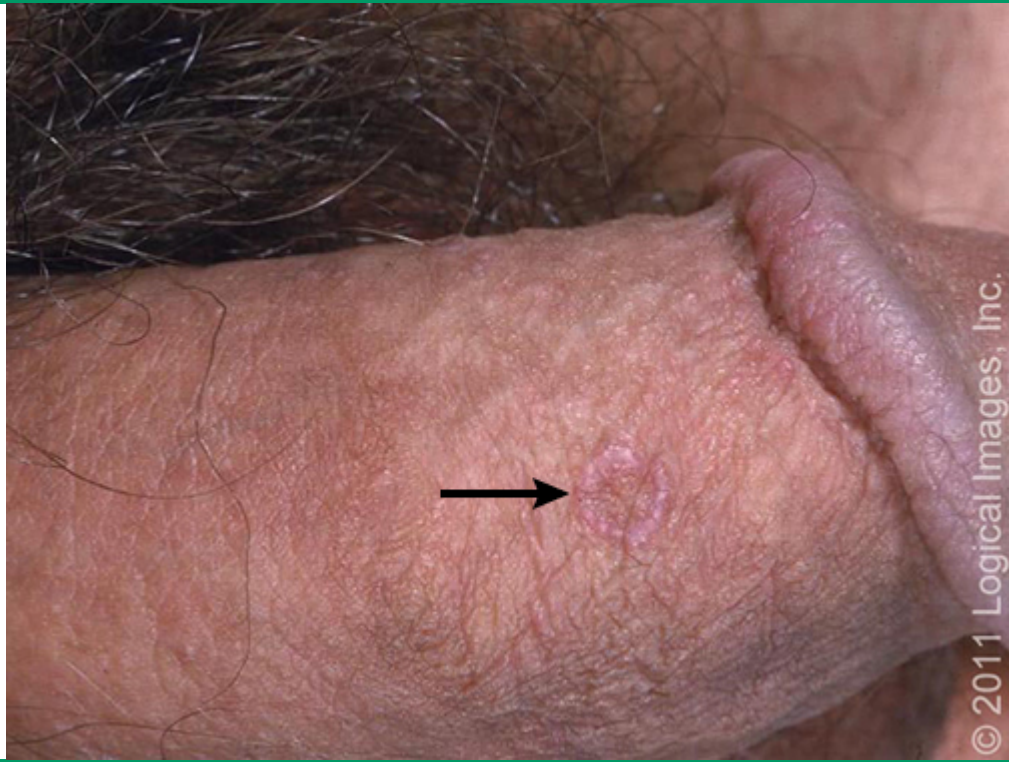


This violaceous and hyperpigmented plaque is a lesion of hypertrophic lichen planus. Fine white lines (Wickham's striae) are visible on the plaque's surface.

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Graphic 54396 Version 6.0

## Annular lichen planus



A violaceous annular plaque is present on the penile shaft.

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Graphic 53806 Version 4.0

## Annular lichen planus



Violaceous annular plaques and papules are present on the penis and scrotum.

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Graphic 76913 Version 5.0

## Annular lichen planus



A violaceous, annular plaque is present.

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Graphic 68197 Version 5.0



## Inverse lichen planus



Violaceous plaques in the axilla.

Graphic 90122 Version 2.0

## Lichen planus



Lichen planus of the nail may present with nail plate thinning, longitudinal ridging (trachyonychia), fissuring, and rarely, pterygium. Nail plate thinning and longitudinal ridging are visible on this patient's nails.

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Graphic 80419 Version 1.0

## Trachyonychia induced by lichen planus



Nail plate roughness, longitudinal ridging, and pitting.

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Graphic 85620 Version 6.0

## Longitudinal nail fissuring caused by lichen planus



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Graphic 85621 Version 6.0

## Lichen planus of the nail

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The proximal nail fold is adherent to the nail bed (pterygium). Longitudinal melanonychia (brown bands in the nail plate) is also present.

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Graphic 86822 Version 5.0

## Lichen planopilaris

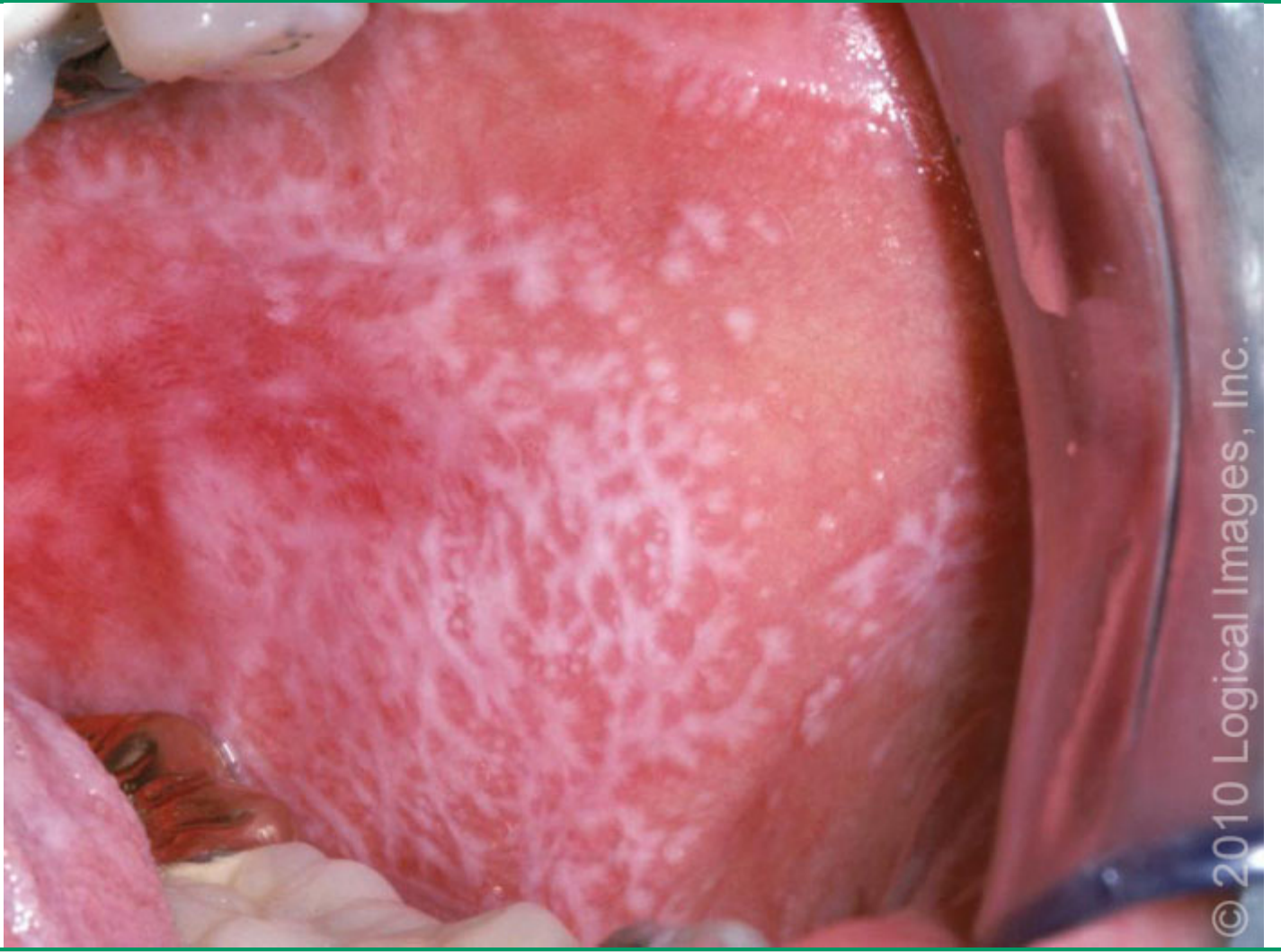


Erythematous, keratotic papules at the base of hair follicles and scarring alopecia are present on the scalp in this patient with lichen planopilaris.

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Graphic 52848 Version 4.0

## Oral lichen planus: Wickham striae



Lacy, white plaques (Wickham striae) are present on the buccal mucosa.

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Graphic 76338 Version 8.0

## Erosive oral lichen planus

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Ulcers are present on the tongue in this patient with erosive oral lichen planus.

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Graphic 62826 Version 5.0



## Oral lichen planus



Erythema and erosions on the gingiva in a patient with oral lichen planus.

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Graphic 54143 Version 6.0

## Oral lichen planus

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Lichen planus. Oral lesions with a white, lacy pattern and an erythematous erosion are present on the buccal mucosa.

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Graphic 80848 Version 1.0

## Penile lichen planus



Lichen planus. Wickham's striae are visible.

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Graphic 60985 Version 2.0

## Lichen planus

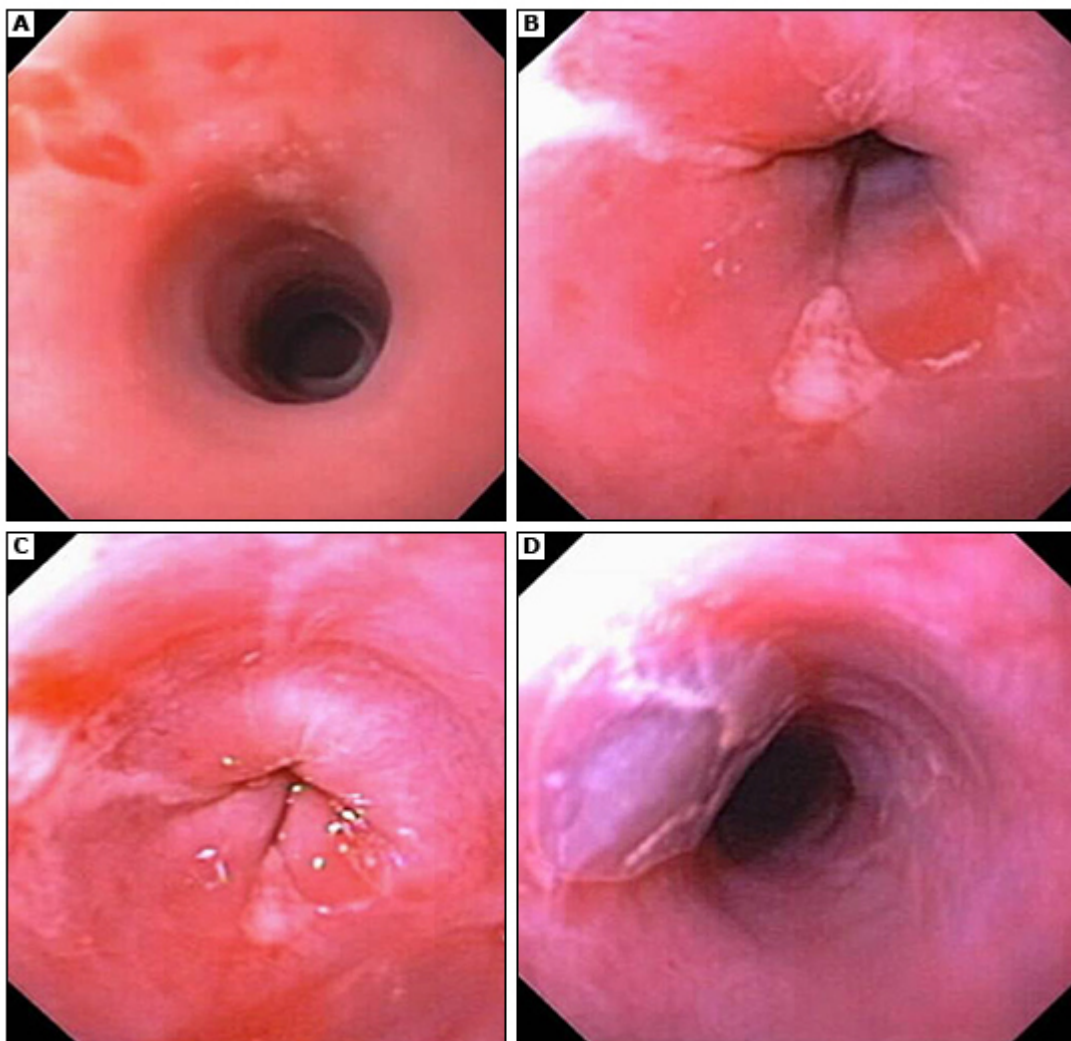


This patient has lichen planus which in this case is clinically indistinguishable from leukoplakia.

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Graphic 72786 Version 2.0

## Esophageal lichen planus

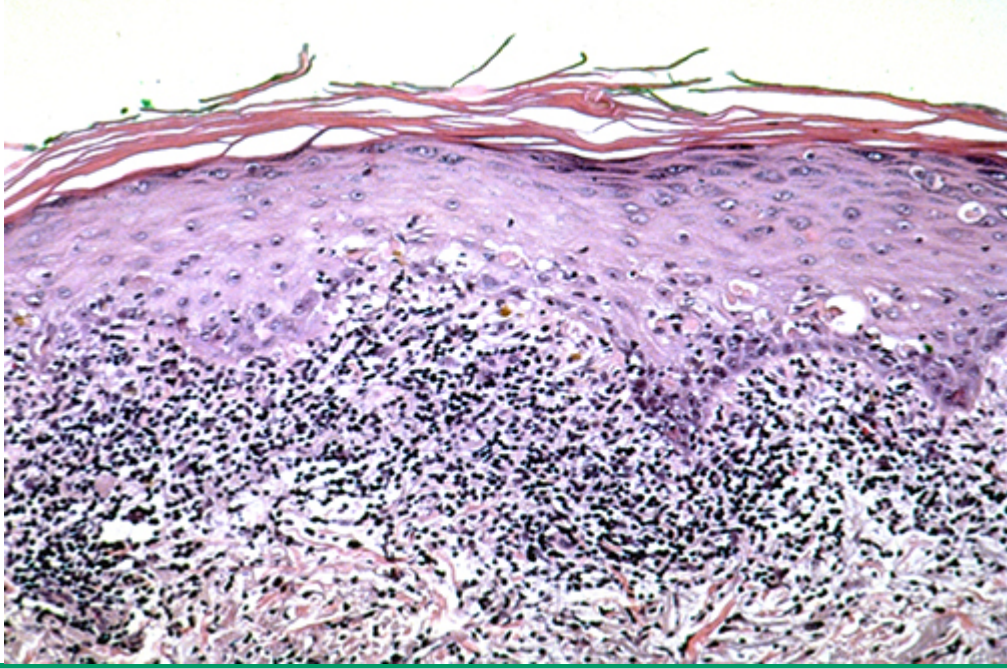


- (A) Endoscopy showing erosions.
- (B) Stricture and erosions.
- (C) Stricture.
- (D) Bulla formation in the esophagus.

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Graphic 72940 Version 4.0

## Cutaneous lichen planus

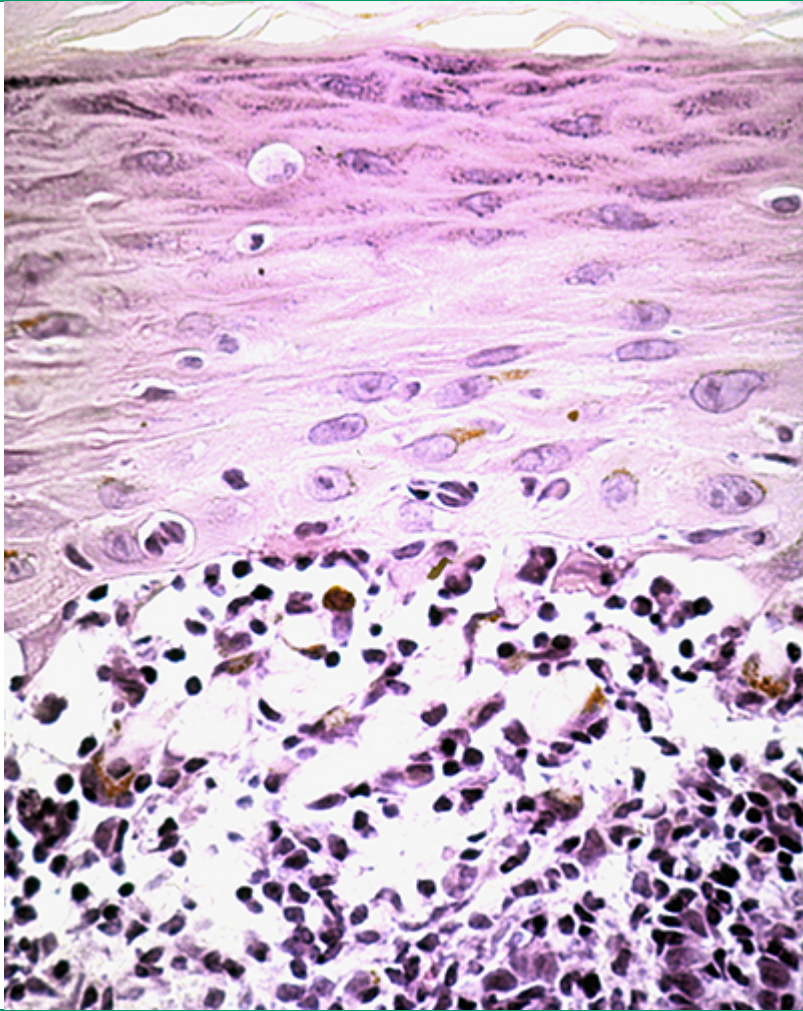


This specimen from a lesion of cutaneous lichen planus demonstrates a band-like lymphocytic infiltrate, saw-tooth rete ridges, multiple apoptotic keratinocytes, and wedge-shaped hypergranulosis.

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Graphic 82025 Version 2.0

## Cutaneous lichen planus



A marked interface dermatitis resulting in vacuolization of the basal layer is present in this pathologic specimen from a lesion of lichen planus. Hypergranulosis is also present.

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Graphic 58826 Version 2.0

## Lichenoid drug eruption



Erythematous to violaceous papules with fine scale are present in this patient with a lichenoid drug eruption.

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Graphic 80165 Version 5.0



## Lichenoid drug eruption



A lichenoid drug eruption manifesting as violaceous and hyperpigmented papules is present in this patient with dark skin.

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Graphic 59258 Version 5.0

## Lichenoid drug eruption (drug-induced lichen planus)



In this patient with a lichenoid drug eruption, flat erythematous papules are typically distributed on the trunk and upper extremities. Grouped and confluent lesions also can be seen.

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Graphic 83767 Version 7.0

## Lichenoid drug eruption (drug-induced lichen planus)



Flat erythematous/violaceous papules on the shoulder and upper arm of a patient with lichenoid drug eruption.

Graphic 83768 Version 2.0

**Comparison of representative topical corticosteroid preparations (classified according to the United States system)**

Potency group*	Corticosteroid	Vehicle type/form	Brand names (United States)	Available strength(s), percent (except as noted)
<b>Super-high potency (group 1)</b>	Betamethasone dipropionate, augmented	Gel, lotion, ointment (optimized)	Diprolene	0.05
	Clobetasol propionate	Cream, gel, ointment, solution (scalp)	Temovate	0.05
		Cream (emollient base)	Temovate E <sup>¶</sup>	0.05
		Lotion, shampoo, spray aerosol	Clobex	0.05
		Foam aerosol	Olux, Olux-E, Tovet	0.05
		Lotion	Impeklo	0.05
		Ointment	Clobetavix	0.05
		Shampoo	Clodan	0.05
		Solution (scalp)	Cormax <sup>¶</sup>	0.05
	Diflucortolone valerate (not available in United States)	Ointment, oily cream	Nerisone Forte (United Kingdom, others)	0.3
	Fluocinonide	Cream	Vanos	0.1
	Flurandrenolide	Tape (roll)	Cordran	4 mcg/cm <sup>2</sup>
Halobetasol propionate	Cream, lotion, ointment	Ultravate	0.05	
<b>High potency (group 2)</b>	Amcinonide	Ointment	Cyclocort <sup>¶</sup> , Amcort <sup>¶</sup>	0.1
	Betamethasone dipropionate	Ointment	Diprosone <sup>¶</sup>	0.05
		Cream, augmented formulation (AF)	Diprolene AF	0.05
	Clobetasol propionate	Cream	Impoyz	0.025
	Desoximetasone	Cream, ointment, spray	Topicort	0.25
		Gel	Topicort	0.05
	Diflorasone diacetate	Ointment	ApexiCon <sup>¶</sup> , Florone <sup>¶</sup>	0.05
		Cream, emollient	ApexiCon E	0.05
	Fluocinonide	Cream, gel, ointment, solution	Lidex <sup>¶</sup>	0.05
	Halcinonide	Cream, ointment, solution	Halog	0.1
Halobetasol propionate	Lotion	Bryhali	0.01	
<b>High potency (group 3)</b>	Amcinonide	Cream	Cyclocort <sup>¶</sup> , Amcort <sup>¶</sup>	0.1
		Lotion	Amcort <sup>¶</sup>	0.1
	Betamethasone dipropionate	Cream, hydrophilic emollient	Diprosone <sup>¶</sup>	0.05
	Betamethasone valerate	Ointment	Valisone <sup>¶</sup>	0.1
		Foam	Luxiq	0.12
	Desoximetasone	Cream	Topicort LP <sup>¶</sup>	0.05
	Diflorasone diacetate	Cream	Florone <sup>¶</sup>	0.05
	Diflucortolone valerate (not available in United States)	Cream, oily cream, ointment	Nerisone (Canada, United Kingdom, others)	0.1
	Fluocinonide	Cream aqueous emollient	Lidex-E <sup>¶</sup>	0.05
	Fluticasone propionate	Ointment	Cutivate <sup>¶</sup>	0.005
Mometasone furoate	Ointment	Elocon	0.1	

	Triamcinolone acetonide	Cream, ointment	Aristocort HP <sup>¶</sup> , Kenalog <sup>¶</sup> , Triderm	0.5	
<b>Medium potency (group 4)</b>	Betamethasone dipropionate	Spray	Sernivo	0.05	
	Clocortolone pivalate	Cream	Cloderm	0.1	
	Fluocinolone acetonide	Ointment	Synalar <sup>¶</sup>	0.025	
	Flurandrenolide	Ointment	Cordran	0.05	
	Fluticasone propionate	Cream	Cutivate <sup>¶</sup>	0.05	
	Hydrocortisone valerate	Ointment	Westcort	0.2	
	Mometasone furoate	Cream, lotion, ointment, solution	Elocon <sup>¶</sup>	0.1	
	Triamcinolone acetonide	Cream	Cream	Kenalog <sup>¶</sup> , Triderm	0.1
		Ointment	Ointment	Kenalog <sup>¶</sup>	0.1
		Ointment	Ointment	Trianex	0.05
Aerosol spray		Aerosol spray	Kenalog	0.2 mg per 2 second spray	
Dental paste	Dental paste	Oralone	0.1		
<b>Lower-mid potency (group 5)</b>	Betamethasone dipropionate	Lotion	Diprosone <sup>¶</sup>	0.05	
	Betamethasone valerate	Cream	Beta-Val, Valisone <sup>¶</sup>	0.1	
	Desonide	Ointment	DesOwen, Tridesilon <sup>¶</sup>	0.05	
		Gel	Desonate	0.05	
	Fluocinolone acetonide	Cream	Synalar <sup>¶</sup>	0.025	
	Flurandrenolide	Cream, lotion	Cordran	0.05	
	Fluticasone propionate	Lotion	Cutivate	0.05	
	Hydrocortisone butyrate	Cream, lotion, ointment, solution	Locoid, Locoid Lipocream	0.1	
	Hydrocortisone probutate	Cream	Pandel	0.1	
	Hydrocortisone valerate	Cream	Westcort <sup>¶</sup>	0.2	
	Prednicarbate	Cream (emollient), ointment	Dermatop	0.1	
	Triamcinolone acetonide	Lotion	Lotion	Kenalog <sup>¶</sup>	0.1
Ointment		Ointment	Kenalog <sup>¶</sup>	0.025	
<b>Low potency (group 6)</b>	Alclometasone dipropionate	Cream, ointment	Aclovote	0.05	
	Betamethasone valerate	Lotion	Beta-Val <sup>¶</sup> , Valisone <sup>¶</sup>	0.1	
	Desonide	Cream	DesOwen, Tridesilon <sup>¶</sup>	0.05	
		Lotion	DesOwen, LoKara	0.05	
		Foam	Verdeso	0.05	
	Fluocinolone acetonide	Cream, solution	Cream, solution	Synalar <sup>¶</sup>	0.01
		Shampoo	Shampoo	Capex	0.01
		Oil <sup>Δ</sup>	Oil <sup>Δ</sup>	Derma-Smoothe/FS Body, Derma-Smoothe/FS Scalp	0.01
Triamcinolone acetonide	Cream, lotion	Cream, lotion	Kenalog <sup>¶</sup> , Aristocort <sup>¶</sup>	0.025	
<b>Least potent (group 7)</b>	Hydrocortisone (base, ≥2%)	Cream, ointment	Cream, ointment	Hytone, Nutracort <sup>¶</sup>	2.5
		Lotion	Lotion	Hytone, Ala Scalp, Scalacort	2
		Solution	Solution	Texacort	2.5
	Hydrocortisone (base, <2%)	Ointment	Ointment	Cortaid, Cortizone 10, Hytone, Nutracort	1
		Cream	Cream	Cortaid <sup>¶</sup> , Cortizone 10, Hytone, Synacort	1

		Gel	Cortizone 10	1
		Lotion	Aquanil HC, Sarnol-HC, Cortizone 10	1
		Spray	Cortaid	1
		Solution	Cortaid, Noble, Scalp Relief	1
		Cream, ointment	Cortaid	0.5
	Hydrocortisone acetate	Cream	MiCort-HC	2.5
		Lotion	Nucort	2

\* Listed by potency according to the United States classification system: group 1 is the most potent, group 7 is the least potent. Other countries use a different classification system with only 4 or 5 groups.

¶ Inactive United States brand name for specific product; brand may be available outside United States. This product may be available generically in the United States.

Δ 48% refined peanut oil.

Data from:

1. Lexicomp Online. Copyright © 1978-2021 Lexicomp, Inc. All Rights Reserved.
2. Tadicherla S, Ross K, Shenefelt D. Topical corticosteroids in dermatology. *Journal of Drugs in Dermatology* 2009; 12:1093.
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## Contributor Disclosures

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