OCTOBER 2025 VOL 9 NO 2

A MEDICINE TODAY PUBLICATION

Dermatology Collection

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Reprints in Dermatology

Postadolescent acne in women: what is the cause and how can it be managed?

Vulval lichen sclerosus – not uncommon, serious and often missed

The use of topical corticosteroids for inflammatory dermatoses

Multiple scaly plaques on the head and arm

A man with a painless papule on the leg

A boy with an eruption of erythematous facial papules

A unilateral facial rash with ocular involvement

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PEER REVIEWED UPDATES FOR MEDICAL PRACTITIONERS

FOREWORD FROM THE EDITOR-IN-CHIEF, DERMATOLOGY COLLECTION

Our October 2025 *Dermatology Collection* offers seven practical and clinically relevant articles covering both common and complex conditions.

Postadolescent acne in women is increasingly recognised as a distinct clinical entity, often hormonally driven and sometimes associated with polycystic ovary syndrome. The article outlines a logical, stepwise approach to assessment and management.

Vulval lichen sclerosus remains one of the most underdiagnosed conditions in women. This comprehensive review details the importance of early recognition, appropriate induction and maintenance therapy with potent topical corticosteroids, and the role of lifelong follow up in preventing scarring and malignancy.

Topical corticosteroids remain the cornerstone of treatment for a wide range of inflammatory dermatoses. A valuable update is provided on their safe and effective use, with practical guidance on selecting appropriate potencies and formulations for different conditions and body sites.

The *Dermatology Clinics* review the differential diagnosis and management of multiple scaly plaques on the head and arm and of dermatofibroma. Two further thought-provoking paediatric and ocular cases — one featuring facial papules in a teenage boy and the other a unilateral facial rash with eye involvement — offer useful reminders about diagnostic reasoning in general practice.

Together, these articles provide concise, evidence-based updates to assist GPs in recognising, treating and referring patients with skin conditions that have both medical and psychosocial impact.

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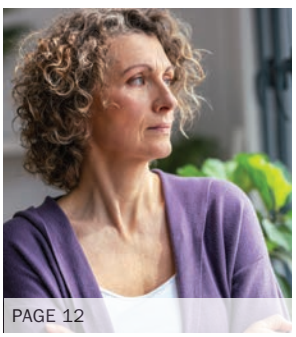

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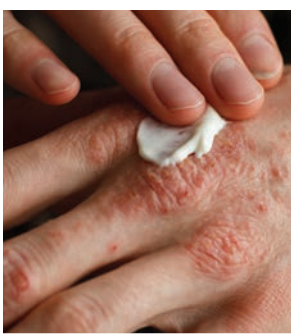

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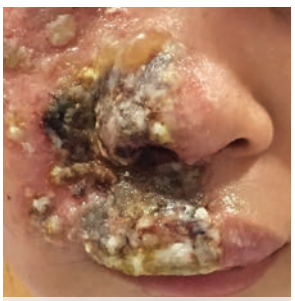

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Postadolescent acne in women

What is the cause and how can it be managed?

JO-ANN SEE FACD; LISA BYROM FACD

Increasing numbers of women worldwide continue to have acne after adolescence or may even develop it in their 20s to 40s. These patients may have underlying hormonal abnormalities and may benefit from hormone therapy.

Acne vulgaris is a common, self-limiting disorder affecting about 9.4% of the world's population, making it the eighth most prevalent disease globally.[1] Women, including transgender and gender-diverse individuals with female reproductive organs, can have postadolescent acne that persists from the teenage years into later life, or late-onset acne that develops at or after the age of 20 years in those who have not had acne during their teens, and may even persist until menopause.[2] The two presentations can also overlap and merge in many patients. It is important to recognise women with postadolescent acne as a distinct group because:

- the number of women affected worldwide is increasing[3]
- the psychosocial effects of acne in this group may be profound and disproportionate to the severity of acne; many women find that their acne affects their working life or professional careers (e.g. self-consciousness when having to give work presentations or meet with clients)[4]
- some women may have abnormal serum androgen levels that require further investigation
- these patients typically respond well to hormone therapy alone or as an adjunct to other acne therapy, even in the presence of normal laboratory investigation findings

KEY POINTS

- Postadolescent acne in women may continue beyond the teenage years or develop at or after the age of 20 years.
- Women with postadolescent acne may have normal or raised serum androgen levels.
- Polycystic ovary syndrome may be an underlying cause of postadolescent acne in women.
- Assessment should include menstrual history taking and examination for clinical signs of hyperandrogenism, such as hirsutism.
- Hormone therapy (usually the combined oral contraceptive pill) is an effective adjunct in the management of postadolescent acne in women, including those with normal serum androgen profiles. Other antiandrogens, such as spironolactone or cyproterone acetate, may need to be taken in addition to the oral contraceptive pill.
- Holistic care with consideration of genetic factors (such as family history) and lifestyle factors (such as skincare and diet) should also be considered.

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1. HISTORY-TAKING CHECKLIST FOR WOMEN PRESENTING WITH POSTADOLESCENT ACNE

- **At what age did the acne start?** Ask the patient if the acne started during the teenage years or later.
- **Where was the acne first noticed, and where did it spread to?** Ask the patient if the acne follows a 'T zone' or 'hormonal' distribution.
- **How long does the acne last?** Ask the patient whether the acne persists for months or years.
- **Does the acne recur in the same area? Are there any symptoms?** Ask the patient about the characteristics of the acne, such as the feeling of tenderness or oily skin.
- **Is there any correlation of the acne with the menstrual cycle?** Ask the patient about her menstrual history. The presence of irregular periods or premenstrual exacerbation of acne may suggest an association between the acne and her menstrual cycle.
- **Are there features of hyperandrogenism other than menstrual irregularity?** Ask the patient if they have noticed symptoms such as deepening of the voice, an increased libido, hirsutism and male-pattern baldness.
- **Does the patient's mother or sister have acne?** Ask the patient if any of their female relatives also had postadolescent acne.
- **Are there lifestyle factors that may promote or exacerbate acne?** Ask the patient about products she may use regularly, such as make-up or sports headgear.
- **Is there an impact on the patient's psychological wellbeing?** Ask the patient about her mental health in the context of the acne.
- **What is the general health of the patient?** Ask the patient about her medical history, including the use of medications that may trigger acne.

- with effective treatment, patients' quality of life scores improve.

Acne, hormones and the sebaceous glands

Acne is a complex multifactorial disorder. Its pathogenesis involves:

- abnormal keratinisation of the pilosebaceous opening
- increased sebum production by the sebaceous glands
- colonisation of *Cutibacterium acnes* with altered phylotypes
- inflammation.[5]

Blockage of the sebaceous duct due to abnormal keratinisation produces a microscopic plug: the microcomedone. Colonisation of *C. acnes* in the pilosebaceous duct contributes to an inflammatory response that manifests as papules, pustules and inflammatory cysts. Effective acne management involves targeting these pathogenic steps; combination therapy is often used to target as many pathogenic factors as possible.

Sebaceous glands are found throughout the body and are present in the greatest quantity and density on the face. These glands secrete sebum in response to androgenic stimulation. In general, patients with acne produce more sebum than individuals without acne, and sebum production is greater in those with more severe acne.[6]

Under normal conditions, the ovaries contribute about 50% of the circulating androgens in women. Overproduction of androgens by the ovaries can occur in conditions such as polycystic ovary syndrome (PCOS) or ovarian tumours. The adrenal glands contribute the remaining 50% of circulating androgens in women. Stress may be a trigger that increases adrenal androgen production, which then stimulates increased sebum production.[7,8]

A detailed discussion of PCOS and other hormonal causes of acne, such as ovarian tumours and congenital adrenal hyperplasia, as well as steroid ingestion as a cause, is beyond the scope of this article.

Figure 1. A woman with acne affecting the lower-third of the face.
Image courtesy of the authors. Image reproduced with patient consent.

Recognising postadolescent acne: history taking

Clinical history taking of a woman presenting with postadolescent acne should include a number of factors, as discussed below and summarised in Box 1.[7,9]

Age of onset

Some patients develop acne with the onset of menstruation (i.e. during the teenage years), whereas some patients develop acne when they are in their 20s or older.

Distribution of acne

In some patients, the clinical pattern of postadolescent acne may look indistinguishable from teenage 'T zone' acne, which affects the forehead, nose and chin, with or without upper chest and upper back involvement. In other patients, there is a more 'hormonal' distribution, with the lower third of the face being affected, particularly the lower cheeks, jawline, chin and neck (Figure 1). The trunk may also be affected, and most patients will note seborrhoea or oily skin.

Duration of acne

Patients may complain that their current acne tends to last longer compared with acne that occurred during adolescence – for weeks, rather than days.

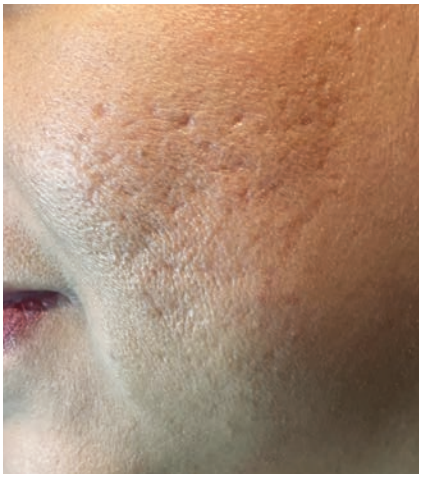


Figure 2. A woman with acne scarring and postinflammatory pigmentation.
Image courtesy of the authors. Image reproduced with patient consent.

Characteristics of acne

Patients often report that their acne feels tender or 'deep seated'. Some may describe their face as hurting. The acne may persist for weeks, heal and then recur weeks or months later in the same area. The patient may present with postinflammatory redness or pigmentation lasting for weeks or months as the acne resolves, which is often distressing for the patient. The inflammatory and chronic nature of such acne may also lead to scarring (Figure 2).

Menstrual history

It is common for acne to develop a week before menses and continue for one to two weeks. Some patients will notice acne activity at ovulation. A careful menstrual history is important in the assessment of a woman who has late-onset acne. About 60 to 70% of women may complain of worsening of their acne on a cyclical basis, usually premenstrually. An irregular menstrual cycle may suggest underlying hyperandrogenism and the presence of PCOS. It may be worthwhile for the patient to chart her menstrual cycle because patients often assume their cycle is regular. Menstrual irregularity is defined as amenorrhoea for more than three months or irregularity of the menstrual cycle of greater than seven days from a standard 28-day cycle over three consecutive cycles.

Hyperandrogenism

Features of hyperandrogenism other than menstrual irregularity may need specific enquiry; these features are listed in Box 2. Hirsutism may not be readily evident because patients may have undergone hair removal by a variety of means, such as waxing, electrolysis or laser treatment. Mild hirsutism and irregular menstrual cycles have been reported in up to 29% and 14%, respectively, of women with postadolescent acne.[10]

Obesity, hirsutism and irregular menstrual cycles are features of PCOS but are not always present in women with the syndrome.[11] Data regarding the number of women with postadolescent acne who have underlying PCOS are conflicting; figures range between 10 and 50%.[9,12] However, the diagnosis of PCOS can be difficult in some cases because the presentation of PCOS has marked clinical heterogeneity (including features that may have different potential causes). The presentation is also affected by individual differences (e.g. weight, ethnicity, lifestyle differences) because there are no universally accepted diagnostic criteria for PCOS.[13,14]

Family history

Individuals with a family history of acne tend to have an increased risk of more severe acne.[8]

Lifestyle factors

Many patients report flares of acne when they are feeling increased stress. Creamy or 'greasy' cosmetics can promote the plugging of the pilosebaceous follicle opening and are comedogenic. Some patients may work in occupations where heat may play a role, such as working in kitchens. Friction or trauma due to occlusive headgear, such as that worn for cycling, rollerblading or softball, may rupture existing comedones and cause inflammatory acne.

The use of personal protective equipment has become more important in the development of acne since the COVID-19 pandemic. Women with a prior history of acne who wore face masks continuously had significantly higher rates of experiencing 'maskne' compared with women without a history of acne.[15]

2. FEATURES OF HYPERANDROGENISM

- Male-pattern baldness
- Hirsutism
- Increased libido
- Acanthosis nigricans
- Deepening of the voice
- Menstrual irregularities
- Insulin resistance

Mental health

The psychological impact of acne can be assessed using questionnaires, such as the Dermatology Life Quality Index. Stigma can be associated with adult acne, which may affect personal and professional relationships.

General medical history

Certain drugs taken for coexisting medical conditions may exacerbate acne. Some examples include phenytoin and lithium, which are prescribed for neurological diseases.[16,17] Use of the 52 mg levonorgestrel-releasing intrauterine system for contraception may be associated with the development of acne, which is the most commonly reported reason for removal of the device.[18]

Examination of postadolescent acne

Postadolescent acne may be clinically indistinguishable from adolescent acne. The examination should focus on:

- the distribution of acne – this can include the lower cheeks, jawline, chin, neck and trunk

TABLE 1. GUIDELINES FOR INTERPRETING SERUM ANDROGEN PROFILES

Screening test	Test result	Possible diagnosis
DHEA-S level	>20 mcmol/L (>700 mcg/dL)	Adrenal tumour
	10 to 20 mcmol/L (350 to 700 mcg/dL)	Congenital adrenal hyperplasia
	>5 mcmol/L (>200 mcg/dL)	PCOS
Total testosterone	>6.9 nmol/L (>200 ng/dL)	Ovarian tumour
	<5.2 nmol/L (<150 ng/dL)	PCOS
LH to FSH ratio	2 to 3, or greater	PCOS

Abbreviations: DHEA-S = dehydroepiandrosterone sulfate; FSH = follicle-stimulating hormone; LH = luteinising hormone; PCOS = polycystic ovary syndrome.

- the severity of acne – the acne may present as nodules, cysts and scarring
- the presence of psychological distress – this includes how the patient feels, and whether that prevents her from engaging in normal activities
- features of hyperandrogenism – these include hirsutism or androgenic alopecia, in particular.

Late-onset acne typically localises to the lower third of the face, especially the lower cheeks, jawline, chin and neck. This distribution contrasts with that of adolescent acne, which is often midfacial in distribution – in the 'T zone' (i.e. the forehead, nose and cheeks). Features of hyperandrogenism should be investigated (Box 2). Although there is often a typical 'hormonal' distribution, the pattern may be the same as that of teenage acne; thus, the two types cannot always be differentiated by the distribution of the acne.

Investigations for postadolescent acne

Although laboratory investigations are not indicated for most patients with acne, hormone investigations are appropriate for women with postadolescent acne, menstrual irregularities or evidence of hyperandrogenism, such as hirsutism. Hormonal contraception and spironolactone should be ceased before conducting hormone investigations.

A basic screening test for androgenic abnormalities should include levels of serum free testosterone, dehydroepiandrosterone sulfate (DHEA-S) and sex hormone-binding globulin, as well as the ratio of luteinising hormone (LH) to follicle-stimulating hormone (FSH). Elevated levels of free testosterone suggest hyperandrogenism but do not indicate the origin of the elevated testosterone. Increased levels of DHEA-S suggest an adrenal cause, which may include congenital adrenal hyperplasia or, rarely, an adrenal tumour.[6] Elevated levels of testosterone with an increased LH to FSH ratio (greater than 2 to 3) are consistent with a diagnosis of PCOS. The interpretation of serum androgen profiles is summarised in Table 1.

Frequently, both the ovaries and adrenals are implicated in androgen overproduction in women with late-onset acne.[6] Blood samples should be obtained in the early follicular phase (days 1 to 7) of the menstrual cycle where possible, and patients taking oral contraceptives should discontinue their medication for one month before testing.

Depending on the clinical circumstances, other investigations may be indicated. These include testing levels of serum fasting glucose and lipids, prolactin, androstenedione and 17α-hydroxyprogesterone, and performing pelvic ultrasound to detect polycystic ovaries. Patients with PCOS often have insulin resistance and are at an increased risk of developing diabetes mellitus and cardiovascular disease.[11] Referral to an endocrinologist or gynaecologist may be indicated. Patients may also benefit from weight reduction or a low-glycaemic-index diet; therefore, a dietitian may also provide expert advice.[19]

Compared with women of the same age without acne, women with postadolescent acne tend to have higher plasma levels of free androgens, often at high normal levels.[6,20] It is important to appreciate, however, that the serum androgen measurements may be normal in many patients with postadolescent acne; this may reflect errors in sampling, contraceptive therapy or the end-organ response to androgens.

Sebaceous glands have a range of enzymes capable of metabolising androgens to more potent forms. For example, dihydroxytestosterone is converted to testosterone by type 1 5α-reductase in acne-prone follicles. The local androgen levels may be more significant in regulating sebum production because of metabolism or end-organ hyper-responsiveness compared with the circulating androgen levels.[6,20] This is important to explain to patients because they often cannot understand why hormone treatments are prescribed in the presence of 'normal' hormonal assay findings.

Management of postadolescent acne

General advice and counselling

Resources on managing acne can be found on the All About Acne website (https://acne.org.au). Excessive washing and the use of antibacterial soaps and scrubs are not necessary for the cleansing of acne-affected skin; adversely, these actions may irritate the skin. Gentle cleansing using an oil-free, soap-free cleanser is appropriate, particularly for those women who have sensitive skin, whereas a foaming cleanser may be more appealing to those who have oily skin. General measures include using oil-free sunscreens, make-up products and moisturisers.

Educating and counselling patients regarding their acne is vital. Myths related to acne (e.g. 'poor hygiene' being a cause) should be dispelled. Patient expectations about treatments should be clarified because it may take up to three months before significant improvements are observed. Patients may need reassurance because they may have had acne for a long time or may be resistant to previously tried conventional treatments.[21] Therefore, they can be frustrated by the time they present to you. Encouragement during this period is helpful to promote treatment adherence.

Women with PCOS may have abnormal lipid profiles and are at an increased risk of type 2 diabetes.[11] Lifestyle modifications, including weight reduction measures and exercise, are recommended for these patients.[14,19] A multidisciplinary team approach involving a dietitian, gynaecologist or endocrinologist may be required.

Addressing the psychological impact of acne may include referring the patient to counselling, psychology or psychiatric services.[9,22] The therapeutic options for acne are summarised in Table 2. A suggested approach for the investigation and management for a patient presenting with postadolescent acne is shown in the Flowchart.

TABLE 2. THERAPEUTIC OPTIONS FOR POSTADOLESCENT ACNE

Medication class	Drugs
Topical agents	
Keratolytics	Azelaic acid 15%
	Benzoyl peroxide 2 to 10%
	Glycolic acid and lactic acid (alpha hydroxy acids)
	Salicylic acid (beta hydroxy acid)
Topical antibiotics	Clindamycin 1%
	Clindamycin 1% and benzoyl peroxide 5% combination
	Clindamycin 1% + tretinoin 0.025% combination
	Erythromycin 2% as compounded formula
Topical retinoids	Adapalene 0.1%
	Adapalene 0.1% or 0.3% plus benzoyl peroxide 2.5%
	Tretinoin 0.01%
	Trifarotene 0.005% (cream)
Topical antiandrogen	Clascoterone 1% (cream)
Oral agents	
Systemic antibiotics (first-line)	Doxycycline 50 to 100 mg daily
	Minocycline 50 to 100 mg daily (if doxycycline is not tolerated)
Systemic antibiotics (second-line)	Erythromycin 250 to 500 mg twice daily
	Trimethoprim 160 mg plus sulfamethoxazole 800 mg, once or twice daily
	Azithromycin 500 mg three times a week
	Cephalexin 500 mg twice daily
Systemic retinoid	Isotretinoin
Hormone therapy	Combined oral contraceptives: • ethinylestradiol plus cyproterone acetate • ethinylestradiol plus desogestrel • ethinylestradiol plus dienogest • ethinylestradiol plus drospirenone • ethinylestradiol plus gestodene
	Cyproterone acetate
	Glucocorticoids: • dexamethasone • prednisolone
	Spironolactone*

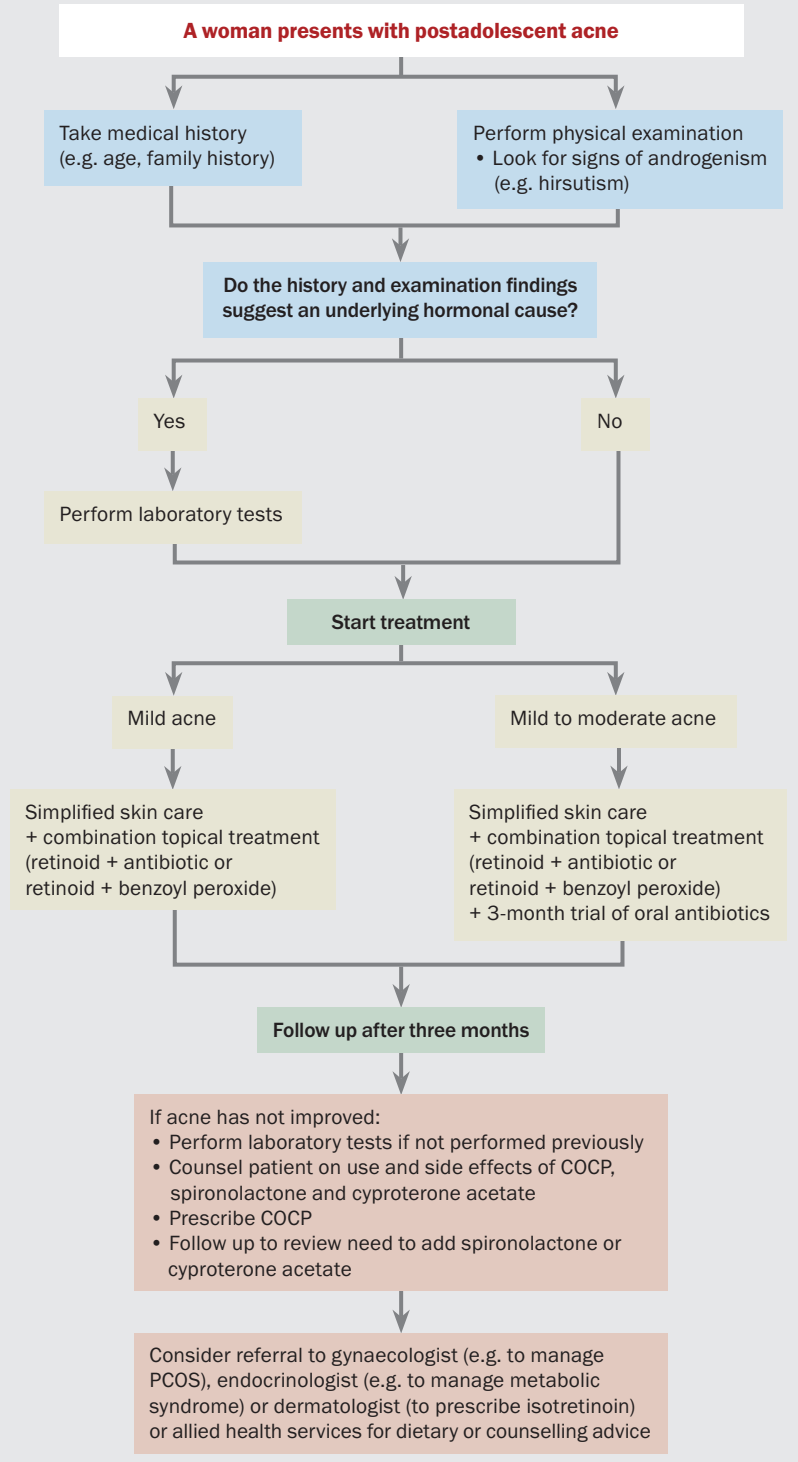
* Used off label for postadolescent acne.

Topical agents

Topical salicylic acid, glycolic acid, azelaic acid and benzoyl peroxide are available without prescription in the form of cleansers or leave-on products. They are keratolytic and reduce comedone formation.

Cleansing, moisturising and protecting against sun exposure are important steps in caring for acne-prone skin. Skin cleansers should be nonirritating, fragrance-free, alcohol-free and noncomedogenic, and should maintain the function of the skin barrier. These may also contain anti-acne ingredients such as salicylic acid or benzoyl peroxide. Moisturisers should be alcohol-free,

APPROACH FOR INVESTIGATION AND MANAGEMENT OF POSTADOLESCENT ACNE



Abbreviations: COCP = combined oral contraceptive pill; PCOS = polycystic ovary syndrome.

oil-free, noncomedogenic and fragrance-free, and should help manage the drying effects of acne treatments without promoting acne formation. Sunscreens should also be nonirritating, contain minimal ingredients, provide broad-spectrum coverage and have SPF30+ as a minimum.[23]

Topical antibiotic acne treatments include clindamycin 1% lotion or gel and erythromycin 2%, which can be compounded. Both these agents may be used in pregnant women, but as they may be secreted in breast milk their use should be avoided during lactation. They are particularly helpful in cases of inflammatory acne.

Topical tretinoin is the original vitamin A analogue in the retinoid class of medications, and these act mainly as keratolytic agents. Adapalene is a third-generation topical retinoid that is photostable and does not cause photosensitivity; therefore, may be applied during the daytime. A combination topical retinoid treatment containing adapalene 0.1% plus benzoyl peroxide 2.5% is available; it should be applied once daily. A stronger preparation containing adapalene 0.03% plus benzoyl peroxide 2.5% is also available. The newest fourth-generation retinoid, trifarotene, is available as a 0.005% cream. Ideally, these retinoids should be applied at night, and patients should be advised about their side effects, which include irritation and photosensitivity. These agents should be avoided in pregnancy.

Clascoterone 1% cream is a topical androgen receptor inhibitor that blocks the stimulation of oil glands in the skin. It does not cause increased skin sensitivity and is nonirritating. It should be applied twice a day.[24]

Combination topical and oral therapy is often required in patients with postadolescent acne.

Systemic antibiotics

Oral antibiotic therapy is effective for inflammatory acne and suppresses acne until spontaneous clearing occurs.

Doxycycline 50 to 100 mg daily and minocycline 50 to 100 mg daily are usually used as the first-line antibiotics. Erythromycin 250 to 500 mg twice daily (or erythromycin ethyl succinate 400 to 800 mg twice daily) and trimethoprim 160 mg plus sulfamethoxazole 800 mg once or twice daily are second-line antibiotic choices. Other options include cephalexin 500 mg twice daily or azithromycin 500 mg three times a week. The choice of antibiotic is influenced by drug interactions.

Ideally, oral antibiotics should only be used for a maximum of three months at a time to minimise potential antibiotic resistance. If longer courses are required, then the antibiotic should be used in combination with topical benzoyl peroxide, as this reduces the risk of developing antibiotic resistance.[21,25] Alternatively, a break of one to two weeks may be taken between three-month oral antibiotic courses during which benzoyl peroxide is used. Therefore, systemic antibiotics are not an ideal option for long-term therapy, which is often needed for hormonal acne.

Oral isotretinoin

Women who have nodulocystic or scarring acne should be referred to a dermatologist for treatment with oral isotretinoin. Oral isotretinoin reduces comedogenesis, reduces sebum secretion and is anti-inflammatory.[26]

Patients should be given counselling with respect to contraception and the risk of birth defects while taking systemic retinoid medication. Pretreatment investigations include a serum lipid level test, a serum pregnancy test and liver function tests; liver function tests should be conducted regularly during therapy.

Although patients with hormonal acne respond well to isotretinoin, they may relapse when their treatment courses are complete because of the underlying hormonal stimulation of the oil glands. Although low-dose or even intermittent oral isotretinoin will help control acne in some patients, oral isotretinoin should be considered a treatment option for refractory cases. If acne tends to recur quickly after a course of isotretinoin has been completed, then antiandrogen hormone therapy, such as the oral contraceptive, should be considered as maintenance treatment.

Hormone therapy

Hormone therapy, usually with the oral contraceptive pill, is very effective in women who have postadolescent acne with or without elevated serum androgen levels.[27] Hormone therapy reduces sebum production by decreasing androgenic stimulation of the sebaceous glands. It may be used in combination with other anti-acne therapies.

Hormone therapy for postadolescent acne in women is indicated:

- in those with ovarian, adrenal or peripheral hyperandrogenism
- in those with PCOS
- for moderate to severe acne unresponsive to other therapies
- if relapse occurs after multiple courses of antibiotics
- if relapse occurs quickly after a course of isotretinoin as an alternative to repeated courses of isotretinoin.

The therapeutic effect of hormone therapy is slow, and patients should be warned not to expect noticeable improvement for three months. Therapy should be continued for at least 12 months. Relapses are common when hormone therapy is ceased.

Combined oral contraceptives

The oestrogenic component of the combined oral contraceptive pill suppresses ovarian production of androgens and stimulates the production of sex hormone-binding globulin, thus reducing free testosterone levels. This is beneficial and hence some oral contraceptives are prescribed off label for acne because the oil glands are exposed to a reduced androgenic stimulus.[28]

Although all combined oral contraceptives are effective for acne because of the oestrogenic component, those containing androgenic progestins, such as norgestrel and levonorgestrel, are theoretically less effective. Preparations containing low-androgenic progestogens, such as desogestrel or gestodene, are considered helpful anti-acne contraceptives, as they impart fewer androgenic effects, such as oily skin that can promote acne.[29] For many years, the 'gold standard' has been the combination of ethinylestradiol 35 mcg plus cyproterone acetate 2 mg. Alternatives include the combinations ethinylestradiol 30 mcg plus dienogest 2 mg, ethinylestradiol 30 mcg plus drospirenone 3 mg and ethinylestradiol 20 mcg plus drospirenone 3 mg.

Side effects of hormone therapy include nausea, breast tenderness, weight gain and headache. There may also be an increased risk of breast cancer and this should be discussed with the patient, along with other relative contraindications.[30] Mention should also be made of the several long-term benefits of oral contraceptive therapy, which include reduced risks of ovarian and uterine cancers.[31]

Appropriate patient selection and counselling are required with the use of the oral contraceptive pill. Although this article focuses on postadolescent acne, hormone therapy is also appropriate for female adolescents with acne; however, oral contraceptive therapy should be avoided before puberty because of the risk of accelerated epiphyseal closure.

The efficacy of oral contraceptives for acne is attributed largely to the oestrogenic component. Progestogen-only pills and implants are therefore unsuitable as anti-acne therapies, and some patients using these have noted a worsening of their acne. Patients should be warned that acne improvement may be slow (taking at least three months) and that treatment is long term (at least one year). Combination treatment may yield improved efficacy; if the acne has not improved significantly after three to six months of oral

contraceptive therapy, then an androgen receptor antagonist, such as cyproterone acetate or spironolactone, can be added.

Cyproterone acetate

Cyproterone acetate is an antiandrogenic progestogen that acts by both inhibiting ovulation and blocking the androgen receptor. The combination of cyproterone acetate 2 mg plus ethinylestradiol 35 mcg is very effective for the treatment of acne in women with mild to moderate hyperandrogenism.[32]

Cyproterone acetate is also available as a single agent in 10 mg and 50 mg tablets and can be prescribed in addition to a combined oral contraceptive preparation containing it, or any other combined oral contraceptive. The dose of cyproterone acetate can therefore be increased if the acne is unresponsive to an ethinylestradiol plus cyproterone acetate oral contraceptive. For example, 50 mg of cyproterone acetate may be added to the first 10 days of a cycle of an ethinylestradiol 35 mcg plus cyproterone acetate 2 mg combined oral contraceptive (or another combined oral contraceptive), starting with the first active pill. Alternatively, 10 mg of cyproterone acetate can be added to the first 15 days of the pill cycle. In postmenopausal women or in those who have undergone hysterectomy, 50 mg of cyproterone acetate may be added to the entire active cycle (21 days) of therapy with an ethinylestradiol plus cyproterone acetate-containing oral contraceptive.

Improvement can be seen in 75 to 90% of women with acne who are treated with cyproterone acetate 50 to 100 mg per day.[33] Oestrogen is necessary in these regimens because cyproterone acetate has strong antioestrogenic effects. Side effects of cyproterone acetate therapy include menstrual abnormalities, breast tenderness and enlargement, mood changes, headache, nausea, melasma and fluid retention.

Glucocorticoids

If a woman's hyperandrogenism is caused by an adrenal disorder, low-dose prednisolone (2.5 mg daily) or dexamethasone (0.25 mg daily) can be used to suppress the adrenal production of androgens. Long-term use of these agents poses a risk of adrenal cortisol suppression, and patients should be monitored for this with periodic adrenocorticotropic hormone stimulation tests. Ideally, these patients should be co-managed with an endocrinologist.

Spironolactone

Spironolactone is useful for women who are intolerant to oestrogens, have a contraindication to oestrogen therapy or do not wish to use oral contraceptives. Spironolactone acts as a competitive androgen receptor antagonist and as an inhibitor of 5α-reductase and is effective in doses of 50 to 200 mg daily. Using it off label as monotherapy at low doses of 25 to 50 mg may improve acne and not alter the menstrual cycle. If higher daily doses are required, these are often combined with the oral contraceptive pill so that the menstrual cycle is kept regular. Treatment may be prolonged (six months or more), but dosages may be reduced once an adequate clinical response is achieved.

Dose-dependent side effects of spironolactone therapy include menstrual irregularities, breast tenderness, hyperkalaemia, headache, dizziness, drowsiness and hypotension. Side effects may be minimised if therapy is started with a low dose of 25 to 50 mg daily. As an antiandrogen, spironolactone may cause feminisation of a male fetus and, therefore, patients should not become pregnant while taking the medication. Although the monitoring of blood pressure and serum electrolytes may be required in some patients, most young and healthy patients show no abnormalities in their blood pressure and do not require laboratory tests.[34]

Other treatments

This article does not address physical therapy modalities that may be helpful in managing postadolescent acne, such as chemical peels and laser therapy. A potentially promising modality is the new 1726 nm laser, which selectively targets sebaceous glands.[35]

Conclusion

Women with postadolescent acne are a relatively common presentation in general practice. Assessment of such patients should include identifying the presence of hyperandrogenism and possible underlying causes. Hormone therapy, such as the combined oral contraceptive pill, is an effective treatment and may be combined with other acne therapies such as topical agents and oral antibiotics. MT

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A list of references is included in the online version of this article (https://mt/2025/october/supplements/dermatology-collection-vol-9-no-2).

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Postadolescent acne in women

What is the cause and how can it be managed?

JO-ANN SEE FACD; **LISA BYROM** FACD

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Vulval lichen sclerosus

Not uncommon, serious and often missed

GAYLE FISCHER OAM, MB BS, FACD, MD

Vulval lichen sclerosus is a chronic, scarring dermatosis affecting genital skin in women of all ages. This article outlines the latest evidence-based approach to diagnosis, management, surveillance and follow up, highlighting practical strategies to improve long-term outcomes.

KEY POINTS

- Vulval lichen sclerosus is a chronic, underdiagnosed condition requiring lifelong treatment and surveillance.
- Untreated disease may result in anatomical distortion and carries a 2 to 6% lifetime risk of malignancy.
- Potent topical corticosteroids are safe, effective and remain the gold standard of treatment.
- Regular follow up and patient education are critical to ensuring adherence and preventing complications.

Vulval lichen sclerosus (VLS) is an uncommon skin disease with a predilection for genital skin. Although exact prevalence is unknown, estimates suggest it affects around 3% of women over the age of 50 years.

VLS is likely underdiagnosed. This reflects both inadequate awareness among health professionals and patient reluctance to present due to embarrassment. Although severe itch typically prompts women to seek help, about 10 to 15% of patients are asymptomatic, especially early in the course of the disease. As regular genital self-examination is uncommon, the condition is often missed by patients themselves.

VLS can occur at any age, with incidence peaking in late childhood and again in the peri- and postmenopausal years. It is relatively rare in girls. In clinical practice, about 5% of cases begin before 18 years of age, 20% between 18 and 50 years of age and 75% after 50 years of age. The mean age of onset is about 55 years.

Accurate VLS diagnosis is crucial for two key reasons. Firstly, without adequate aggressive treatment, at least half of VLS cases result in significant scarring, shrinkage and distortion of the vulva, as well as stenosis of the introitus. If left untreated, the labia minora eventually becomes reabsorbed, and the clitoris becomes entrapped and buried.[1] Secondly, untreated VLS carries a 2 to 6% lifetime risk of developing squamous cell neoplasia of the vulva, including vulval intraepithelial neoplasia (VIN) and invasive squamous cell carcinoma.[2]

Adequate treatment can prevent or significantly reduce the risk of both complications and, in patients with prior malignancy, lower the risk of recurrence, thereby altering the course of the disease.[1,3]

Although there are no data on how often VLS genuinely remits, it is safest to assume that treatment should be lifelong, with ongoing observation and individually titrated treatment regimens, as would be done for any chronic condition.

Aetiology

The true aetiology of VLS remains unknown; however, there is a well-documented association with autoimmune disease, particularly Hashimoto's thyroiditis and vitiligo.[4] Cases of severe VLS have also been reported in patients treated with checkpoint inhibitors for cancers, particularly melanoma, further supporting the autoimmune theory of aetiology.[5]

Clinical presentation in adult women

VLS may occur on any part of the skin, but it is almost always primarily a genital condition, involving the vulva and, at times, extending to the perineum and perianal skin. The most common presenting symptom is a severe itch, often disrupting sleep and daily life. Pain resulting from excoriations or fissures, distressing

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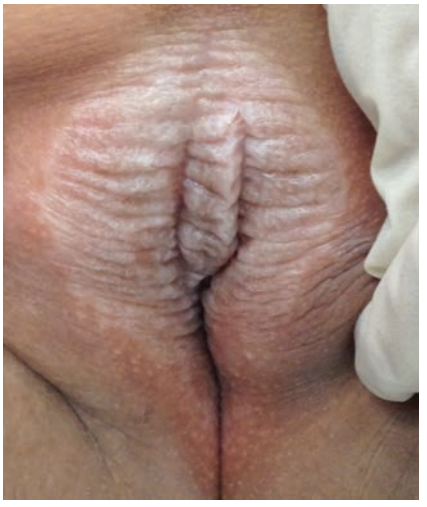


Figure 1. Classic appearance of vulval lichen sclerosus showing a well-defined white sclerotic plaque with an atrophic, wrinkled surface.
Image courtesy of the author.

clitoral hyperaesthesia and dyspareunia are also common.

In asymptomatic cases of VLS, diagnosis may occur incidentally during examination by the patient themselves or by their GP (e.g. during a routine smear test). These cases may present late, and thus be at advanced stages of disease or complicated by malignancy.

The hallmark clinical sign is a well-defined white sclerotic plaque with an atrophic, wrinkled surface (Figure 1). Although purpura and erosions may occur, they are less common. However, VLS can present with a wide range of clinical signs (Box 1).

VLS distribution is highly variable. The classic textbook description is of a figure of eight encircling the vulva, perineum and perianal skin. However, it can affect only the perianal region, clitoris, internal surface of the labia majora or labia minora, and the vaginal opening (i.e. introitus). VLS does not involve the vagina proper (that is, beyond the hymen) unless a patient has a vaginal prolapse that protrudes beyond the hymen.

In transgender women, VLS affects the same sites as in cisgender women, even when the labia majora are formed from scrotal skin, which is not typically involved in biological males. This suggests a possible role for oestrogen, although this has not been proven.

1. CLINICAL SIGNS OF VULVAL LICHEN SCLEROSUS

- Multiple white papules or macules
- Hyperkeratotic lesions
- Plaques limited to small areas, such as the tips of the labia minora, the clitoris or the clitoral hood
- Oedema on a background of pallor
- Telangiectasia, purpura or haemorrhagic blistering on a background of pallor
- Fissures
- Erosions
- Co-existing vulval psoriasis, typically appearing erythematous
- Patchy brown hyperpigmentation resembling melanosis vulvae, which can supervene

A key diagnostic clue for VLS, particularly in the later stages (but sometimes within six to 12 months from onset), is that the vulval shape is abnormal. Virtually all women develop labia minora. These are present, although diminutive, in prepubertal girls and enlarge around puberty. The size of the labia minora is highly variable, but their absence – or a clitoris that has shrunk or is buried under scar tissue – is very suggestive of VLS (Figure 2). The only other condition that will produce this is lichen planus, which can be distinguished from VLS as it is not white but erythematous and often eroded.

VLS obeys the Koebner phenomenon, which means it localises to areas of friction and trauma. This possibly explains why it is usually most recalcitrant on the perineum and the inner surfaces of the labia minora.

Extragenital lesions of lichen sclerosus may be found on any part of the skin, but are most common on the neck, buttocks, inner thigh, shoulders and wrists. Extragenital lichen sclerosus can also occur as multiple small 'confetti' lesions.

Scarring

The tendency for VLS to gradually change the shape of the vulva is well recognised

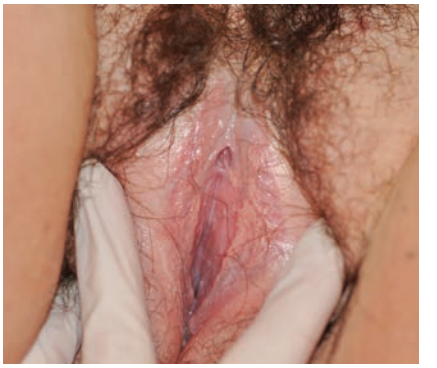


Figure 2. Lichen sclerosus showing loss of the labia minora.
Image courtesy of the author.

but underreported, and is a far more common complication than malignancy.[1] The labia minora may completely resorb or fuse beneath the clitoris. The resulting fusion line is brittle and prone to tearing during intercourse. The clitoris itself may disappear; although the nerve supply is usually preserved and most patients can still achieve orgasm, the external structure may be entirely lost (Figure 3).

The end result is a vulva with completely absent normal anatomy. In rare, severely neglected cases, the introitus may become significantly stenosed, leading to pooling of urine within the vagina and mimicking urinary incontinence. In end-stage disease, epithelial changes may no longer be visible, and gross distortion of the vulva may be the only remaining sign.

Clinical presentation in girls

When VLS occurs in girls, it typically presents during the middle years of primary education. Late diagnosis is not uncommon. The most common presenting symptoms are itching and soreness. Other symptoms and signs at presentation may include purpura, dysuria, constipation, genital erosions and extragenital lesions. As in women, about 10% of girls are asymptomatic.[6]

Dysuria and pain with defaecation leading to constipation are presentations that differ from those in women, who more commonly present with itch and dyspareunia. It is not uncommon for girls with

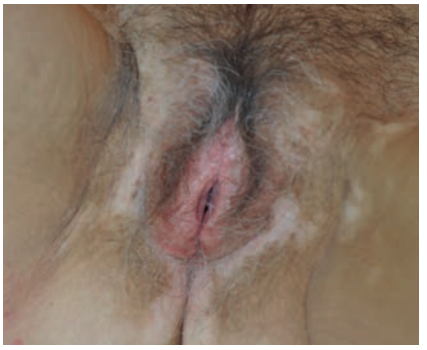


Figure 3. Lichen sclerosus showing loss of the clitoris and labia minora.
Image courtesy of the author.

VLS to be referred to urologists or gastroenterologists. If purpura is present, they may be referred to child protection units.

The clinical appearance of VLS in girls is similar to that in women, with atrophy, fusion of the labia and loss of vulval architecture. Although prepubertal girls naturally have diminutive labia minora, a well-defined clitoris should be visible if the clitoral hood is deliberately retracted during examination. Clitoral phimosis is common in girls with VLS, but it may go unnoticed unless specifically looked for.

In girls who have not received adequate treatment, the labia minora may be lost or fail to develop during puberty. The risk of irreversible architectural loss in undertreated or untreated girls is significant.[7]

Diagnosis and investigation

VLS is often a clinical diagnosis, but it is not always possible to be certain. The symptoms of VLS – itching, soreness and dyspareunia – are common to many vulval conditions. Signs include white macules, papules and plaques; thickening of the vulval skin; swelling; fissures; and scarring.

The definitive diagnostic test is a vulval biopsy. The histology is distinctive and consistent across ages and genders. It typically shows an atrophic epidermis with hydropic degeneration of basal cells and a homogenous pale zone in the upper dermis. A lichenoid infiltrate, composed mainly of mononuclear cells, may also be present in the dermis.

A biopsy should be performed to confirm the diagnosis if there is any doubt about the clinical presentation or a concern about malignancy. All patients should be offered the option of a histopathological diagnosis. A definite tissue diagnosis provides patients with the certainty that long-term maintenance treatment is necessary.

Diagnosis requires only a very small skin biopsy, taken with a 2 to 4 mm punch biopsy from the most densely white area of involvement. It is important to note that prior treatment with topical corticosteroids may render the histological findings nonspecific.

Histopathological false negatives are rare. If biopsy is not feasible or is declined by the patient, a photographic record should be kept. This can be useful if the patient relocates or changes medical practitioners. Treated disease may appear clinically normal, so it is important to have a clear histopathological record of the diagnosis before treatment commences, to help prevent future clinicians from discontinuing treatment because clinical examination findings are now normal.

In girls, a clinical diagnosis is almost always sufficient due to the difficulties of biopsy, the limited differential diagnoses and the fact that neoplastic transformation has never been reported in paediatric VLS.

Should we test for associated diseases?

When patients are diagnosed with VLS and begin researching it on the internet, they invariably encounter frightening accounts of associated diseases and may become concerned that their immune system is compromised. Several conditions have been found to occur in association with VLS (Box 2). It is reasonable to test for thyroid function and thyroid autoantibodies; however, even when autoantibodies are present, thyroid function is often normal. Nevertheless, their presence is a useful marker and warrants ongoing monitoring for the development of frank thyroid disease. Other nondirected testing is unlikely to yield clinically significant findings.

2. CONDITIONS ASSOCIATED WITH VULVAL LICHEN SCLEROSUS

- Autoimmune thyroiditis (e.g. Hashimoto's disease)
- Vitiligo
- Morphoea (localised scleroderma)
- Alopecia areata
- Pernicious anaemia
- Diabetes mellitus
- Psoriasis
- Coeliac disease
- Rheumatoid arthritis

Genetic factors

VLS can run in families, and several attempts have been made to identify a genetic association. Although no link has been found with the autoimmune-associated human leukocyte antigens (HLA) antigens HLA-A1, -B8 or -DR3, an association has been shown with the HLA class II antigen HLA-DQ7.

Although these documented HLA associations are of interest, the current evidence base is limited, and the strength of these associations remains inconclusive.

Differential diagnoses

Lichenified skin often appears white, particularly in Caucasian women. In women, the differential diagnosis includes lichenification of any origin, such as dermatitis, lichen planus and psoriasis. These conditions, although typically inflammatory and red, may appear white due to a thickened stratum corneum. Other conditions that may present with a white surface include extramammary Paget's disease, genital warts, nonpigmented seborrhoeic keratosis and VIN. Vitiligo, which can co-occur with VLS, presents as sharply demarcated white patches but lacks symptoms and textural changes. Vitiligo lesions are fluorescent on ultraviolet light examination.

Vulval lichen sclerosus and associated malignancy

Before it was realised that VLS could be effectively treated, about 60% of all vulval

keratinocyte carcinomas showed histological evidence of adjacent VLS, and it was well established that affected women had a 2 to 6% lifetime risk of developing vulval cancer. Any degree of VLS, even mild disease, carries this non-negligible malignancy risk. It is not possible to predict which patients will develop malignancy, and the interval from disease onset to cancer development has not been determined.

In adult women, but not girls, VLS is associated with vulval squamous cell carcinoma and VIN. A recently described entity, vulval aberrant maturation, may also be associated with VLS. Carcinoma in situ, known as differentiated VIN, as well as invasive carcinoma, can occur.[8] Interestingly, extragenital lichen sclerosus is not associated with malignancy.

The clinical appearance of vulval squamous cell carcinoma can include nodules, persistent fissures, hyperkeratotic plaques, nonhealing ulcers and fungating tumours. Any change in an area affected by VLS that does not resolve promptly with topical treatment must be biopsied (Figure 4).

Although vulval malignancy has not been reported in girls, squamous cell carcinoma of the vulva has been described prior to 40 years of age in patients with childhood-onset VLS. The association with genital malignancy has important implications for management: patients must be made aware of the risk, educated about warning signs and provided with regular treatment and follow up.

Malignancy after treatment

Large cohorts of patients with adequately treated VLS appear to have a malignancy rate much lower than 6%. This has led to the suggestion that the risk of malignancy is significantly reduced when VLS is diagnosed early and treated appropriately. A prospective study conducted at The University of Sydney, published in 2015, followed 507 women with VLS and compared outcomes in those who adhered to treatment and those who did not.[1] The study demonstrated that topical corticosteroid treatment that maintained objectively normal skin greatly reduced the risk of cancer. These findings confirmed earlier retrospective studies and the observations by other authors, supporting the conclusion that adequate control of VLS minimises the risk of subsequent malignancy.

Over the 10 years since the publication of The University of Sydney study, none of the patients in the original cohort who have remained under observation and on regular topical corticosteroid treatment have developed a cancer. The study also found that regular treatment significantly reduces the risk of developing new scarring and the progression of scarring existing at the time of first presentation – an observation supported by long-term follow up of the cohort.[1]

These findings suggest that the best outcomes for women with VLS are achieved through consistent efforts to obtain complete disease suppression and ongoing, careful surveillance.

Management

An Australian consensus statement on the diagnosis and management of VLS was published in 2021.[9] In adult women, VLS is a lifelong disease that is unlikely to remit. It is unknown how many patients relapse upon ceasing treatment, but given the potential consequences of untreated disease, it is safest to assume that permanent remission is unlikely. Relapse may take many months and is often silent.

This reality can be difficult for some patients to accept. It is important to reinforce the chronic nature of VLS at every visit until it is clear the patient understands and accepts the need for long-term treatment. Drawing a comparison to diabetes, a chronic but manageable condition widely understood to be incurable, can be a helpful analogy.

Even in cases where the disease appears to have remitted, long-term observation is crucial, as VLS can reactivate after many years of dormancy.

No universally accepted severity scale for VLS exists. However, a practical approach combines patient-reported quality of life impact – such as that measured by the vulval quality of life index, which was validated in Australia – with clinical observation of the degree of whiteness and scarring.[10]

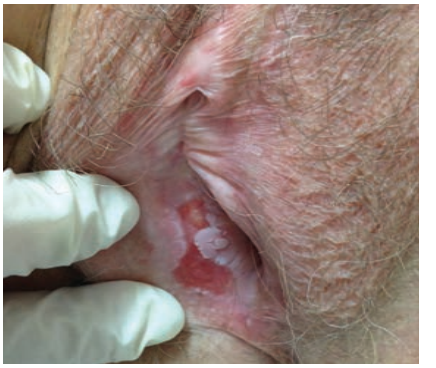


Figure 4. Eroded area in a patient with vulval lichen sclerosus, resistant to topical corticosteroid treatment, that would warrant further investigation via biopsy to rule out malignancy.
Image courtesy of the author.

The principles of management are set out in Box 3.[10]

Topical therapy

There are two phases of treatment for VLS: induction of remission and maintenance treatment.

Potent topical corticosteroids are widely accepted as the gold standard for obtaining remission in VLS. The first report of this approach, published in 1991, used clobetasol propionate 0.05%, a superpotent topical corticosteroid.[11] Prior to this, it was considered inappropriate to apply such strong corticosteroids to genital skin. As a result, treatment regimens relied on weaker topical corticosteroids, testosterone and progesterone, and VLS was considered very difficult to manage.

The 1991 study demonstrated that potent corticosteroids were both effective and safe, shifting the paradigm of VLS treatment. Since then, VLS has become one of the easiest vulval conditions to manage. Numerous subsequent studies using potent and superpotent topical corticosteroids have confirmed the safety and efficacy of this approach.

VLS is so responsive to topical

3. MANAGEMENT GOALS AND ASSESSMENT OF OUTCOMES FOR VULVAL LICHEN SCLEROSUS

Treatment goals

- Resolution of symptoms
- Improvement in quality of life
- Preservation or restoration of sexual function
- Objective resolution of clinical signs
- Prevention of complications
- Minimal treatment side effects, although some degree of erythema is acceptable

Assessment of response

- Symptom control
  - patient reported
  - absence of itch, soreness, pain, burning, dysuria
- Quality of life and sexual function
  - validated quality of life tools (e.g. vulvar quality of life index)[10]
  - sexual function assessment (e.g. Female Sexual Distress Scale or direct patient reporting)
  - in postmenopausal women, additional use of topical oestrogen may be needed to address vaginal dryness or atrophy
- Clinical signs
  - improvement in clinical appearance: reduction in whiteness, lichenification, purpura and fissures
  - monitoring for signs of scarring or disease progression
- Long-term follow up
  - ongoing surveillance for malignancy
  - assessment of sustained symptom control and prevention of anatomical changes

corticosteroids that a lack of improvement should prompt reconsideration of the diagnosis, concerns about treatment adherence or evaluation of confounding factors such as allergy or superinfection.

Most subsequent studies have also used clobetasol propionate, although more recent trials have employed mometasone furoate 0.1%.[12] Until recently, clobetasol was only available in Australia as a compounded, unapproved medication, whereas it was widely available in New Zealand. However, in practice, any potent topical corticosteroid can achieve the desired outcome. The main focus of treatment should not be on the specific product used, but on the treatment goal: to attain and maintain normal skin.

There is no single regimen that suits all patients, and clinicians should use their judgement, taking into account disease severity and patient preference for daily versus intermittent treatment. It appears, however, that regimens that work best are generally used at least three to four times per week; very intermittent regimens are less effective and more likely to be forgotten.

Induction of remission

The potency of topical corticosteroid should be selected based on the degree of hyperkeratosis (thickening) of the vulval skin. Although existing scarring at presentation may influence symptom severity, it cannot be reversed by topical treatment. In select cases, surgery may be appropriate.

Suggested regimens based on severity are listed below.

- **Severely hyperkeratotic disease** (Figure 5): superpotent topical corticosteroid (e.g. clobetasol propionate 0.05% ointment or betamethasone dipropionate 0.05% in optimised vehicle containing propylene glycol) applied twice daily until itching ceases (usually one to two weeks), then once daily until review at six weeks.
- **Hyperkeratotic to moderately hyperkeratotic disease** (Figure 6 and Figure 7): potent topical corticosteroid (e.g. betamethasone dipropionate 0.05% ointment or mometasone furoate 0.1% ointment) applied twice daily until itching ceases, then once daily until review at six weeks. Mometasone furoate may sting, and is particularly poorly tolerated by girls.
- **Mildly hyperkeratotic disease** (Figure 8 and Figure 9): moderate-potency topical corticosteroid (e.g. methylprednisolone aceponate 0.1% fatty ointment) applied daily until review at six weeks.

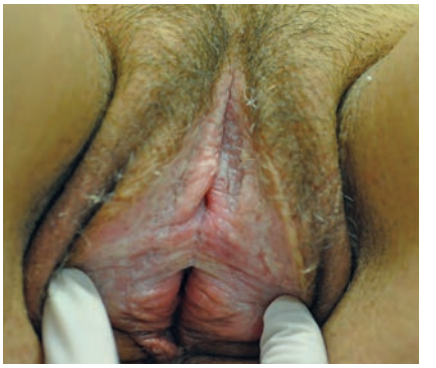


Figure 5. Very thick, white plaques seen in severely hyperkeratotic vulval lichen sclerosus.
Image courtesy of the author.

- **Very mildly hyperkeratotic (burnt out) disease:** mild-potency topical corticosteroid (e.g. hydrocortisone 1.0% ointment) applied once daily until review at six weeks.

The six-week review is critical to assess treatment response, monitor for side effects and provide emotional support. VLS is often a difficult diagnosis for most women to accept, and at this point most feel significantly better and may assume they are cured. It is important to emphasise that long-term treatment is essential to prevent cancer and scarring.

The initial potency of topical corticosteroid should be continued until both skin texture and colour return to normal. Residual hyper- or hypopigmentation may persist but are not signs of active disease. Clinically, the skin surface usually shows marked improvement.

Further reviews should be scheduled at three months and then every six months for the first two years. During this time, topical corticosteroid potency should be slowly titrated down to a moderate to mild formulation for maintenance therapy.

The aim of treatment is both symptom resolution and the disappearance of abnormal clinical signs. Symptoms typically resolve quickly, but abnormal signs – particularly skin thickening and colour changes – may take longer. Patients must therefore continue their regular treatment even after symptom resolution. Decisions

regarding management should be guided by objective clinical response. Adherence is best when treatment is incorporated into the patient's daily routine. Patients who struggle with adherence benefit from regular six-monthly reviews.

The average time to achieve normal skin is four to six months of consistent treatment. Changes such as scarring and loss of structure are not reversible with topical corticosteroid treatment, although clitoral phimosis may resolve, particularly if not severe.

Flowchart 1 shows an approach to the management of VLS.[1]

Maintenance treatment

Regimens for the long-term treatment of VLS are far less well defined than those for initial disease control, primarily due to a lack of robust data. Although many reviews and published articles state that VLS does not resolve spontaneously and requires ongoing management, there is no consensus on what this long-term treatment should involve. The main limitation of most published studies is their short follow-up duration, typically no more than three years.

There is no single long-term regimen suitable for all patients, as treatment must be tailored to the severity of the disease. Practitioners who limit their prescribing to agents only supported by published data may find their therapeutic options too narrow. It is essential to understand the relative potencies of available topical corticosteroids, noting that their availability differs between countries (Box 4).

In general, ointments are preferable to creams as they adhere better and are less likely to sting. However, patient preference must be considered, as this improves long-term adherence.

The overarching aim of maintenance therapy is to preserve normal vulval skin texture and colour. Treatment should be titrated accordingly. A typical maintenance regimen involves a moderate-potency topical corticosteroid used daily or two to three times per week – but this represents the minimum frequency of use. For forgetful patients, a routine involving a moderate-potency corticosteroid such as methylprednisolone aceponate 0.1%, applied two to three times per week, alternating with a mild-potency corticosteroid such as hydrocortisone 1% on other days, could be helpful in establishing a daily routine.

However, it is important not to follow a rigid, 'recipe-style' approach to managing VLS since there is no single regimen that suits all patients. As with any chronic disease, treatment should be titrated according to disease severity. Regimens may range from daily application of a superpotent topical corticosteroid to daily use of a mild-potency corticosteroid, but treatment should not be less frequent than once weekly.

A common technique involves initiating treatment with a potent to superpotent preparation daily until skin normalises, then stepping down: alternating superpotent with moderate-potency, then moderate-potency alone, and finally alternating moderate- with mild-potency topical corticosteroids. Treatment intensity should be adjusted to maintain normal skin colour. If the skin is white, potency should be increased; if red, it should be reduced.

Potential problems with long-term potent topical corticosteroid use on the

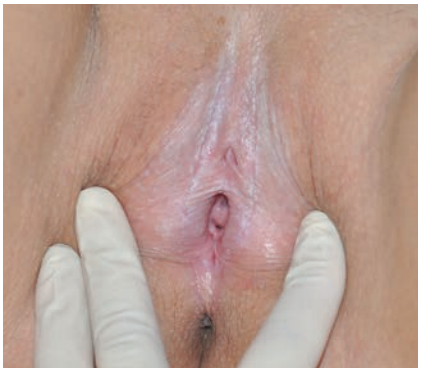


Figure 6. Thick white plaque seen in hyperkeratotic vulval lichen sclerosus.
Image courtesy of the author.

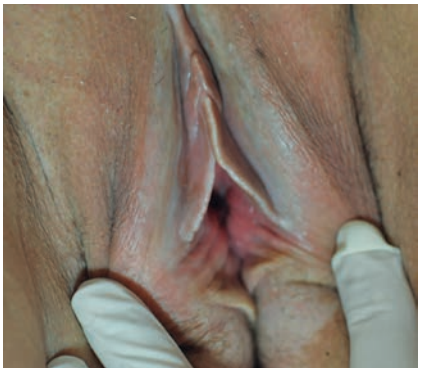


Figure 7. Moderate pallor and hyperkeratosis seen in moderately hyperkeratotic vulval lichen sclerosus.
Image courtesy of the author.

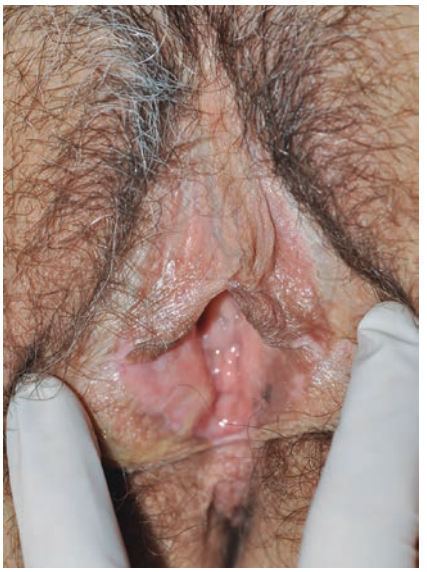


Figure 8. Minimal pallor and hyperkeratosis seen in mildly hyperkeratotic vulval lichen sclerosus.
Image courtesy of the author.

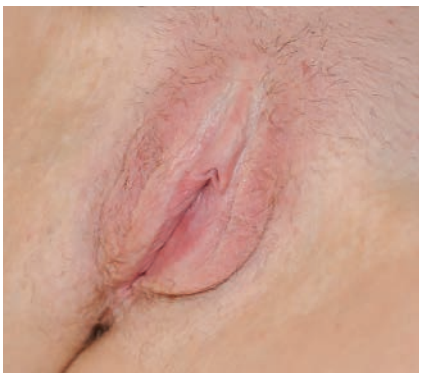


Figure 9. Mildly hyperkeratotic vulval lichen sclerosus still showing loss of labia minora.
Image courtesy of the author.

4. RELATIVE POTENCIES OF TOPICAL CORTICOSTEROIDS

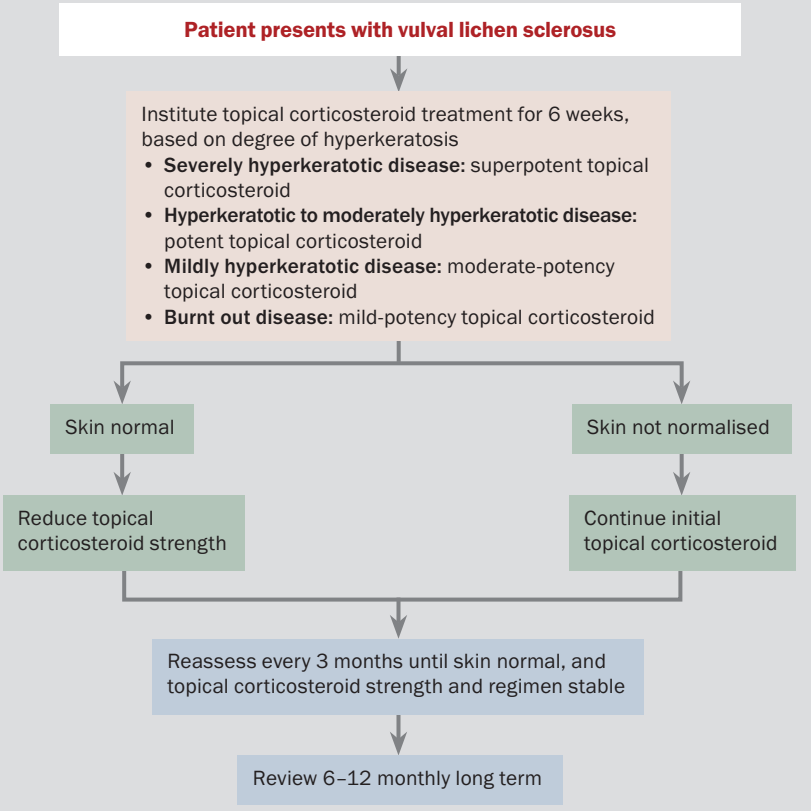
Superpotent
- Clobetasol 0.05% ointment or cream
- Betamethasone dipropionate 0.05% in optimised vehicle containing propylene glycol

Potent
- Mometasone furoate 0.1% ointment or cream
- Betamethasone dipropionate 0.05% ointment or cream
- Betamethasone valerate 0.1% cream or ointment

Moderate-potency
- Methylprednisolone aceponate 0.1% ointment, lotion, fatty ointment, cream
- Betamethasone valerate 0.05% and 0.02% cream or ointment
- Triamcinolone acetonide 0.02% ointment or cream

Mild-potency
- Desonide 0.05% lotion
- Hydrocortisone 1% ointment or cream
- Clobetasone butyrate 0.05% cream

1. AN APPROACH TO TREATING VULVAL LICHEN SCLEROSUS WITH TOPICAL CORTICOSTEROIDS[1]



genital skin include atrophy (evidenced by fragility and striae), corticosteroid-induced erythema and, rarely, superinfection, usually with Candida. However, most studies report that such side effects are rare. Corticosteroid-induced erythema can occur, particularly with stronger agents, but is usually asymptomatic and reversible with dose reduction. Importantly, it is preferable to recurrence of active disease. Therefore, concerns about corticosteroid-induced skin atrophy do not justify undertreatment of VLS.

Treatment should be reassessed every six months until stable remission is achieved, allowing titration to the lowest effective maintenance regimen. Topical corticosteroid potency is guided by the degree of hyperkeratosis, typically reflected by skin whiteness. If whiteness relapses, the strength of treatment should be increased. If atrophy or corticosteroid dermatitis (manifesting as irritability and redness) occurs, potency should be reduced. This tailored approach is successful, safe, inexpensive and outstandingly effective, and appears to reduce the incidence of malignancy to near zero, while significantly limiting disease progression and scarring. More than 90% of patients achieve complete and sustained symptom control, and among those who are sexually active, more than 90% report resolution of dyspareunia. A recent study also demonstrated significant improvement in quality of life with good disease control.[13]

It is unwise to adopt an 'as needed' approached focused solely on symptom control, even though this is supported in some guidelines. Suppressing objective disease activity – not merely relieving symptoms – should be the target outcome, as asymptomatic patients are still at risk of scarring or malignant transformation. Many patients who later developed cancer or progressive disease reported poor treatment adherence, often because symptom resolution led them to believe they were cured.

Once a patient has been in a stable remission for two years, review can occur annually. Intervals longer than this are associated with poorer outcomes due to lapses in treatment adherence. Recurrence in a patient who has finally achieved remission after years of suffering can be psychologically devastating. Each review is an opportunity to reinforce the importance and safety of maintenance treatment.

Although some authors argue that regular follow up is a burden on the health system, VLS is relatively uncommon, and the cost of even a single case of vulval cancer far outweighs that of ensuring

regular follow up in multiple patients. Ideally, competent nurse practitioners could be trained to carry out meaningful follow up and monitoring of VLS patients.

When topical corticosteroids are ineffective, not tolerated or refused

Although resistance to topical corticosteroids is rare and tolerance is generally excellent, some patients decline treatment due to safety concerns. Corticosteroid phobia remains a challenge, often reinforced by misunderstandings regarding the role of corticosteroids in dermatological conditions and risk aversion among health professionals.

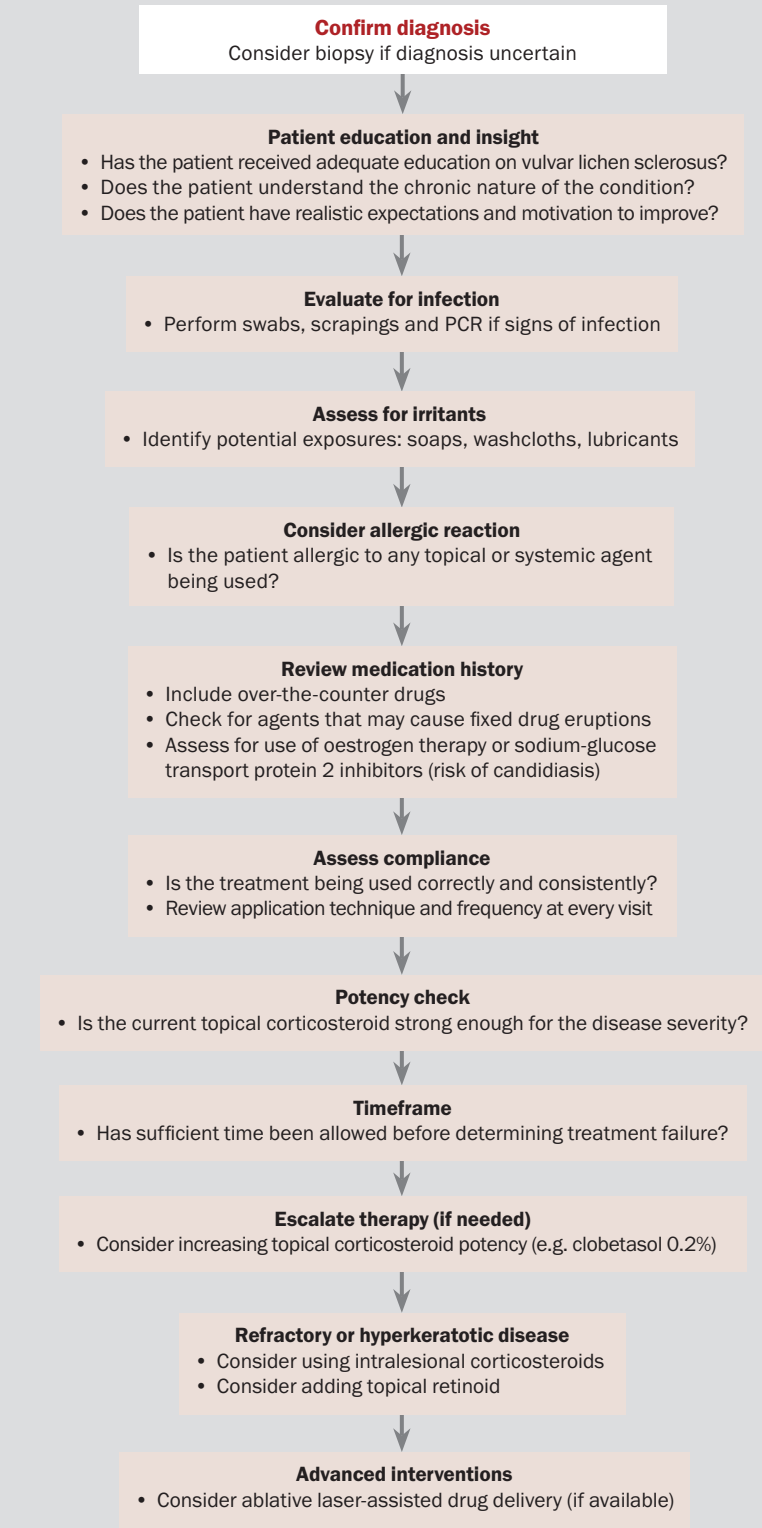
Nonresponse is most often due to misdiagnosis, as VLS symptoms are nonspecific and overlap with other vulval conditions. While white plaques with purpura or cigarette-paper wrinkling are pathognomonic, they are not always present. Other lichenified skin conditions, such as lichen planus, can also cause scarring. As such, loss of vulvar structure is very characteristic but not confined to VLS.

Occasionally, normal anatomical variation – such as a small labia minora or an inconspicuous clitoris – may be mistaken for VLS. However, structural loss alongside typical white plaques strongly suggests the diagnosis.

Ongoing symptoms may also result from concurrent infection, allergy to or irritancy caused by medication and other applied substances, continence issues, postmenopausal dryness, scarring and chronic pain conditions associated with pelvic floor dysfunction or neuropathy.[14] Nonadherence is another frequent cause, driven by anxiety, denial or misunderstanding. It is therefore important to explore patient beliefs and attitudes towards long-term treatment at every visit.

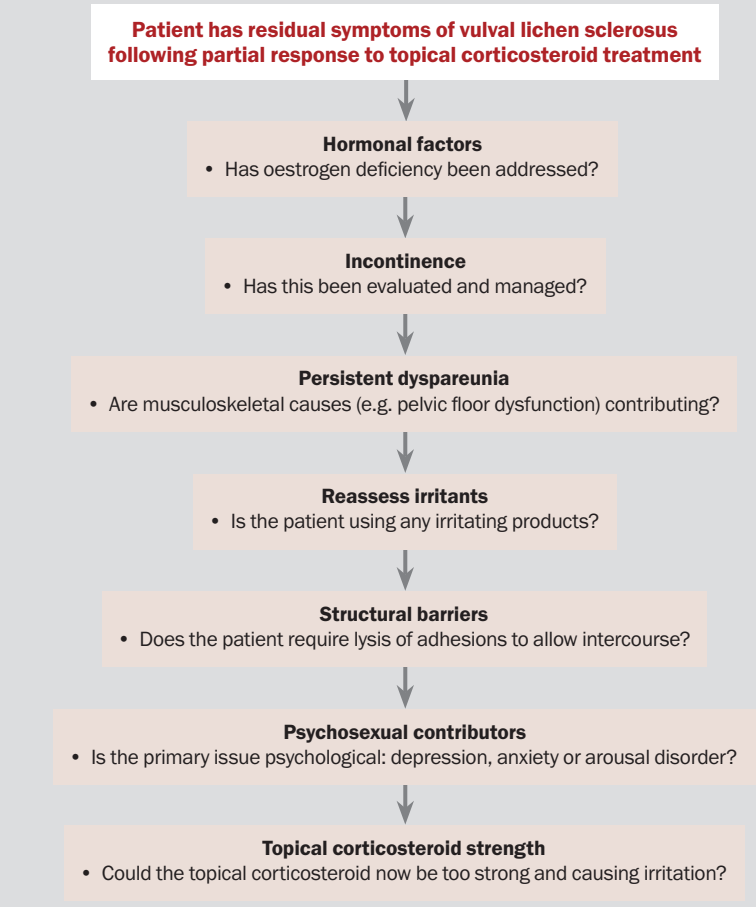
An approach to managing patients with VLS who do not respond to treatment is shown in Flowchart 2. Flowchart 3 outlines the approach to managing ongoing symptoms despite objective clinical improvement.

2. AN APPROACH TO MANAGING PATIENTS WITH VULVAL LICHEN SCLEROSUS NOT RESPONDING TO TOPICAL CORTICOSTEROID TREATMENT



Abbreviation: PCR = polymerase chain reaction.

3. AN APPROACH TO MANAGING ONGOING SYMPTOMS OF VULVAL LICHEN SCLEROSUS DESPITE OBJECTIVE IMPROVEMENT



Treatment alternatives to topical corticosteroids

Platelet-rich plasma, often misrepresented online as a stem-cell treatment, has been proposed as a treatment for VLS, but lacks evidence, is invasive and expensive. In very hyperkeratotic disease, ablative laser (laser-assisted drug delivery) may help in reducing hyperkeratosis and facilitating corticosteroid absorption, but must be used in conjunction with a topical corticosteroid.

Topical immunosuppressants such as tacrolimus and pimecrolimus have been proposed as a VLS treatment in girls and women, but concerns remain due to the premalignant nature of the condition and the potential risk of promoting malignancy. Cases of squamous cell carcinoma have been reported in women with VLS in association with the use of such topical immunosuppressants.[15] These agents offer no advantage over topical corticosteroids, are more expensive, are very likely to sting or burn and lack long-term safety data.

Topical testosterone is no longer used as a VLS treatment, due to ineffectiveness and the risk of androgenisation in girls. Similarly, topical oestrogen has no role in VLS, except for managing hypo-oestrogenic symptoms in sexually active postmenopausal women.

Systemic therapies – including methotrexate, retinoids, hydroxychloroquine and adalimumab – have been used anecdotally but are rarely needed, as most cases respond well to appropriately potent and sustained topical corticosteroid therapy.[16,17]

Management in girls

The situation in girls is less well documented than in women; however, the principles of treatment with topical corticosteroids are the same. Historically, it was believed that childhood VLS would improve or remit at puberty, but this is incorrect. Although few studies have examined long-term outcomes in adolescent girls who developed VLS in childhood, all available data casts doubt on this assumption.[18]

It is therefore essential that both parents and patients understand that resolution at puberty is unlikely, and that the condition requires long-term follow up, just as it does in women. A recently completed retrospective study of 46 girls with VLS, comparing those who were adherent with treatment with those who were not, showed that when normal skin is attained and maintained, disease progression ceases and scarring and atrophy do not occur.[6] However, any scarring that is present prior to treatment will not reverse. The most common changes are loss of the labia minora and clitoral phimosis. The latter may improve with treatment, but once labia minora have resorbed, they will not regrow.[6]

More recent research has shown that the risk of scarring is greatly increased in girls who are not adherent with treatment, particularly around puberty, when the genital area is developing adult proportions.[7] Follow up during adolescence is difficult, as many teenage girls feel embarrassed about genital examination. To avoid this, some may reassure their parents and doctors that they are asymptomatic, leading to a loss to follow up.

VLS may have a profound psychological impact on both young and adolescent girls. Many develop unvoiced fears around sexuality and reproduction, and often feel isolated or different from their peers. Fear of using tampons is common, and usually stems from concerns about their genital

area. Around puberty, when girls should be developing autonomy, many with VLS begin to resist treatment and wish to self-manage. This is a crucial developmental stage, and if control of the condition is lost, there is a risk of long-term physical and psychological consequences.

A recent retrospective study of women with a history of childhood VLS supports these concerns and highlights the need for individualised regimens and regular follow up.[19] More work is required in this area of social science to determine how best to support these young women.

Lichen sclerosus and sexual abuse in girls

Concerns about possible sexual abuse frequently arise when girls with VLS are examined, due to the presence of erosions, fissures, purpura, bleeding or scarring. There have been numerous reports of girls with classic VLS features undergoing extensive and inappropriate evaluations for sexual abuse. Although increased awareness of sexual abuse among healthcare professionals has led to more timely referrals for correct diagnosis of VLS, the additional emotional trauma inflicted on the child and family in these cases is entirely unnecessary.

Sexual abuse is unfortunately common, with retrospective studies suggesting that approximately 20 to 25% of females have experienced such abuse in childhood. However, girls who have been sexually abused rarely have clinical signs at the time of examination. A diagnosis of VLS neither supports nor rules out the possibility of abuse, and both conditions must be considered independently.

Surgical therapy

Historically, vulvectomies have been performed in women with VLS, but recurrence of the disease following surgery was common. This approach is no longer considered acceptable and is now completely contraindicated.

In girls, surgery is rarely indicated unless there is significant fusion of the labia. A variety of procedures have been employed to treat labial and peri-clitoral adhesions. Simple division of adhesions can provide an excellent outcome, provided that potent topical corticosteroids are applied daily postoperatively until healing is complete. In some cases, the corticosteroid may need to be applied using a dilator to ensure adequate contact with the affected area.[20]

Can patients with lichen sclerosus resume normal sexual activity?

In most cases, the answer is yes. Physically, particularly in younger women or those treated before significant disease progression, there is usually no barrier to resuming a normal sex life.

Women with a long history of painful intercourse often develop pelvic floor dysfunction and may benefit from physiotherapy. Others may have developed an aversion to sex prior to diagnosis and might require psychological support. Some older women decline intervention due to a lack of interest in sex after menopause. However, those who wish to become sexually active again usually do so successfully.

When to refer

Referral decisions depend on a GP's experience and confidence in diagnosing and managing VLS.

If VLS is suspected, confirmation with biopsy is ideal, for the reasons discussed above. If the GP is not comfortable performing a biopsy, referral to either a dermatologist or a gynaecologist is appropriate. Dermatologists generally have more experience and knowledge of VLS, as it is a skin condition; however, gynaecologists are also capable of performing biopsies. Although male dermatologists are typically well versed in VLS, some female patients may prefer to see a female practitioner, which can lead to deskilling in some male clinicians over time.

It is not acceptable to make a diagnosis of VLS without initiating treatment. If a dermatologist confirms the diagnosis but the patient is referred back without a clear plan, topical corticosteroid therapy should be commenced as outlined above.

GP-led long-term management of VLS could significantly reduce the burden on specialist services and improve access for new patients. Referral is appropriate when a patient is not responding to treatment despite an accurate diagnosis, or if there is suspicion of malignancy.

Some patients prefer regular specialist follow up, and if this supports adherence to treatment and provides reassurance, it is a valid reason for referral. In general, annual review is recommended, or every two years in very stable cases.

Conclusion

VLS is a potentially disfiguring and premalignant condition that responds well to early and consistent treatment. Long-term disease control hinges on regular use of potent topical corticosteroids, careful surveillance and patient education. With an individually tailored approach and ongoing care, clinicians can help patients maintain function, quality of life and avoid long-term complications. MT

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A list of references is included in the online version of this article (https://mt/2025/october/supplements/dermatology-collection-vol-9-no-2).

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Vulval lichen sclerosus

Diagnosis and management

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The use of topical corticosteroids for inflammatory dermatoses

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Topical corticosteroids have been a cornerstone in the management of inflammatory dermatoses for decades, offering potent anti-inflammatory effects while minimising the risk of the systemic adverse effects of oral corticosteroids. Carefully selecting the optimal preparation is crucial to maximise treatment efficacy and reduce complications.

KEY POINTS

- Topical corticosteroids are highly effective in treating a wide range of inflammatory dermatoses because of their anti-inflammatory, immunosuppressive and antiproliferative effects.
- The potency and vehicle of a topical corticosteroid significantly affect its clinical efficacy, with various formulations offering different advantages depending on the severity of the condition and the area of skin involved.
- Misconceptions and fears about the safety of topical corticosteroids can lead to underuse and inadequate treatment, whereas inappropriate use may cause side effects; therefore, patient education is essential.
- Proper application techniques, including using the finger-tip unit system to guide dosing and combining topical corticosteroids with emollients, can improve treatment outcomes, and occlusion therapy may enhance effectiveness in severe or resistant cases.
- Nonsteroidal treatments, such as topical calcineurin inhibitors, phosphodiesterase-4 inhibitors and Janus kinase inhibitors, may be suitable alternatives in certain cases, although cost and availability can be limiting factors.

Topical corticosteroids (TCS) are used widely to treat a range of inflammatory dermatoses, including atopic dermatitis, contact dermatitis, psoriasis, lichen planus, immunobullous disorders, morphoea, vitiligo, cutaneous lupus erythematosus, and dermatomyositis.[1,2] Their anti-inflammatory, immunosuppressive and antiproliferative effects make them highly effective in managing these conditions.[3] Despite their efficacy, misconceptions about TCS use and safety among patients and healthcare providers can lead to inadequate treatment, whereas inappropriate use of TCS may result in unwanted side effects.[1,3] Corticosteroid phobia remains a major obstacle to effective treatment.[4] Understanding how to select an appropriate TCS preparation is key to optimising patient outcomes.[1]

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TABLE 1. SUGGESTED POTENCY OF TOPICAL CORTICOSTEROIDS BY CONDITION

Potency of topical corticosteroid	Dermatitis	Other skin conditions	Notes
Mild • Hydrocortisone 0.5–2%	• Atopic dermatitis: face, eyelids, axilla, groin	• Seborrhoeic dermatitis	–
Moderate • Clobetasone butyrate 0.05% • Hydrocortisone butyrate 0.1% • Betamethasone valerate 0.02–0.05% • Triamcinolone acetonide 0.02–0.05% • Methylprednisolone aceponate 0.1%	• Atopic dermatitis: trunk, limbs • Contact dermatitis • Discoid eczema	• Seborrhoeic dermatitis • Psoriasis: face, natal cleft	–
Potent • Betamethasone dipropionate 0.05% • Betamethasone valerate 0.05–0.1% • Mometasone furoate 0.1%	• Atopic dermatitis: trunk, limbs, scalp • Severe contact dermatitis • Discoid eczema • Venous stasis dermatitis	• Psoriasis: trunk, limbs, scalp • Vitiligo • Lichen simplex chronicus, lichen planus, lichen sclerosus • Cutaneous lupus erythematosus • Pemphigus and pemphigoid	• Avoid face, neck, genitals, intertriginous areas • Initial treatment for genital lichen sclerosus is usually a potent or very potent topical corticosteroid used under specialist supervision
Very or ultrapotent • Clobetasol propionate 0.05% • Betamethasone dipropionate in optimised vehicle 0.05%	• Atopic dermatitis: palms and soles • Severe contact dermatitis	• Alopecia areata • Lichen planus, lichen sclerosus • Psoriasis (hands and feet) • Vitiligo • Granuloma annulare • Necrobiosis lipoidica • Sarcoidosis	• Avoid face, neck, genitals, intertriginous areas • Initial treatment for genital lichen sclerosus is usually a potent or very potent topical corticosteroid used under specialist supervision

This article explores the key factors to consider when selecting a TCS preparation, examines the role of different TCS vehicles and potencies, and discusses TCS safety. It also identifies potential complications associated with prolonged or improper TCS use and discusses alternative, localised treatment options.

Mechanism of action of TCS

TCS exert anti-inflammatory properties, similar to oral corticosteroids, but are delivered in a localised manner. When applied locally, they can effectively reduce inflammation, pruritus and oedema at the site of application, without the systemic side effects associated with oral corticosteroids, such as osteoporosis, weight gain, hypertension and hyperglycaemia.[3,5] TCS exert their anti-inflammatory effects through multiple actions by:

- inducing vasoconstriction, which reduces the delivery of inflammatory mediators to the affected area
- inhibiting phospholipase A2, leading to decreased production of prostaglandins and leukotrienes
- stabilising lysozymes in neutrophils, preventing degranulation, thereby limiting inflammation[2,6,7]
- altering gene transcription, thereby increasing the expression of anti-inflammatory genes
- reducing the expression of cytokines and other mediators involved in the aberrant inflammatory response.[2,6,7]

TCS also have an antimitotic effect that is thought to be particularly beneficial in patients with psoriasis.[8,9]

Selecting an appropriate TCS

When selecting a TCS preparation, various factors should be considered, including: potency of TCS and choice of vehicle; disease site and severity; and patient characteristics, such as age.[3,10]

TCS potency

The potency of a drug describes the amount of drug required to produce the desired therapeutic effect.[2] The potency of a TCS depends on its specific molecular structure (e.g. betamethasone valerate or dipropionate), formulation (cream, ointment or solution), and the extent of transdermal absorption.[1]

TCS are categorised based on potency, ranging from mild (e.g. hydrocortisone 1%) to very potent (e.g. clobetasol propionate 0.05%).[11] TCS cannot be diluted as their potency does not significantly depend on concentration.[12] Potency is reduced

TABLE 2. SUGGESTED TOPICAL CORTICOSTEROID POTENCY BY SITE FOR ATOPIC DERMATITIS AND PSORIASIS

Site	Atopic dermatitis	Psoriasis	Notes
Soles and palms	Appropriate to use potent or very potent ointment or cream	Use potent or very potent ointment or cream	–
Limbs	Moderate or potent foam, ointment or cream	Moderate or potent foam, ointment or cream	Avoid ointment if concerns about folliculitis in hair bearing areas
Axilla, groin, skin folds, genitals, neck	Low to moderate potency cream or ointment	Low to moderate potency cream or ointment	–
Face	Low to moderate potency cream or ointment, depending on severity	Low to moderate potency cream or ointment, depending on severity	Intermittent use mainly
Scalp	–	High/very high potency lotion or shampoo	Shampoo applied to wet scalp, left for 30 minutes, then rinsed out

by choosing a less potent corticosteroid and therefore the relative potencies of different molecules need to be committed to memory.[11] Ideally, clinicians should select the least potent TCS that will effectively treat the condition, considering disease site, disease severity and patient characteristics (Table 1). The penetration of TCS is increased in disease states of inflammation and desquamation because of skin barrier disruption.[13,14]

Vehicle choice

When delivering a TCS preparation, the active steroid is carried by a vehicle or a carrier (e.g. a cream or ointment base).[15] The vehicle can affect TCS bioavailability by their interaction with the skin and by changing the characteristics of the steroid.[15] TCS are available in various formulations, including ointments, creams, lotions, gels, foams and shampoos.[16,17]

- Ointments: these are mostly composed of a petroleum base, which provides superior occlusion, enhancing absorption and efficacy. Ointments are particularly useful for dry, hyperkeratotic lesions, and are effective as barrier moisturisers because they remain on the skin surface and prevent moisture loss. Less preservative is required, reducing the risk of contact allergy. However, ointments are greasy, and the degree of occlusion may increase the risk of folliculitis and heat rash.[13]
- Creams: these are composed of more equal proportions of water and oil, and tend to be easily absorbed. Creams leave less residue on the skin surface and may be preferable for areas with hair or acute exudative inflammation. Creams may contain preservatives or alcohol that may sting or cause irritant or allergic contact dermatitis.[18]
- Lotions: these contain a higher proportion of water and are useful for hairy areas, such as the scalp, as they are nonocclusive. Lotions can contain alcohol that may sting inflamed skin and cause irritation, as previously discussed, and may also have a drying effect on the skin.[19]
- Gels: these are aqueous, rapidly absorbed and nongreasy.[10]
- Foams: these preparations of TCS offer cosmetic and pharmacodynamic advantages over cream and ointment vehicles. Foams are highly effective for steroid delivery and are associated with excellent patient compliance with treatment.[20]
- Shampoos: those containing clobetasol propionate 0.05% are safe and effective for use in scalp psoriasis.[17]

Underlying skin disease

Some dermatoses, such as those outlined below, require a high-potency TCS.

- Oral lichen planus: TCS cream or ointment may be used safely and effectively inside the oral cavity.[21] High-potency TCS may be required, especially for erosive or atrophic oral lichen planus.[21] Oral candidiasis may be a complication of oral TCS use, and nystatin drops or amphotericin lozenges may be required.[21] Intralesional corticosteroid injections may also be effective.[22]
- Lichen sclerosus: first-line treatment is a potent or ultrapotent TCS, even in the genital region. In most cases, the potency can be reduced for maintenance treatment.[23] This treatment appears to be safe and effective long term and improves quality of life.[24]
- Vitiligo: may require potent or ultrapotent TCS for effective treatment.[25]
- Bullous pemphigoid: localised

disease may be managed with a potent TCS.[25]
- Cutaneous lupus erythematosus: may require a more potent TCS.[25]

Severity and disease site

The recommended potency of TCS depends on the severity and disease site (Table 2). Areas such as the face, genitals and intertriginous regions (e.g. axilla, groin) are more prone to localised adverse effects, particularly with prolonged use of potent corticosteroids, and care should be taken when addressing inflammatory dermatoses in these areas. Low-potency TCS are recommended for these areas if required to minimise localised adverse effects.[2,26]

- Palms and soles: inflammation in thicker skin areas such as the palms and soles (with a thicker stratum corneum) may require higher-potency TCS, such as clobetasol propionate, for effective penetration.[2,26]
- Eyelids: low-potency TCS, such as hydrocortisone 1%, appear to be safe for use in eyelid dermatitis. There is no evidence that periorbital application of weak TCS result in ocular complications such as glaucoma.[27]
- Face: high-potency TCS should be avoided on the face to avoid periorificial dermatitis.[28]

Combination of TCS products

Combination products consisting of a TCS combined with another active ingredient, such as an antibiotic, antifungal or calcipotriol, are also available and may be prescribed for specific indications.[12] The combination of betamethasone dipropionate 0.05% with calcipotriol appears to be more effective for psoriasis than either product alone. However, in general, the use of combination products is not considered best practice, except for the combination of 1% hydrocortisone and an antifungal product, which may be useful to treat dermatoses in the intertriginous zones where *Candida* colonisation can be a cofactor in inflammation.

Proper application of TCS

Dosage and frequency

TCS are usually used once to twice daily.[11] There may be no therapeutic benefit in applying TCS more than once daily after the first few days of application, although the risk of complications might be increased.[29] Emollients are recommended in combination with TCS because they have greater efficacy when used together compared with TCS alone.[30] Treatment should be continued until active inflammation is resolved to minimise the likelihood of rebound recurrence and exacerbation of disease.[31,32] As TCS are generally used for chronic-relapsing cutaneous dermatoses, intermittent treatment may be required for flares.[33]

There is no requirement to use TCS sparingly. This word is often printed on medication labels by pharmacists even when not written on the prescription. An adequate amount of TCS should be applied to cover the affected area.[11] The traditional guidance to 'apply thinly' or 'use sparingly' may contribute to steroid phobia, fuelled by misinformation on social media platforms, potentially leading to inadequate treatment.[34]

Finger-tip unit

Judicious use of TCS is the key to maximise the efficacy of treatment and minimise the risk of adverse effects. The finger-tip unit (FTU) system was proposed in 1991 to simply quantify TCS use.[35] One FTU refers to the amount of cream squeezed out of its tube onto the finger pad of the terminal phalanx of the index finger (from the tip to the skin fold overlying the palmar aspect of the distal interphalangeal joint). This equates to approximately 0.4 g in an adult woman, 0.5 g in an adult man and less in children.[35]

The recommended FTUs per body site are as follows:

- one palm: 0.5 FTU
- face and neck: 2.5 FTUs
- one arm: 3 FTUs
- one leg: 6 FTUs
- trunk (front and back): 14 FTUs
- entire body: about 40 FTUs.

Occlusion therapy

TCS may be applied under occlusion with a tubular bandage or plastic wrap to enhance penetration in severe or recalcitrant lesions. However, chronic use of large quantities of potent TCS used under occlusion over large surface areas can theoretically lead to systemic absorption and localised adverse reactions at the site of application.[1,26,32,36]

Complications and relative safety of TCS use

It is essential to educate patients about appropriate use of TCS. Although systemic absorption is rare, prolonged, inappropriate, use of high-potency TCS over large surface areas or under occlusion may lead to hypothalamic–pituitary–adrenal axis suppression and Cushing's syndrome.[37]

Localised adverse effects of TCS may occur in situations where TCS are used for long periods for an inappropriate condition or site. These include:

- skin atrophy
- striae
- easy bruising
- acne
- rosacea
- periorificial dermatitis
- ocular complications (periocular dermatitis, glaucoma and cataracts, when applied to the eyelids)
- hypertrichosis
- masking of fungal infections.[1,3]

As with medications in general, it is the inappropriate use of TCS that leads to complications. If TCS are used at the right potency for the site, severity and condition being treated, they are outstandingly safe.[38] They remain the mainstay of

treatment for mild atopic dermatitis and many other conditions.

Nonsteroidal treatments for cutaneous dermatoses

For patients who require long-term management of cutaneous inflammation, experience adverse effects or prefer nonsteroidal treatments, several treatments are available, as outlined below.

Topical calcineurin inhibitors

Topical tacrolimus and pimecrolimus may be used as steroid-sparing agents for sensitive areas such as the face and eyelids. These are immunosuppressant treatments that suppress T-cell activation and cytokine release.[26] Pimecrolimus 1% cream is PBS listed for treating facial and eyelid atopic dermatitis in adults and children who are at least 3 months of age, when TCS are contraindicated or fail to control the disease with intermittent use. Tacrolimus is more potent that pimecrolimus, is not PBS listed and needs to be compounded.

Topical phosphodiesterase type-4 inhibitors

Crisaborole 2% ointment is a topical phosphodiesterase-4 inhibitor that is TGA approved for mild-to-moderate atopic dermatitis in patients aged 2 years and older, but is not PBS listed. It has been shown to be well tolerated in clinical trials; however, cost may be a barrier.[39]

Topical Janus kinase inhibitors

The Janus kinase signalling pathways are key downstream mediators of inflammatory dermatoses and are an emerging target for the topical treatment of cutaneous disease. Several studies show significant promise for the use of Janus kinase inhibitors, including delgocitinib and ruxolitinib, as topical treatment for several inflammatory conditions, such as atopic dermatitis, alopecia areata and vitiligo.[40] As with phosphodiesterase-4 inhibitors, cost and availability are barriers. These are currently not commercially compounded in Australia.

If TCS are used at the right potency for the site, severity and condition being treated, they are outstandingly safe

Intralesional corticosteroids

For localised, recalcitrant lesions, such as thick psoriatic plaques, prurigo nodularis or keloid scars, intralesional triamcinolone acetonide injections may be useful.[41]

Conclusion

TCS remain the mainstay of treatment for inflammatory dermatoses because of their efficacy and rapid onset of action. However, appropriate selection of potency, vehicle and duration of use is crucial to minimise complications. Clinicians should be aware of site-specific risks, educate patients on proper application techniques and consider alternative therapies if needed. Ideally, clinicians should select the weakest possible TCS that will treat the condition; however, it is important to avoid using a subtherapeutic dose for the condition being treated. Balancing efficacy with safety through judicious TCS use and adjunctive treatments optimises long-term patient outcomes while mitigating risks associated with steroid overuse or misuse. MT

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A list of references is included in the online version of this article (https://mt/2025/october/supplements/dermatology-collection-vol-9-no-2).

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The use of topical corticosteroids for inflammatory dermatoses

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Multiple scaly plaques on the head and arm

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Test your diagnostic skills in our regular dermatology quiz. What is the cause of these scaly plaques on a background of chronically sun-damaged skin?

Case presentation

An 80-year-old Caucasian man presents for his annual full skin examination. He has scaly plaques over sun-exposed areas, including his head (Figures 1a and b) and arm (Figure 2). There is no tenderness or itch associated with the lesions.

The patient had a squamous cell carcinoma excised from his scalp two years previously. He has no history of immunocompromise or use of photosensitising medications. He denies a family history of melanoma or other skin cancer.

The patient is otherwise well and taking no regular medications. Before retirement, he worked as a builder and he still enjoys playing golf.

On examination, multiple scaly plaques are observed on the right side of the patient's face and the dorsum of his right hand and forearm. A background of sun-damaged skin is noted. There is no associated cervical, axillary or inguinal lymphadenopathy. He has Fitzpatrick skin type 2.

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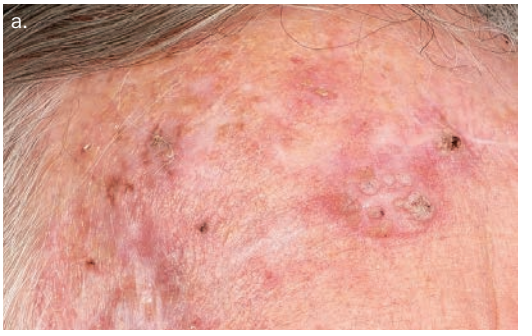

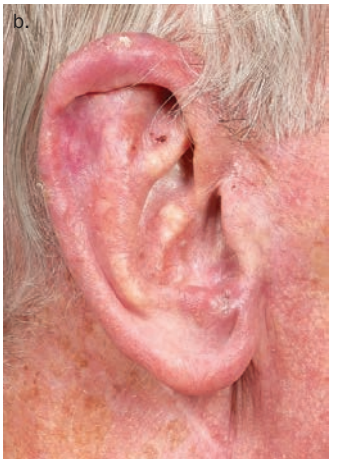

Figures 1a and b. The case patient at presentation, with multiple erythematous, scaly plaques visible on the right side of his forehead (above) and ear (right).
Images published with patient consent.

Differential diagnoses

Conditions to consider among the differential diagnoses include the following.

Squamous cell carcinoma in situ

Squamous cell carcinoma (SCC) in situ, which is also known as intraepidermal SCC or Bowen's disease, is considered to be a premalignant lesion. If left untreated, it has the capacity to progress to an invasive malignancy; however, this is rare (published rates are between 3% and 5%).[1] SCC in situ presents as a plaque that is red, scaly and crusty and often asymptomatic. Sun-exposed areas, such as the upper or lower limbs, are typical locations. A skin biopsy may be needed to establish a diagnosis.

Cutaneous squamous cell carcinoma

Cutaneous SCC (cSCC) occurs when dysplastic squamous cells invade the dermis. They typically present as progressively enlarging hyperkeratotic nodules that can be tender or indurated and are often ulcerated. The diagnosis is usually clinical and can be confirmed by biopsy. High-risk SCCs include lesions that are located

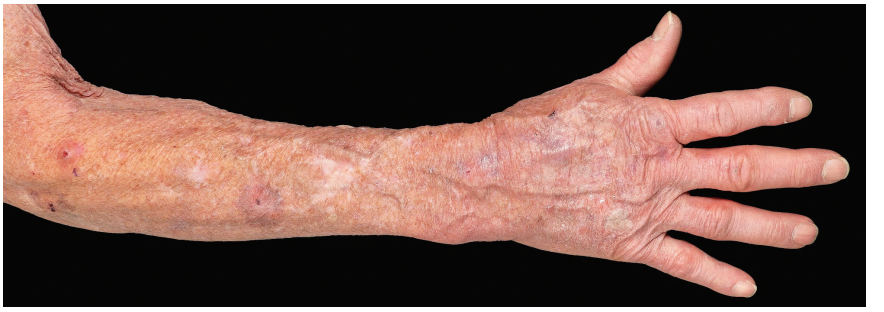

Figure 2. Lesions on the right forearm and hand (case patient).
Image published with patient consent.

on mucosal surfaces, such as the lips or genitalia, as these have a strong propensity to metastasise, particularly in patients who are immunosuppressed.[2,3]

Superficial basal cell carcinoma

Basal cell carcinoma (BCC) is the most common cancer arising from the basal cells of the epidermis. Clinically, a superficial BCC can resemble an SCC in situ as a small erythematous plaque with associated ulceration; however, a BCC may have a visible shine over its surface. Superficial BCCs tend to ulcerate and are not usually scaly. They tend to be locally invasive but metastasis is rare.

Nodular basal cell carcinoma

A nodular BCC typically presents as a slow-growing, pearly, elevated nodule that can bleed and ulcerate centrally and has a surrounding fleshy, shiny rim. They commonly arise on the face, particularly on the nose or near the eyelids, and are usually solitary. Arborising telangiectasia can be noted on dermoscopic examination, which is pathognomonic for BCC.

Seborrhoeic keratosis

Seborrhoeic keratosis is a common benign cutaneous eruption associated with increasing age, the cause for which is unknown. The clinical presentation can vary but they often occur as a warty solitary plaque or as a papule. The hallmarks of seborrhoeic keratoses include fissures, furrows forming a cerebriform pattern, white milia-like cysts and comedo-like openings, which can all be visualised on dermoscopy.

Actinic keratosis

This is the correct diagnosis. Actinic keratosis (AK), also known as solar keratosis, is a lesion that manifests following cumulative exposure to UV radiation, which causes irreversible DNA damage within keratinocytes. Australia has the highest prevalence of AK, where it is estimated to affect more than 40% of adults over the age of 40 years.[4,5] Risk is increased for Caucasian people (Fitzpatrick skin type 1 and 2) and people who live in close proximity to equatorial regions, where there is greater exposure to UV radiation.[6] Increasing age, male sex, immunocompromise, photosensitising drugs (e.g. azathioprine) and occupational exposure to arsenic are other risk factors. Solid organ transplant recipients are up to 250 times more likely to develop AK.[7]

AK has a predilection for sun-exposed areas of skin. Commonly affected sites include the face (particularly the nose), the upper and lower limbs, including the dorsum of the hands and feet, and the scalp in bald men.

The clinical appearance of AKs can vary significantly, both within and between individuals, often making this common condition a challenging diagnosis.[8] Clinically, AK can present as a macule or a

MEDICAL THERAPIES FOR ACTINIC KERATOSIS

5-Fluorouracil

5-Fluorouracil (5-FU) is a chemotherapeutic agent that has a cytotoxic effect on actinic skin, with efficacy dependent on the degree of inflammation, erosion and ulceration elicited. In a randomised control trial, a single two- to four-week course of topical 5-FU showed effective chemoprevention of cutaneous squamous cell carcinoma for one year.[13]

5-FU is available in two cream formulations: a 5% strength (applied once or twice daily) and newer 4% strength (applied once daily). The use of twice daily 5% 5-FU or once daily 4% 5-FU cream has demonstrated similar rates for complete clearance of actinic keratosis (AK), with the 4% cream showing superior tolerability.[14] The 4% cream, which is indicated for the treatment of AK of the face, ears and/or scalp, is applied for a period of four weeks, as tolerated. The 5% cream is usually applied for three to four weeks.

A liquid solution of 0.5% 5-FU in combination with 10% salicylic acid is available, which is indicated for targeted lesion and/or small-field therapy. It is applied once daily for up to 12 weeks.

Combination treatment of 5-FU plus calcipotriol has been described as being effective.[15] However, this is not routinely used in Australia as calcipotriol is not PBS listed.

Patients need to be counselled about the expected inflammatory response to 5-FU. Common side effects include soreness and pain at the site of application, itchiness or irritation, darkening or reddening of the skin, burning, crusting, increased photosensitivity and scarring. To improve tolerability, patients are usually advised to treat one anatomical segment at a time (e.g. first the forehead and temples, then cheeks and nose, then quadrants of the scalp and ears), often during winter when sun exposure is more easily avoided.

Imiquimod

Imiquimod 5% cream is a topical immunomodifying field treatment. It promotes the secretion of proinflammatory cytokines, which in turn stimulate the host immune response to induce apoptosis in precancerous cells in AK.

Imiquimod is available in a 5% cream formulation. For the treatment of AK, it is generally applied three times per week for up to four weeks initially followed by a treatment-free period (cyclical regimen), or up to 16 weeks (continuous regimen).[11,12,16] It has a similar side effect profile to 5-FU but is less cost effective. The treatment is usually applied directly to actinic skin, covering a small area of the face at a time to improve tolerability. It is generally left on overnight for approximately eight hours and washed off with water afterwards.

Photodynamic therapy

Photodynamic therapy (PDT) is a two-step process for destroying precancerous cells that involves applying a photosensitising chemical to actinic skin and then exposing the treated area to light. Daylight PDT utilising methyl aminolevulinate (MAL) is an efficacious field treatment for AK, especially for large areas of chronic actinic damage, and is generally well tolerated.[17] Conventional PDT, which utilises 5-aminolevulinic acid or MAL as a photosensitising agent and a source of red light, can be difficult to access in Australia.

Diclofenac sodium

Diclofenac sodium 3% gel (in 2.5% hyaluronic acid) is a topical NSAID that can be used for AK treatment. It is applied twice daily for up to three months.[18] This treatment has limited use in clinical practice due to poor efficacy compared with the treatments discussed above.

hyperkeratotic palpable plaque or papule on a background of normal skin or on skin that has a confluent erythematous appearance due to chronic sun damage. There is variation in the colour of AKs – lesions can be flesh-coloured, inflamed or erythematous or, rarely, associated with pigment.[9] The consistency of the lesions can also vary, being associated with a slight scale, often resembling a wart in appearance.

Clinically, there is thought to be a continuum between actinic keratosis, SCC in situ and cSCC; however, progression is uncommon. The risk of malignant transformation of AK to SCC within one year is reported to be less than 1 in 1000.[10] Untreated, the majority of AKs remain stable or regress.[9,10] On examination, SCC in situ tend to be more plaque-like and prone to bleeding than AKs. Tenderness is uncommon for AK but cSCC are often tender.

Management

Evidence-based treatment for AK is informed by Cancer Council Australia's *Clinical Practice Guidelines for Keratinocyte Cancer* and the American Academy of Dermatology's *Guidelines of Care for the Management of Actinic Keratosis*.[11,12] Both physical and medical therapies are used to treat AK. The main goals are to treat symptoms and to improve cosmesis.

Cryotherapy, ideally with a liquid nitrogen spray, is a practical and efficient treatment option for AK.[12] It is important to note that lesions will recur as a rule. Routine excision of AK is not generally recommended.

Medical therapies for AK are listed in the Box.[11-18] For patients whose lesions are widespread and resistant to previous treatment, a field therapy will be the treatment of choice. Addition of a keratolytic agent (e.g. 10% salicylic acid) can improve penetration.

In addition, patients should be counselled about the need for sun protection. This includes wearing long-sleeved, dark-coloured clothing with a close weave and broad-brimmed hats and applying sunscreen. Patients should be informed that regular use of sunscreen not only prevents the development of AK, but also enhances the remission of existing lesions.[19]

Outcome

For the case patient, the diagnosis of AK is consistent with his history of significant cumulative sun exposure. He recalls blistering sunburns as a child growing up in Victoria, and he had an outdoor occupation and sporting hobby. At the time of this presentation, his AKs had been treated with cryotherapy over a period of years, but the lesions were recurring and increasing in number.

As the patient's AKs were resistant to previous cryotherapy and he had widespread actinic damage, a field therapy was selected. He commenced treatment with 5% 5-fluorouracil, to be applied once daily for four weeks during the winter months, and advised to treat one area at a time (first the forehead and temples, then the cheeks and nose, then the quadrants of the scalp and ears, and then the right hand and arm), to eventually cover all areas of concern. He was counselled about the expected inflammatory response and need to avoid sunlight exposure because of the photosensitising nature of the cream. He was also given advice about sun protection, including the regular use of SPF50+ sunscreen, to optimise his clinical response, and encouraged to continue his annual skin-cancer surveillance. MT

References

A list of references is included in the online version of this article (https://mt/2025/october/supplements/dermatology-collection-vol-9-no-2).

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Multiple scaly plaques on the head and arm

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A man with a painless papule on the leg

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Test your diagnostic skills in our regular dermatology quiz. What is this solitary lesion overlying the leg?

Case presentation

A 40-year-old man presents with a one-year history of a solitary papule on his leg (Figure 1a). The lesion is not tender but is mildly pruritic. The area has not been subjected to infection or trauma. He has no history of skin cancer but is concerned that the lesion may be malignant.

On examination, a centrally pink papule with a pigmented periphery is observed on the lateral aspect of the right leg. The lesion measures 8 mm in diameter, is firm on palpation and dimples inwards when lateral pressure is applied. The patient has Fitzpatrick skin type I.

Dermoscopic examination shows a white scar-like centre with a delicate peripheral pigment network (Figure 1b).

Differential diagnoses

Conditions to consider among the differential diagnoses for a patient presenting with a solitary papule on the leg include the following.

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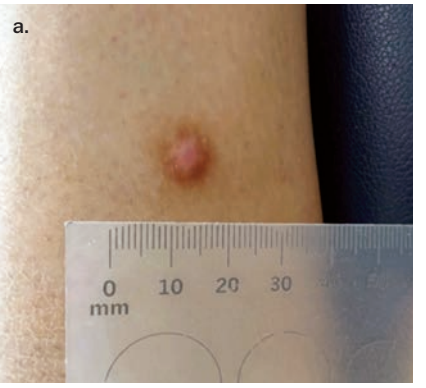

Figure 1a. The papule with a pink centre and pigmented periphery (case patient).

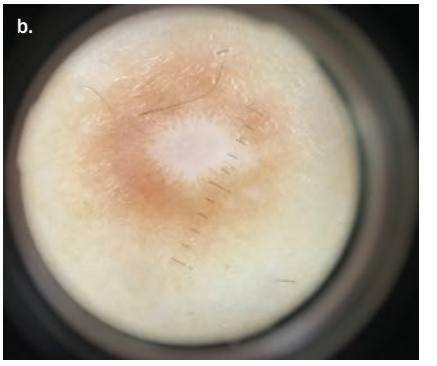

Figure 1b. Dermoscopic examination of the lesion shows a white scar-like central area with delicate peripheral pigment network.

Acquired melanocytic naevus

Acquired melanocytic naevus is a pigmented mole that appears after birth. It is the most common benign neoplasm, usually arising in the first three decades of life and occurring in greatest number in individuals of Caucasian background.[1] Predisposing factors include lighter skin type and sun exposure, as well as a family history of high naevus count.[2-4]

Acquired melanocytic naevus is thought to be caused by mutations in the *BRAF* or *NRAS* genes, which activate the mitogen-activated protein kinase pathway leading to an increase in melanocyte proliferation.[5] *BRAF V600* mutations have been found in over 80% of acquired melanocytic naevi.[5,6]

Acquired melanocytic naevus can be classified as common or dysplastic. There are three developmental stages, which are described according to the location of melanocyte nests:[7,8]

- junctional (proliferating melanocytes in the dermal-epidermal junction), which present as brown-black macules
- compound (melanocytes spanning the dermis and dermal-epidermal junction), which present as slightly raised, brown ovoid or round papules
- intradermal (melanocytes entirely within the dermis), which present as soft, light brown or flesh-coloured, dome-shaped papules with a smooth or papillomatous surface.

Typically, but not always, an acquired melanocytic naevus will progress through these three stages before senescence and involution later in life.[8]

For the case patient, the lesion was firm on palpation and showed a scar-like centre on dermoscopy. These features were not consistent with an acquired melanocytic naevus.

Nodular basal cell carcinoma

Basal cell carcinoma (BCC) is a malignant neoplasm of the basal cells. BCC is the most common type of skin cancer in Australia, accounting for up to 80% of nonmelanoma skin cancers, and its incidence is steadily increasing.[9,10] The metastatic potential of BCC is very low and estimated to be between 0.05 and 0.1%.[11-13]

The pathogenesis of BCC involves aberrance of the Hedgehog signalling pathway and point mutation in the *TP53* gene, which promotes neoplastic growth.[14] The most significant risk factors include exposure to

UV radiation, lighter skin type and increasing age.[15,16] UV radiation is particularly important because of its mutagenic effects on keratinocyte progenitor cells.[17]

A BCC can arise anywhere on the body. The vast majority occur in sun-exposed areas, with 80% of lesions arising on the head and neck, but they are not uncommon on the arms and legs.[18] Nodular BCC is the most common subtype (50 to 80% of BCCs) and presents as a papule or nodule with a pearly appearance and rolled edge.[17-19] They are occasionally pigmented, with the most commonly observed pigment structures on dermoscopic examination being large blue-grey ovoid nests; usually arborising telangiectasias are visible on dermoscopy.[20]

For the case patient, the lesion did not have a pearly appearance or a rolled edge. The pigment was not that of blue-grey ovoid nests typical of a pigmented BCC and there were no arborising telangiectasias on dermoscopic examination.

Amelanotic melanoma

Amelanotic melanoma is a rare subtype of melanoma, comprising approximately 8% of all melanomas.[21] Lacking the pigment classically associated with melanomas, they often masquerade as more benign entities.[22,23] Although visually deceptive, amelanotic melanomas are malignant tumours of melanocytes and can occur as any histological subtype of melanoma.[24] Risk factors are generally similar to those of melanoma (a history of sun exposure and a lighter, sun-sensitive phenotype); however, amelanotic melanomas are usually diagnosed in individuals over 50 years of age.[24]

The mechanism causing the lack of pigment in amelanotic melanoma remains unclear, but a few theories have been suggested. One theory is that amelanotic melanoma may be a poorly differentiated subtype of melanoma.[25] Another theory is that amelanotic melanoma may result from de-differentiated melanocytes that have lost their normal phenotype.[26] A third theory is that amelanotic melanoma may arise from multipotent melanocytes, resulting in cells that retain their melanocyte identity but form different phenotypes.[27]

Amelanotic melanomas usually present as pink, erythematous or flesh-coloured macules, papules or nodules. Dermoscopy is an important tool in identification because the vasculature offers valuable diagnostic clues – a predominance of central vessels, dotted vessels, linear irregular vessels or polymorphic vessels are concerning for amelanotic melanoma.[28,29] Other concerning dermoscopic features include milky-red areas and, if the melanoma is hypomelanotic rather than amelanotic, it may have a blue-grey veil.[28] A diagnosis of amelanotic melanoma is made on the basis of histological and immunohistochemical findings.

For the case patient, the lesion does not demonstrate any of these features of amelanotic melanoma macroscopically or under dermoscopic examination.

Dermatofibrosarcoma protuberans

Dermatofibrosarcoma protuberans (DFSP) is a rare, locally aggressive, cutaneous soft tissue sarcoma. It is predominantly diagnosed in adults between 30 and 50 years of age.[30]

DFSP is most often located on the trunk or limbs.[31] The lesion starts as an asymptomatic, pink or skin-coloured indurated plaque that slowly enlarges and progresses to a red-brown or violaceous nodule.[32] On palpation, it is firm and attached to the underlying subcutaneous tissue.

Histologically, DFSP is comprised of spindle-shaped cells in a dense but uniform arrangement.[33] Immunohistochemistry greatly assists diagnosis, as CD34 positivity occurs in the majority of DFSP lesions.[34]

For the case patient's lesion, the history does not support a diagnosis of DFSP because it has shown stability in a papular form since onset.

Dermatofibroma

This is the correct diagnosis. A dermatofibroma, also known as a cutaneous fibrous histiocytoma, is a benign lesion that occurs most frequently on the lower limbs in adults.[35] It does not usually cause symptoms but can occasionally be pruritic. The precise aetiology is not known, but it is postulated to be a local tissue response to trauma or inflammation, such as an insect bite or a traumatised hair follicle.[36]

Dermatofibroma usually presents as a solitary pink, tan or brown papule or nodule.[32] It is firm on palpation and produces a positive 'dimple sign' (downward dimpling upon application of lateral pressure on either side of the lesion). The most common dermoscopic findings are a scar-like white centre and a surrounding delicate pigment network.[35] Histologically, dermatofibromas are composed of a nodular proliferation of fibroblasts and myofibroblasts in the dermis, with frequent hyperpigmentation and hyperplasia of the epidermis.[32,36] If there is concern regarding the diagnosis, a biopsy can be performed to exclude the conditions discussed above.

For the case patient, the history, examination and dermoscopic findings of the lesion are consistent with a diagnosis of dermatofibroma.

Management

Dermatofibromas do not require intervention. For patients who have cosmetic concerns about their lesion, surgical excision can be offered, but they should be made aware of the possibility of recurrence and postsurgical scarring that can be more extensive and noticeable than the initial lesion. Other interventions, such as shave biopsy, cryotherapy and laser treatments, have been used, but the results have been variable and recurrence rates will likely be higher than for surgical removal.[37]

Outcome

The case patient was diagnosed with a dermatofibroma. He was reassured about the benign nature of the lesion, which did not undergo intervention. At follow-up appointments, the lesion was observed to be stable in size and appearance and did not cause the patient any issues. MT

References

A list of references is included in the online version of this article (https://mt/2025/october/supplements/dermatology-collection-vol-9-no-2).

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A man with a painless papule on the leg

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A boy with an eruption of erythematous facial papules

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Test your diagnostic skills in our regular dermatology quiz. What is the cause of this skin eruption in a healthy boy?

Case presentation

A 12-year-old boy presents with an eruption of erythematous papules around his mouth and nostrils that began several months ago. At the time of presentation, he had been treating the lesions with topical triamcinolone acetonide, which led to initial improvement but worsening each time he stopped applying it.

The patient is otherwise well. He lives with his family, who are all well and do not have a history of relevant skin disease.

On examination, multiple erythematous, monomorphic papules are observed with a perioral and perinasal distribution (Figures 1a and b). There are no visible comedones.

Differential diagnoses

Conditions to consider among the differential diagnoses include the following.

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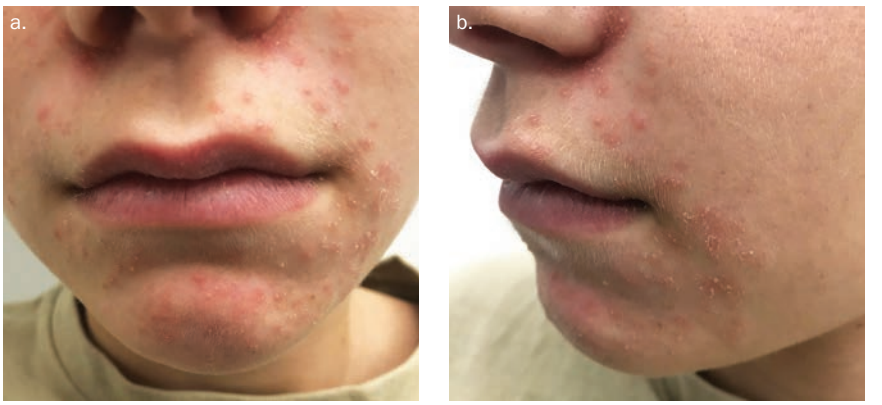

Figures 1a and b. The case patient at presentation. Frontal (a, left) and side (b, right) views of the eruption of erythematous, monomorphic papules in the perinasal and perioral areas.
Images published with patient consent.

Acne vulgaris

Acne vulgaris is a chronic inflammatory condition of the pilosebaceous unit. It predominately affects adolescents and young adults, with a global prevalence of 85% in individuals aged 12 to 24 years.[1] The aetiology is multifactorial, involving androgen-driven sebaceous hyperactivity, follicular hyperkeratinisation, dysbiosis with *Cutibacterium acnes* and an inflammatory cascade that is mediated by innate immunity.[2] A genetic predisposition, hormonal fluctuations and psychological stress are contributing factors.[3]

The lesions of acne vulgaris are divided into noninflammatory (open and closed comedones) and inflammatory (papules, pustules, nodules and cysts) groups, which are located primarily on the face, chest and back.[4] The presence of comedones is a requirement for the diagnosis. Severe disease may lead to scarring and hyperpigmentation. There may be a significant psychosocial impact, including anxiety and depression.

A diagnosis of acne vulgaris is primarily clinical and based on lesion type, distribution and severity. Acne that is secondary to other causes, such as drug-induced acne and hormonal acne (e.g. associated with polycystic ovary syndrome), must be excluded in clinically relevant contexts.

This was not the correct diagnosis for the case patient, for whom no comedones were seen on examination.

Rosacea

Rosacea is a chronic inflammatory skin condition with centrofacial distribution. It is predominantly seen in women aged 30 to 60 years, particularly those of Celtic and northern European descent and skin phototype I or II, but all individuals may be affected, regardless of age, ethnicity or sex.[5] The aetiology of rosacea is multifactorial, involving an interplay of genetic predisposition, immune system dysregulation, vascular hyper-reactivity and environmental triggers.[6] The exacerbating factors include exposure to ultraviolet radiation, heat, alcohol, spicy foods, hot beverages and psychological stress, as well

as certain medications, such as prolonged use of topical corticosteroids.[6]

The clinical presentation of rosacea varies but generally includes transient or persistent centrofacial erythema, telangiectasia, and inflammatory papules and pustules that are localised to the cheeks, nose, forehead and chin.[7,8] In advanced disease, patients may develop phymatous changes and/or ocular involvement.

The diagnosis of rosacea is primarily clinical, based on the characteristic distribution and morphology of lesions and a history of centrofacial flushing.[9] Dermoscopy may reveal dilated blood vessels on a background of erythema. Early recognition and management are crucial to prevent progression and minimise the impact on quality of life.

This was not the correct diagnosis for the case patient, who did not have a history of centrofacial erythema, and no telangiectasia were seen.

Allergic contact dermatitis

Allergic contact dermatitis is caused by a delayed (type IV) hypersensitivity reaction. Initial sensitisation occurs when there is direct skin contact with an offending substance that leads to activation of T lymphocytes, which trigger an eczematous inflammation of the skin when subsequent exposures occur.[10] It is estimated that at least 20% of the general population have a contact allergy to at least one environmental allergen, with the prevalence being significantly higher in women than men.[11] Common allergens include nickel (e.g. in costume jewellery), fragrances in perfumes and cosmetics, and accelerants used in the manufacture of rubber products (e.g. in gloves, shoes).[11]

Allergic contact dermatitis has a variety of different morphologies, including erythema, oedema, pruritic vesicles, blisters and an eczematous eruption. Chronic or repeated exposure may lead to lichenification, scaling and fissuring.[12] It most frequently affects areas that are in direct contact with environmental allergens (hands, feet, lips). However, although the eruption is generally confined to areas of exposure, it may not remain limited to the initial site of contact.

The diagnosis of allergic contact dermatitis is based on clinical examination and supported by a history of exposure to a putative allergen. Patch testing may be used to identify the cause – this involves applying allergens to the skin and observing for reactions after 48 to 96 hours.[12]

This was not the correct diagnosis for the case patient. Although topical triamcinolone may cause allergic contact dermatitis, the morphology of his eruption (monomorphic papules) was not consistent with this diagnosis.

Seborrhoeic dermatitis

Seborrhoeic dermatitis is a common inflammatory skin condition affecting individuals of all ages and ethnicities.[13] Its aetiology is multifactorial, involving genetic predisposition, immune dysfunction and hormonal factors. Colonisation of the skin by *Malassezia* species, a commensal yeast, is believed to be pivotal in the pathogenesis, triggering an inflammatory response that leads to flares.[14] There are many postulated triggers, including cold weather, stress, nutritional deficiencies (B-group vitamins, essential fatty acids, vitamin D, zinc), hormonal fluctuations, immunosuppression, certain medications (androgenic medications, antipsychotics, immunosuppressants, lithium) and some neurological disorders, such as Parkinson's disease and epilepsy.[15,16]

Seborrhoeic dermatitis presents as poorly defined, erythematous, scaly patches with little or no pruritus.[17] In adults, the condition mainly affects sebaceous gland-rich areas, such as the face (especially the eyebrows, glabella, nasolabial folds and area around the ears), scalp, axillae, chest and skinfolds. In infants, it usually affects the scalp (cradle cap), axillae and groin. Scalp involvement is characterised by greasy, thin, yellowish scales that may adhere to hair shafts. There is no associated scarring or permanent hair loss.

Diagnosis is based primarily on clinical characteristics. Trichoscopy reveals thinner, yellowish scales and diffuse erythema.[18] If the diagnosis is unclear, a scalp biopsy may be performed and will typically reveal parakeratosis, spongiosis and mild perivascular infiltrates; however, this is seldom required.[19]

This was not the correct diagnosis for the case patient. Although his skin eruption was distributed in the nasolabial folds, its morphology was not consistent with seborrhoeic dermatitis.

Periorificial dermatitis

This is the correct diagnosis. Periorificial dermatitis is a common inflammatory facial dermatosis characterised by clusters of erythematous or skin-coloured papules, pustules and scaling. The perioral region is the most likely site of distribution, often with sparing of the vermilion border; however, the periocular and paranasal skin can also be affected.

Although the exact cause of periorificial dermatitis remains unknown, several contributing factors have been identified. One of the most recognised triggers is prolonged or frequent use of topical corticosteroids, which can disrupt the skin barrier function, induce vasodilation and alter local microbial flora – all of which promote inflammation.[20] Certain facial creams, moisturisers and fluoridated toothpaste have been implicated, either through irritation or occlusion.[21,22] Dysbiosis of the skin microbiome, including overgrowth of *Candida* species, may contribute to inflammation and skin barrier dysfunction.[23,24] Environmental and lifestyle factors, such as exposure to ultraviolet radiation, psychological stress and improper skincare practices (e.g. overuse of harsh facial cleansers) are other contributors.[23]

Periorificial dermatitis is most common in women aged 20 to 25 years but can affect all people, regardless of age or sex.[25] Its prevalence has been rising in recent years due to the increasing popularity of cosmetic products.[26] Paediatric cases are often associated with inhaled corticosteroids used for managing asthma.[27]

The diagnosis of periorificial dermatitis relies primarily on clinical features. A

thorough patient history is essential and should include details about the use of topical or inhaled corticosteroids, skincare products and toothpaste. For cases that are atypical or unresponsive to treatment, a skin biopsy may be considered. Histopathology findings are nonspecific and often include perifollicular and perivascular lymphohistiocytic inflammatory infiltrate with sparse plasma cells and occasional giant cells.[28,29]

Management

The effective management of periorificial dermatitis will require a multifaceted approach, beginning with the elimination of known triggers. Topical corticosteroids must be discontinued – this is essential for long-term improvement. Abrupt withdrawal often leads to a temporary worsening of symptoms, and gradual tapering may be necessary for severe dependence.[28]

For mild cases of periorificial dermatitis, topical treatments such as metronidazole 0.75% gel or cream, clindamycin 1% lotion or solution, or pimecrolimus 1% cream may be effective. These agents reduce inflammation, promote skin barrier repair and address potential microbial dysbiosis.[28]

For moderate or severe cases, systemic antibiotics, such as doxycycline 50 to 100 mg once or twice a day, minocycline 100 mg once or twice a day, or erythromycin 250 to 500 mg daily, may be used for their anti-inflammatory and antimicrobial properties. Treatment typically lasts for 12 weeks.[30,31]

Modifications to skincare routines may be necessary. Long-term successful management requires patient education about using gentle noncomedogenic cleansers and maintaining the skin barrier with moisturisers. Avoiding known triggers, heavy cosmetics and fragranced skincare products is crucial.[25]

With timely and appropriate treatment, periorificial dermatitis typically resolves within weeks to months. However, recurrence is common, particularly if triggers such as topical or inhaled corticosteroids or irritants are reintroduced. The overall prognosis is excellent.[31]

Outcome

The case patient was given a clinical diagnosis of periorificial dermatitis. He was advised to stop applying topical triamcinolone acetonide, which is too potent for use on the face. Although he had had an initial therapeutic response, he then experienced a withdrawal flare, which is a hallmark of periorificial dermatitis.

The diagnosis was explained and the boy was commenced on oral doxycycline 50 mg daily for 12 weeks. He was educated about the importance of avoiding strong corticosteroids on his face and given information about skincare measures to protect the barrier function. On review 12 weeks later, he had no signs of periorificial dermatitis. MT

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A unilateral facial rash with ocular involvement

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Test your diagnostic skills in our regular dermatology quiz. What has led to the development of this young woman's severe skin eruption?

Case presentation

A 24-year-old woman presents with a three-day history of a painful eruption on the right side of her nose and upper lip that extends onto her cheek (Figure). The rash initially appeared as small red papules but has progressed to clustered vesicles, bullae with crusting and ulceration. She reports severe burning pain, increasing facial swelling and heightened sensitivity to light.

The patient describes a low-grade fever of 37.5°C with malaise and a 'tingling' sensation on the affected side of her face three days before the rash appeared. She has a history of rheumatoid arthritis, which is treated with methotrexate.

On examination, an extensive unilateral rash of vesiculopustular lesions and areas of necrosis is observed that particularly affects the upper lip and perinasal and periorbital regions. There is mild conjunctival injection but no significant ocular changes.

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Differential diagnoses

Conditions to consider among the differential diagnoses include the following.

Autoimmune blistering diseases

Autoimmune blistering diseases are a group of rare disorders in which the immune system attacks proteins that are essential for maintaining skin and mucosal integrity, resulting in blister formation. There are two main categories: intraepidermal blistering (such as pemphigus foliaceus and vulgaris) and subepidermal blistering (such as bullous pemphigoid and epidermolysis bullosa acquisita).[1] The incidence of autoimmune blistering diseases varies worldwide, with bullous pemphigoid being the most common, particularly among individuals over 60 years of age.[2] Pemphigus vulgaris is more frequently seen in younger individuals, with a higher prevalence in Mediterranean and Middle Eastern populations.[2]

Clinically, autoimmune blistering diseases present with pruritus, blisters and erosions affecting the skin and mucous membranes. They have predilections for different body sites, with blisters forming on the skin or mucous membranes lining the mouth, nose, throat, eyes or genitals, depending on the disorder. Patients may experience pain and secondary infections, leading to impaired quality of life.

The aetiology of autoimmune blistering diseases involves autoantibodies targeting structural proteins like desmogleins (in pemphigus disorders) or hemidesmosomal proteins (e.g. in bullous pemphigoid). The onset of disease can also be triggered by genetic predisposition, environmental factors and certain medications – these include dipeptidyl peptidase-4 inhibitors, PD-1/PD-L1 inhibitors, loop diuretics and penicillin and its derivatives, as well as certain antibiotics.[3,4] Investigations may include histopathology, direct immunofluorescence testing and serum antibody assays.

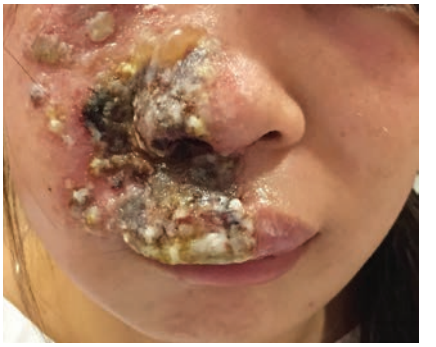

Figure. The case patient at presentation, with a rash of vesiculopustular lesions and areas of necrosis on the right side of the face.
Image published with patient consent.

An autoimmune blistering disease was a less likely diagnosis for the case patient, whose rash was unilateral with a dermatomal distribution and had evolved rapidly. These diseases typically present with more widespread blisters or erosions.

Folliculitis

Folliculitis, an inflammatory condition of the hair follicle, is prevalent worldwide (particularly in areas with a warm, humid climate) and can affect any individual, regardless of age or gender. It is more common in patients with a history of diabetes and obesity and is often linked to prolonged use of antibiotics and immunosuppression, as well as shaving and wearing of occlusive clothing.[5]

Folliculitis presents clinically as small, erythematous pustules or papules centred around hair follicles, often accompanied by

itching or tenderness. It can affect any hair-bearing area of skin, most commonly on the chest, back, buttocks, arms and legs. Folliculitis can be classified as superficial or deep, depending on the extent of follicular involvement, and severe cases may progress to abscess formation or scarring. Common forms include bacterial and fungal folliculitis and pseudofolliculitis barbae.[6] Bacterial folliculitis is most often caused by *Staphylococcus aureus* but Gram-negative organisms can be implicated. Fungal folliculitis is usually caused by *Malassezia* species. Pseudofolliculitis is caused by ingrowing hairs that re-enter or remain trapped within the skin, leading to an inflammatory response that is characterised by papules and pustules; it commonly occurs in areas that are subjected to frequent hair removal, such as the face, neck and groin.

The diagnosis of folliculitis is primarily clinical. However, bacterial cultures or fungal scrapings may be required for confirmation in refractory cases.[6,7]

This was not the correct diagnosis for the case patient. The severity of her vesiculopustular lesions and their distribution (unilateral and following a dermatomal pattern rather than folliculocentric) were more consistent with a different disease.

Allergic contact dermatitis

Allergic contact dermatitis is caused by a delayed (type IV) hypersensitivity reaction. Sensitisation occurs when an allergen activates T lymphocytes, which trigger inflammation on subsequent exposures.[8] At least 20% of the general population have a contact allergy to at least one common environmental allergen, with the prevalence being significantly higher in women.[9] Common allergens include fragrances, nickel and rubber accelerants.[10]

Allergic contact dermatitis results in eczematous inflammation that is often confined to areas of exposure, particularly the hands, feet and lips, but the eruption may not remain limited to the initial site of contact. Patients may present with erythema, oedema, pruritic vesicles and blisters. Chronic or repeated exposure can lead to lichenification, scaling and fissuring.[11]

Allergic contact dermatitis is generally a clinical diagnosis that is supported by a history of exposure to a putative allergen. If suspecting this diagnosis, it is important to ask carefully about potential exposure to new substances, including the use of personal care and cosmetic products and occupational and environmental exposure, as well as any prior allergic reactions. Patch testing, which identifies specific allergens responsible for the reaction, may be used to confirm the cause.[11]

The case patient did not have a history of exposure to new allergenic products before the rash onset and the unilateral distribution was not consistent with a diagnosis of allergic contact dermatitis.

Erysipelas

Erysipelas is a bacterial skin infection characterised by a bright patch of erythema and oedema with a well-demarcated border. It primarily affects the upper dermis and superficial lymphatics and is most commonly caused by *Streptococcus pyogenes* (group A streptococcus).[12]

Erysipelas presents with sudden onset of fever, chills and malaise, followed by the appearance of a bright red area on the skin that is firm and swollen with a sharp, raised border. The lower extremities are most commonly affected; however, facial involvement can occur, with patients often displaying a characteristic 'butterfly' distribution across the cheeks and nose. The lesion expands rapidly via lymphatic vessels and may be associated with bullae or petechiae.

The risk of erysipelas is increased by skin barrier disruptions. Entry points for infection include sites of skin trauma (e.g. wounds, insect bites), ulcers or fungal infections such as tinea pedis.[13] Erysipelas is more common in infants and elderly patients, and the incidence is higher in those with chronic venous insufficiency, lymphoedema and immunocompromised states such as diabetes or alcohol dependence.[14]

For the case patient, the vesicular eruption on an erythematous base, progressing from papules through vesicles to crusting lesions in a unilateral distribution, was not typical of this diagnosis. Erysipelas presents with a uniformly bright erythematous area and associated swelling, typically in a butterfly pattern crossing the midline when affecting the face.

Herpes zoster

This is the correct diagnosis. Herpes zoster (shingles) is a reactivation of the varicella-zoster virus (VZV) – the virus that is responsible for chickenpox. Following primary infection, VZV remains latent in the dorsal root ganglia and can reactivate years later, typically when cellular immunity is waning. This reactivation leads to a painful vesicular dermatomal rash accompanied by neuropathic pain, which can persist after rash resolution (postherpetic neuralgia).[15] The incidence of herpes zoster increases with age, particularly beyond 50 years, and is more common in those who are immunosuppressed due to conditions such as HIV infection or malignancy or receiving immunosuppressive therapy.[16]

The global burden of herpes zoster is significant: the estimated lifetime risk is 25 to 30%, and this figure increases to over 50% in individuals who reach 85 years of age.[17] The introduction of VZV vaccines has reduced the incidence in vaccinated populations; however, breakthrough cases still occur.[18]

Herpes zoster has a prodromal phase characterised by localised pain, itching or burning in the affected dermatome, which may precede the rash by several days. The rash itself consists of grouped vesicles on an erythematous base that follow a unilateral dermatomal distribution, most commonly affecting thoracic, cervical and ophthalmic regions.[19] In individuals who are immunocompromised, the rash may become disseminated, extending beyond the primary dermatome. Systemic symptoms such as fever, headache and malaise can also occur.[20]

The aetiology of herpes zoster is closely tied to immune function. Although VZV-specific immunity remains stable

for years following varicella infection, advancing age and the development of chronic diseases (e.g. diabetes, chronic kidney disease) and immunosuppressive conditions lead to a decline in VZV-specific T-cell immunity, which allows for viral reactivation.[21] Psychological stress, trauma and exposure to immunosuppressive medications, such as corticosteroids or chemotherapy, are recognised triggers.[22] Herpes zoster is infectious to people who have not previously had chickenpox.

Diagnosis and investigations

Herpes zoster is primarily a clinical diagnosis made on the basis of the characteristic unilateral, dermatomal vesicular rash. However, it can be challenging to diagnose, particularly in the prodromal stage when patients may present with nonspecific symptoms that can be mistaken for musculoskeletal pain, neuropathy or other dermatological conditions. Clinicians should maintain a high index of suspicion, especially in high-risk populations, such as elderly and immunocompromised individuals.

Laboratory confirmation may be required for atypical cases or immunocompromised patients. Polymerase chain reaction (PCR) testing is the gold standard for detecting VZV DNA in vesicular fluid or crusts or, for patients with herpes zoster meningitis or encephalitis, in the cerebrospinal fluid.[23] Direct fluorescent antibody testing of skin lesions is another testing option but has lower sensitivity than PCR.[24]

Serological tests are not useful in the diagnosis of acute reactivation of VZV. In the primary infection (chickenpox), IgM is often detectable early; however, the response is inconsistent in herpes zoster and often absent or weak.[25] The prior exposure results in persistent VZV IgG positivity.[23]

Management

Early commencement of antiviral therapy, started within 72 hours of rash onset, is crucial to reducing the severity and duration of symptoms in herpes zoster and can significantly reduce complications such as postherpetic neuralgia and prolonged pain syndromes.[26-28] Three antiviral agents – aciclovir, famciclovir and valaciclovir – are available for the treatment of herpes zoster. The current Australian guidelines recommend valaciclovir (1 g three times daily for seven days) or famciclovir (500 mg three times daily for seven days [10 days for immunocompromised patients]) as first-line treatments and aciclovir (800 mg five times daily for seven days) as second-line treatment, noting evidence that famciclovir and valaciclovir are more effective in reducing acute pain in patients with herpes zoster.[29] Famciclovir and valaciclovir have superior bioavailability and more convenient dosing than aciclovir.[26] Intravenous aciclovir (10 mg/kg three times daily) is reserved for severe cases, such as disseminated disease or central nervous system involvement.[26,29]

Pain management is an integral component of management.[30] A stepwise approach is recommended, starting with simple analgesics like paracetamol or NSAIDs.[26] For more severe pain, opioids may be considered.[28] Adjunctive therapies such as gabapentin or pregabalin can be used, particularly if there is a risk of persisting neuropathic pain after resolution of the rash (postherpetic neuralgia).[25,28]

Vaccination is available for the prevention of herpes zoster. The recombinant zoster vaccine (RZV, Shingrix) is preferred over the live attenuated vaccine because it has higher efficacy, especially in older adults.[31] RZV is recommended for individuals aged 50 years and over, administered according to a two-dose schedule.[31] As of November 2023, RZV has been funded under the National Immunisation Program for certain groups: it is available for all adults aged 65 years and over, Aboriginal and Torres Straight Islander people aged 50 years and over, and selected groups aged 18 years and over with moderate or severe immunocompromise.[31]

Outcome

The case patient was diagnosed with herpes zoster on the basis of her clinical features. The rash of vesiculopustular lesions followed the distribution of the right maxillary branch of the trigeminal nerve, and sensory examination revealed hyperaesthesia in the affected dermatome. A PCR test, performed on a specimen from vesicular fluid, returned a positive result for VZV DNA. It is likely that methotrexate, which has immunosuppressive activity, was a trigger for a severe episode of the disease.

The patient was treated with valaciclovir and simple analgesics. She was referred for ophthalmic review, where no epithelial defects were identified. Ophthalmologists play a crucial role in diagnosing and managing herpes zoster ophthalmicus to prevent long-term complications such as corneal scarring, chronic pain and vision loss.

The patient's skin eruption resolved after several weeks. Follow-up at three months showed gradual pain improvement with no lesion recurrence, but she was left with residual scarring. If she continues on long-term immunosuppressive therapy, she would satisfy National Immunisation Program criteria for funded RZV vaccination. Immunocompetent people can wait at least 12 months after an episode of herpes zoster before they receive a zoster vaccine as a lower recurrence rate is observed in the first year; immunocompromised people are at higher risk of recurrence and should receive VZV from three months after the acute illness.[31] MT

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A list of references is included in the online version of this article (https://mt/2025/october/supplements/dermatology-collection-vol-9-no-2).

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