PRACTICE POINTER

Periocular rash

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What you need to know

• Take a focused history of the rash and consider examining the whole skin surface, not just the periocular site, to narrow down the differential diagnosis
• Expect improvement in a periocular rash around 7-10 days into a trial of treatment (aside for suspected rosacea)
• If the rash does not improve, check how much and how frequently treatments have been used so far, and review the diagnosis
• For topical treatment, creams may be more cosmetically acceptable to patients than ointments, but ointments penetrate the skin more effectively
• Consider referral for those who have not responded to treatment, where there is diagnostic uncertainty, the person is systemically unwell, or for patch testing

Skin problems around the eyes can be challenging to diagnose because the differential is wide. They can also be difficult to manage because the periorbital skin is sensitive, and there are a multitude of treatment options, with little specific guidance on their use. This practice pointer outlines the common causes of a periocular rash in an adult, and offers an approach to help diagnosis and management.

How to approach the periocular rash

Start by taking a focused history of the rash, including the time course and symptoms. A diagnostic approach and key clinical features of each diagnosis are summarised in the infographic. Consider examining the whole skin surface, not just the periocular site—often the distribution of the rash on the face and other body sites is key to making an accurate diagnosis.¹ Box 1 provides further pointers to consider when examining the patient. Factors in the history and examination can help differentiate which type of pathology is the predominant problem. For example, inflammation (endogenous such as atopic eczema, or exogenous such as irritant dermatitis), oedema, dyspigmentation, and systemic diseases can all cause periocular rashes.

Endogenous inflammatory causes

Endogenous inflammatory causes of periorbital skin problems should be considered particularly, but not exclusively, where there is a relevant personal or family history.

Atopic eczema

This is the most common cause of endogenous eyelid dermatitis in both children and adults (fig 1), although there is often a concomitant contact allergic component.³ There will often be a personal or family history of eczema or atopy. Treat the acute rash with regular emollients (box 2) and offer a short course of once daily, low potency, topical corticosteroids (box 3). Consider topical tacrolimus or pimecrolimus in people...
Seborrhoeic dermatitis

This common condition affects up to 3% of adults.\(^9\) It can be asymptomatic or can cause varying degrees of pruritus (fig 2). The yeast *Malassezia* is associated with the condition, but whether this is the cause remains unknown. It is associated with chronic neurological conditions including Parkinson’s disease.\(^11\) In recalcitrant or severe cases consider testing for immunodeficiency, including HIV.

Shampoos containing zinc pyrithione or ketoconazole 2% reduce *Malassezia* on the skin.\(^12\) Instruct patients to leave the suds in contact with the scalp for 5-10 minutes and to use the suds to wash the face. Topical antifungal cream, either alone in or in a combined preparation with a mild potency corticosteroid, may be applied twice daily for 7-10 days. Advise patients to wash their eyelids with baby-shampoo applied with cotton buds.

Psoriasis

Eye involvement occurs in up to 58% of people with psoriasis, more commonly in those with psoriatic arthritis.\(^13\) As well as direct ocular involvement, such as blepharitis and conjunctivitis,\(^10\) the periorcular skin can be involved, with psoriatic plaques occurring around the eyes. Look for psoriasis elsewhere on the body and ask if there is a family history.

Treat with a short course of mild potency topical corticosteroid.\(^14\) Alternatives include twice daily topical tacrolimus or a topical vitamin D analogue such as calcipotriol ointment or cream for 2-4 weeks. Counsel patients that vitamin D analogues can sometimes be irritant initially.
Ocular rosacea

Rosacea is common, affecting approximately 5% of adults. 19 Ocular rosacea is found in up to 30% of cases (fig 3), and may precede cutaneous signs in up to 20% of cases. 20

If there are inflammatory papules, treat with topical treatments such as azelaic acid and tretinoin. 21 Consider prescribing an oral antibiotic such as doxycycline if topical treatments are ineffective, 22 although definitive clinical evidence to support this is lacking. 23

Exogenous inflammatory causes

Allergic contact dermatitis

Allergic contact dermatitis is an immunological reaction to an external agent that the person has been exposed and sensitised to before. It has an estimated prevalence of 21.2% 24 and is more common in people with co-existent atopic eczema. 25 It typically presents 24–48 hours after exposure to allergen. The thin skin of the eyelids may be the first area affected from allergens applied to the face (fig 4). Allergens can be found in cosmetics, creams, mascara, and make-up remover—these may also cause an irritant dermatitis. Applicators and eyelash curlers may contain nickel, a common contact allergen. More recently, P-phenylenediamine (PPD), found in hair dye, is a potent sensitizer that can cause a sometimes dramatic facial rash with perioral erythema and oedema. 26 Eye drops, contact lens solution, and acrylates (found in nail varnish and nail extensions) 27 are other potential culprits. An occupational history is important as airborne allergens often affect the eyelids. 28

Stopping contact with the suspected allergen is the mainstay of treatment. 29 Consider a short course of mild potency topical steroid to the periorbital area, or oral steroids in severe cases. Refer to dermatology in severe cases or if patch testing may help to confirm the allergen in cases of diagnostic uncertainty.

Irritant dermatitis

Irritant dermatitis is due to the direct irritant effects of a substance that has been in contact with the skin. Periorbital irritant dermatitis is reported to occur alone in 1–15% of patients with eyelid dermatitis. 30 31 Cosmetics and skincare products are common causes, and several products applied together have a cumulative effect. Volatile substances through occupational exposure may cause an exposed-site dermatitis, often affecting the eyelids. As products often contain a number of irritant ingredients, it can be very difficult to pinpoint a specific offending agent. Therefore, stopping all potential irritant products is key. A short course of topical corticosteroid treatment may be required, but often an emollient and soap substitute will suffice. 30 31 Cosmetics can be restarted once the skin has fully healed, but advise the patient to avoid the suspected irritant in the future to minimise risk of recurrence.

Periorificial dermatitis

This condition, also known as perioral dermatitis, mainly affects young adults. It is often seen in those who have been using topical corticosteroids and in about 3% of people who take inhaled corticosteroids. 32 33 Periocular involvement has been reported in 25% of cases. 32 Treatment is to stop the offending corticosteroid. Suggest a regular non-greasy emollient and a soap substitute and advise that the skin may worsen initially. Consider prescribing an oral tetracycline for six weeks to speed up resolution. 35

Periorbital oedema

Non-infectious causes of acute periorbital oedema include angioedema and allergic contact dermatitis. It is important to exclude periorbital cellulitis and erysipelas (fig 5), in which the patient may be systemically unwell, as these conditions require prompt antibiotics.

Angioedema

The cause is often idiopathic. Consider viral triggers, drugs such as angiotensin converting enzyme (ACE) inhibitors, allergic causes, and rarer causes such as C1-esterase inhibitor deficiency and autoinflammatory conditions. If there is respiratory compromise, manage it as a medical emergency as per anaphylaxis guidelines.

Treat uncomplicated angioedema with a high dose non-sedative antihistamine, up to four times the standard dose. Consider adding an H2 antagonist and montelukast if no improvement is seen. 34

Dyspigmentation

Dyspigmentation is an abnormality in the formation or distribution of pigment in the skin, and can be congenital or acquired. The differential diagnosis is wide. The commonly encountered causes are described below.

Congenital dyspigmentation

Congenital conditions should be considered in people with longstanding lesions present since birth or early infancy.

Congenital naevi

Congenital melanocytic naevi are benign brown or black naevi and are often palpable. They are often single but can be multiple. The risk of melanoma is very low in small single lesions, at approximately 0.1%. 35

Naevus of Ota

This is a longstanding unilateral pigmented lesion affecting the eyelid, sclera, and conjunctiva, or may be confined just to the eye. Ophthalmological referral should be made because of the risk of ocular melanoma and glaucoma. 36 37

Vascular malformations

Port-wine stains are due to capillary malformations. There seems to be a higher incidence of Sturge-Weber syndrome (skin features with cerebral and ocular complications including glaucoma) in port-wine stains affecting any part of the forehead and upper eyelids. 38 Consider referral to ophthalmology and neurology for first presentations.

Acquired dyspigmentation

Facial hypermelanosis is relatively common and can be a diagnostic and therapeutic challenge. Altered pigmentation is generally more prominent in darker skin types, particularly in those of Asian and Middle Eastern origin.
Melasma

This common cause of hyperpigmentation may present anywhere on the face. It is frequently seen in women aged 20-40 years, and is more common in light brown skin types.

Key clinical features:
- Macular brown dyspigmentation
- Usually symmetrical
- Often a history of taking oral contraceptive or of pregnancy
- Worsened by sun exposure.

Sun protection with regular application of high factor sunscreen is recommended as a long term treatment.39 As oestrogen and progesterone have been linked to the development of melasma,40 changing the oral contraceptive to an alternative contraception is often suggested, although the results are variable and evidence is based on small case-series.41

Skin lightening formulations can be tried, including triple-combination therapy comprising hydroquinone, a retinoid, and a corticosteroid. In one open-label study of 173 patients, there was complete clearance in over 90% of cases, and, although 57% experienced at least one adverse effect, only 1% withdrew as a result.42 This should not be used for more than six months due to the risk of developing paradoxical blue-black pigmentation in the long term, otherwise known as ochronosis. Azelaic acid is another treatment option.43 It should be applied twice daily for two to three months.

Vitiligo

This is localised pigment loss that can affect any part of the body, including the periocular region. Most cases occur between the ages of 20 and 30 years.

Key clinical features:
- Areas of macular complete or partial pigment loss
- Follicular openings may be spared but may depigment with time
- Patient may have co-existent autoimmune conditions.

Treatment response is variable. Topical calcineurin inhibitors such as tacrolimus 0.1% or pimecrolimus can be applied twice daily to the affected area for up to six months.44 Topical corticosteroids are not generally advised because of their potential side effects and the evidence suggesting that calcineurin inhibitors achieve similar outcomes.45

Melanocytic lesions

These should always be considered in the differential diagnosis of pigmented (and rarely non-pigmented) skin lesions, and if there is a suspicion of skin malignancy an urgent referral for specialist advice should be sought.

Systemic conditions

The differential diagnoses for localised periorcular dyspigmentation due to underlying systemic conditions can be wide. Dermatomyositis and systemic amyloidosis are described here, but there are many other rare associations, and causes of diffuse pigmentation should also be considered. These include endocrinopathies, metabolic and nutritional conditions, progressive systemic sclerosis, neoplastic conditions, and toxin or drug induced dermatoses.46

Dermatomyositis

Consider this diagnosis if you see a purplish or lilac “heliotrope” rash on the upper eyelids associated with oedema. Other diagnostic features include Gottron’s papules (erythematous plaques on the knuckles), dilation of the nail fold capillaries, and the “shawl sign” (rash around upper back and neck). The patient may report symptoms of muscle weakness signifying myositis. Consider underlying malignancy, found in up to 30% patients.

Systemic amyloidosis

This condition may present with a myriad of cutaneous manifestations.47 Purpura, petechiae, and ecchymoses around the eyes may be seen. Other presentations include smooth, waxy yellowish subcutaneous nodules and plaques. The differential diagnosis includes necrobiotic xanthogranuloma, which is often associated with paraproteinaemias.

Exogenous

Various chemicals can result in skin dyspigmentation. These include metals such as gold, silver, mercury and bismuth which can be encountered through the patient’s occupation or due to iatrogenic causes. Drugs such as the antimalarials, phenothiazines and minocycline are also well recognised culprits.

Photosensitive causes

The differential diagnosis of photosensitive rashes on the face is broad but includes cutaneous manifestations of lupus and other connective tissue disorders such as dermatomyositis. It is always important to exclude drugs as a common and easily treated cause of photosensitive rash. Examination typically reveals more extensive facial involvement, with affected sites including the prominences of the face (cheeks, nasal bridge, and ears) and other sun-exposed sites on the body. Helpful clues indicating a photosensitive facial rash include sparing shaded sites on the face, such as the upper eyelids and behind the ears. Treatment includes sun-protection measures, and assessment at a specialist centre to determine the underlying cause.

Follow-up and referral

What to do after you have made the diagnosis

- Explain in detail how to use the treatments prescribed—dose or volumes, frequency, and duration
- Consider when to follow up—Typically there should be some improvement within 7-10 days of treatment (longer in cases of rosacea requiring systemic antibiotics)

What to do if the treatment doesn’t work

- Inquire specifically which treatments have been used and how—if they were not used as prescribed, then ask why, as patient preference in regimens and formulation is key to successful treatment
- Reconsider the diagnosis if the treatments prescribed have not been effective
- Consider whether there may be more than one cause

When to refer

- A rash that doesn’t respond to standard therapy
- Severe cases for which specialist advice is felt to be needed
- Systemically unwell patients
- Diagnostic uncertainty
- To consider patch testing in cases of suspected contact allergy
Figures

Fig 1 Eyelid eczema: erythema and scaling affecting the upper and lower eyelids with evidence of excoriations suggesting pruritus

Fig 2 Seborrhoeic dermatitis: bilateral orange-red diffuse scaly erythema affecting eyelids with accentuation around the hair-bearing sites, including the eyebrows

Fig 3 Rosacea (left): diffuse telangiectasia and erythema affecting periocular region, cheeks, and the bridge of the nose, with associated inflamed papules and pustules. Chalazion (right): localised erythematous swelling of the lower eyelid, which can be a feature of rosacea and rosaceal blepharitis
Fig 4 Allergic contact dermatitis: well demarcated erythema affecting right lower periocular region with sharp cut-off at right nasal ala

Fig 5 Erysipelas: unilateral, segmental, erythema and oedema affecting the skin of the right middle and lower face, often associated with systemic symptoms of infection
### Periorical Rash: Diagnosis Guide

#### Acute Onset Conditions

<table>
<thead>
<tr>
<th>Condition</th>
<th>History</th>
<th>Morphology</th>
<th>Distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphoedema</td>
<td>Swelling is usually permanently present but is often worse in mornings</td>
<td>Periorcal swelling without significant erythema</td>
<td>Swelling of the face and neck is common</td>
</tr>
<tr>
<td>Angioedema</td>
<td>Acute, often dramatic onset</td>
<td>Periorcal swelling without significant erythema</td>
<td>Often associated with oedema of lips, tongue, and larynx</td>
</tr>
<tr>
<td>Contact Allergic Dermatitis</td>
<td>Typically presents 24 to 48 hours after exposure to an allergen</td>
<td>Scaly rash, Papules may also be present</td>
<td>Localised or generalised, with rash starting at the point of contact</td>
</tr>
<tr>
<td>Irritant Dermatitis</td>
<td>Caused by direct irritant effects of a substance that has been in contact with the skin</td>
<td>Scaly rash, Papules may also be present</td>
<td>Tends to be localised to affected sites, the cheeks are particularly susceptible</td>
</tr>
<tr>
<td>Erysipelas</td>
<td>Acute spreading infection</td>
<td>Erythema with visually indistinct borders, oedema, warmth, and tenderness</td>
<td>Principally involves the dermis and subcutaneous tissue</td>
</tr>
<tr>
<td>Periorbital Cellulitis</td>
<td>Infection may be due to superficial tissue injury such as insect bite or chalazion</td>
<td>Erythema with visually indistinct borders, oedema, warmth, and tenderness</td>
<td>Occurs in the eyelid tissues superficial to the orbital septum</td>
</tr>
</tbody>
</table>

#### Chronic Conditions

<table>
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<tr>
<th>Condition</th>
<th>History</th>
<th>Morphology</th>
<th>Distribution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Atopic Eczema</td>
<td>May be personal or family history of atopy</td>
<td>Itchy erythema with papules, plaques, and associated epidermal scale</td>
<td>Often affects the upper eyelids, periorbital involvement, common</td>
</tr>
<tr>
<td>Psoriasis</td>
<td>Can be triggered by illness, stress, or drugs</td>
<td>Erythematos scaly plaques, sometimes associated with blepharitis</td>
<td>Usually around the eyes, but can also Koebnerise into areas of trauma or dermatitis</td>
</tr>
<tr>
<td>Seborrhoeic Dermatitis</td>
<td>Associated with chronic neurological conditions including Parkinson's disease</td>
<td>Scaly erythematosous rash, Papules may also be present</td>
<td>Affects highly sebaceous areas, on beard, eyebrows, forehead, nasolabial folds, ears, and scalp</td>
</tr>
<tr>
<td>Rosacea</td>
<td>Characterised by episodes of remission and recurrence</td>
<td>Combinations of erythema, telangiectasia, papules, and pustules</td>
<td>Mainly around the central face, may also feature eyelid 'sandpapering', gritty feeling eyes</td>
</tr>
<tr>
<td>Periorificial Dermatitis</td>
<td>Often seen in those who have been using topical or injected corticosteroids</td>
<td>Erythematosus papules and pustules</td>
<td>Often around the mouth, with sparing of the vermilion border</td>
</tr>
<tr>
<td>Congenital Dyspigmentation</td>
<td>Longstanding conditions such as vascular malformation and congenital naevus</td>
<td>Vascular malformation: port-wine stains, Congenital naevus benign brown or black naevi</td>
<td>Congenital naevus may affect the eyelid, cheeks, and conjunctiva, or just the eye</td>
</tr>
<tr>
<td>Acquired Dyspigmentation</td>
<td>Vitiligo, facial melanosis, systemic conditions, melanocytic lesions, and exogenous causes</td>
<td>Altered pigmentation, Generally more prominent in darker skin types</td>
<td>Various, depending on underlying cause</td>
</tr>
<tr>
<td>Photosensitive Rash</td>
<td>Differential includes cutaneous lupus, dermatomycoses, and other connective tissue disorders</td>
<td>Erythema on prominences of the face (cheeks, nasal bridge, and ears)</td>
<td>Sparing of shaded parts of the face, such as upper eyelids and behind ears</td>
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