The Red face

Many skin problems produce redness—here’s how to sort them out

Skin conditions that present with redness of the face are common in everyday dermatology practice. From the doorway, all red faces may appear similar, but each condition often has a unique history and morphological presentation that can help us identify them accurately. Below we provide an overview of four of the most common causes of the "red face."

**Perioral dermatis**

This is a common, inflammatory cutaneous condition of unknown etiology that typically affects the skin around the mouth. Perioral dermatis is not to be confused with lip licker’s dermatitis.

Perioral dermatitis can occur in all age groups including children, but it typically affects females between the ages of 20 and 45 years. It invariably appears in the perioral area, but can also affect the nasolabial folds, chin and eyes.

The precise cause is unknown, but a variety of potential environmental factors have been implicated. These include:

- corticosteroids: prolonged application of strong topical corticosteroid creams to the face; inhaled and nasal steroids also implicated;
- cosmetic products, makeups and moisturizers;
- fluoridated toothpastes;
- sunscreens;
- hormonal factors; and
- infectious agents (controversial).

Perioral dermatis presents spontaneously over a few weeks as erythematous papules, papulovesicles or papulopustules around the mouth, nose and chin. One striking clinical sign of perioral dermatis is that it spares the lip border, leaving a distinct perioral halo.

The lesions are painless but may cause burning and itching. The condition often appears dry and scaly. When the lesions are confluent, the skin can appear bright red.

Perioral dermatis can vary in severity but is not a dangerous condition nor a marker for underlying systemic disease. If left untreated, the condition may wax and wane for months or years. However, the clinical appearance and resultant impact on self-esteem lead most patients to seek continued on • page 14
from page 13 medical care expeditiously.

The diagnosis of perioral dermatitis is clinical. It can resemble acne but is differentiated by its lack of comedones. The relationship to rosacea, both clinically and histologically, is not entirely clear; although both conditions are treated similarly.

Lip lickers dermatitis is a distinct condition that pre- sent as scalp, pink or red patches and plaques around the mouth in young children from repeated lip licking. It typically does not spare the lip border. Lip lickers dermatitis is often erroneously labeled perioral derma- titis in clinical practice. Seborrheic dermatitis presents with erythema and scaling without discreet papules.

Patients with perioral dermatitis often report distinct improvement in their condition after being treated with topical steroids prescribed by their physician; however, this improvement is short-lived and their condition violently flares upon steroid withdrawal. Cyclic re- treatment with topical steroids only exacerabtes and perpetuates the condition.

Fortunately, perioral dermatitis is highly responsive to treatment with few recurrences, unlike acne vulgaris and rosacea.

The mainstay of treatment involves the elimination or even avoidance of all topical steroids, but also heavy moisturizers, makeups, sunscreens and even fluorid- ated toothpastes. Patients need to be educated about the experiences of steroid withdrawal flare.

Oral anti-inflammatory antibiotic therapy is the most effective treatment for perioral dermatitis. Short courses of oral tetracycline, minocycline or doxycycline over six to 12 weeks worked very well in the majority of patients with minimal side-effects. Oral erythro- mycin has also been reported to work well in patients intolerant to cycline antibiotics, pregnant women and children.

A variety of topical therapies have been employed to treat perioral dermatitis, although they are not as effective as oral therapy. For steroid-induced peri- oral dermatitis, topical calcineurin inhibitors (e.g., pimecrolimus) have been shown to be efficacious by way of their non-steroidal anti-inflammatory effect.

Topical antibiotics such as mectnizidol, eryth- romycin and clindamycin, as well as anti-acne agents such as azelaic acid, retinoids and benzoyl peroxide have all been used with varying degrees of success to treat perioral dermatitis.

**Actinic keratoses**

Also known as solar keratoses, these are shallow, red, rough, cutaneous lesions that typically develop on sun- exposed surfaces of older, fair-skinned individuals. It is important to recog- nize actinic kerato- ses (AKs), as they have the ability to transform into inva- sive squamous cell carcinoma (SCC). They represent the most common site of cutaneous malignancy encoun- tered in clinical practice and are not to be confused with benign seborrheic keratoses.

Chronic exposure to ultraviolet radiation in the form of sunlight or artificial tanning beds is respon- sible for the development of nearly all actinic kera- toses. Cumulative UV exposure results in a series of sequential mutations that cause keratinocyte dysplasia and the development of actinic keratoses. Rarely, actinic keratoses may be caused by exposure to X-rays or industrial chemicals.

Risk factors include:
- male gender;
- advanced age;
- fair skin, blond or red hair, blue, green or

hazed eye colour;
- skin that freckles or burns when in the sun;
- high-intensity or high cumulative exposure to UV radiation, sunburn;
- living close to an irritant, having an outdoor occupation or lifestyle;
- immunosuppression in the setting of organ trans- plant, immunosuppressive medications/chemo- therapy and from AIDS-related diseases;
- genetic conditions such as albinism or xeroderma pigmentosum.

AKs appear as asymptomatic red, flesh-coloured or yellow dry, rough, keratotic papules or plaques on sun-exposed areas of the skin. They can present as single lesions but more often present in multiples, which can give rise to the appearance of diffuse redness of the affected areas (the “red face”). They usually measure less than 1 cm in diameter and are often felt more than they are seen.

The scalp (in bald men), face, ears, neck, chest, backs of hands, forearms and lips are commonly affected.

The skin surrounding AKs (the “field”) often shows signs of chronic UV exposure, including telangiectasia, roughness, wrinkles and telangectesias. The term “field cancerization” refers to non-palpable, non-visible (subclinical) pre-neoplastic lesions that surround areas of AKs. This concept becomes important when deciding on a treatment strategy.

AKs can persist indefinitely, regress or transform into hypertrophic AKs, SCC in situ (Bowen’s disease) or squamous cell carcinoma. The risk of an AK to an SCC has been reported to vary anywhere between 1% to 10%.

The diagnosis of AKs is clinical; however, lesions that look atypical can be biopsied for confirmation. The differential diagnosis for AKs include irritated sebor- rheic keratoses, Bowen’s disease, basal cell carcinomas, discoid lupus and pokercerosis. AKs can sometimes resemble discoid psoriasis or actinic pruritic eczema.

Treatment can be divided into lesion-directed and field-directed approaches; however, combinations are often used. Lesion-directed treatments are used when treating a small number of lesions and include physically destructive techniques such as cryotherapy as well as surgical approaches such as excision or cur- ettage. Cryotherapy using liquid nitrogen remains the most common modality for treating AKs in the office setting.

Field therapies aim to treat multiple clinical and sub-clinical lesions in the field of sun-damaged skin. Field-directed therapies involve the topical agents fluorouracil (5-FU), imiquimod 5% or 3.75% and the newer agent ingenol mebutate 0.015% or 0.05%. Fluorouracil (5% cream) is a topical anticaner agent that directly affects and destroys cancer cells and is used twice daily over two to four weeks. Imiquimod induces cytokines that target atypical keratinocytes and is used topically in different regimens over six to 16 weeks. Ingenol mebutate works through direct cytotoxic and immunomodulating effects and is used for two or three consecutive days depending on the locations being treated. All field therapies can be associated with mild to severe inflammation of the skin. Photodynamic therapy is also a field-directed treatment, but one not commonly employed in office-based settings.

The prevention of AKs involves the compre- hensive sun safety strategy of sun avoidance, sun- protective clothing and the regular use of broad- spectrum sunscreens.

**Rosacea**

Rosacea is a common, chronic inflammatory skin disease primarily of the comedones of the central face. Its course is characterized by eruptions and eruptions. Although most often seen in patients with fair skin, rosacea occurs in all skin types. Women are more often affected than men and the onset is typically after age 30.

Pathogenesis may involve increased vascular reac- tivity and inflammatory mediators in the skin, espe- cially increased production of antimicrobial proteins such as cathelicidins. Also, the role of the Demodex mite is being re-evaluated, as topical ivermectin is

showing promise in treating rosacea. Erythemat- otelangiectatic rosacea may present with flushing alone. The papulopustular rosacea subtype affects acne vulgaris without any comedones. Acne and rosacea can occur together, so keep this in mind if comedones are vis-ible. Phymatous ros-acea often develops after or in combination with other sub- types. Phyma refers to a distinct swelling caused by lymph- edema and hypertrophy of subcutaneous tissue, and particularly affects the nose (rhinophyma). In ocular rosacea, ocular signs and symptoms have therefore cutaneous manifestations in up to 20% of patients. A watery or bloodshot appearance of the eyes is a good diagnostic feature. Meibomian gland dysfunction manifesting as a chalazion or sty are other features of rosacea-related ocular disease.

Patients should be advised to avoid aggravating factors including spicy food, alcohol and hot weather. Patients should also be warned not to apply potent topical corticosteroids to the face.

Medical therapies help to reduce the inflammation that causes rosacea, improving the papules and pustules.

There is now a therapy approved for the flushing variant of rosacea to help reduce the background eryth- rema. Topical brimonidine is an alpha-2 adrenergic agonist that causes temporary vasoconstriction of cutaneous small vessels at the site of application. It is not helpful for telangiectasia. Effects can be seen within half an hour, peak at three hours and may last up to 12 hours. Patients are instructed to apply evenly to affected red areas in the morning. It should be avoided in patients with vasoactive conditions (e.g. Raynaud’s disease) and patients should be cautioned about the possibility of rebound.

As background erythema can be challenging to treat, even with topical and laser options, cosmetic camouflage may be especially helpful for females. Telangiectasia can be effectively treated by various physical measures. Vessels can be ablated with min- imal scarring using electrical hyfrecators, vascular lasers and intense pulsed light lasers.

Phyma also require physical ablation or removal. Surgical options include paring and electrosurgery.

Medical treatment mainly involves topical and sys- temic anti-inflammatory antibiotics. Oral tetracyclines (250 to 500 mg twice daily) are well-established. Other tetracyclines, i.e. doxycycline 100 mg once daily, offer the advantage of simpler dosing, plus their absorption is less influenced by dietary calcium and they can be taken with food. These are limited by anti- biotic resistance. Fortunately, we now have the option of using a low-dose, slow-release preparation to give 40 mg daily that does not reach the antimicrobial threshold can be taken on an ongoing basis to control inflammatory rosacea. Topical metronidazole 0.1% gel applied once daily has been shown to produce significant results after two months. Azelaic acid gel is another option.

Oral retinoids are sometimes considered for more severe presentations.

Therapies undergoing investigation include topical ivertinectin for treating inflammatory rosacea.

**Seborrhoeic dermatitis**

Seborrhoeic dermatitis (SD) is a chronic, inflammatory disease of the skin characterized by greasy, yellow, erythematous scaling plaques in areas rich in seb- aceous glands. It affects up to 5% of the population, men more
often than women. Distribution tends to be bimodal, peaking in adolescents and young adults, as well as in adults over age 50. Although it can affect all ages, it is much more common when sebaceous glands are active (i.e., the first few months of life and post-puberty).

Severe and recalcitrant SD can be a cutaneous sign of HIV infection. Parkinson’s disease is also associated with severe, refractory SD. It can occur concomitantly with acne, rosacea, psoriasis and pityriasis versicolor.

SD is a non-infectious condition thought to be caused by reaction to Malassezia yeast, a common skin organism. SD has a preference for sebaceous areas and large body folds, occurring most notably on the face, especially the nasolabial folds, glabella, beard, ears, scalp, central chest, upper back and intertriginous areas.

It commonly exhibits a mild course with moderate discom- fort. It tends to be chronic with frequent relapses but no systemic effects. Areas of SD can be irritated by topical products, heat, sunlight and fever. It is rarely generalized. In infants, it often appears as early as one week, and the course is self-limited.

Infant SD is characterized by greasy yellow-brown scales on the scalp vertex spreading to the entire scalp with inflammation and oozing, resulting in a thick scaly crust covering most of the scalp, hence the term “cradle cap.” Intertingogenous SD in infants should be differ- entiated from irritant diaper dermatitis (i.e., confined to diaper area, sparing skin folds), infantile psoriasis, candidiasis and atopic dermatitis (pruritus causes marked irritability and sleeplessness).

In adults, dandruff (pityri- iasis sicca) can be a common presentation. SD of the scalp is characterized by diffuse within scalp margins, with white to yellow greasy flakes, dandruff, as well as underlying erythema that is often itchy. It tends to be more diffuse than psoriasis, extending just beyond the hairline at the forehead and is sharply demarcated.

SD is a clinical diagnosis. Biopsy can help rule out other possibilities such as psoriasis, and fungal culture may be considered especially for scalp lesions to rule out tinea capitis.

SD is a chronic relapsing and remitting condition that responds to both antifungal and anti-inflammatory approaches, but there is no definitive cure. Since patients will be required to treat and re-treat their lesions, therapies that are well-tolerated are key. Bathing, emollients and mild shampoo can help remove crusts and scales.

Keratolytics and anti-yeast agents commonly used include selenium sulfide, tar, sali- cylic acid and zinc pyrithione shampoo. Shampoos should be used two to three times per week and left on the scalp for at least five minutes before rinsing to ensure scalp penetration.

Low-potency topical steroids suppress the inflammation of SD. They have a good rapid effect, especially for itching. For SD on the face, patients can use topical steroids once or twice daily. Topical calcineurin inhibitors (tacrolimus and pimecrolimi- mus) have anti-inflammatory properties without the adverse effects of topical steroids.

Antifungals treat SD by decreasing Malassezia yeast counts. Many antifungals are effective, including ciclopi- ron, selenium sulfide, azoles (clotrimazole, ketoconazole, fluconazole) and terbinafine. The azoles represent the largest class of antifungals used in the treatment of SD. Topical azoles (shampoos and creams) are up to 90% effective. They should be used daily until resolution, often within several weeks.

Given the dual nature of SD, combining topical steroids and azoles (twice daily to affected areas for two weeks) for facial SD may be better than mono-therapy. Consider compound- ing 1% hydrocortisone powder in an azole cream. Consider a maintenance dose of twice weekly to help prevent flares. Severe cases may require adding an oral antifungal to a topical regimen. MP

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