Common Dermatologic Conditions

Jay C. Vary, MD, PhD, Kim M. O’Connor, MD

INTRODUCTION

The skin serves many functions other than simply wrapping and containing all the deeper structures.\(^1,2\) It is composed of an uppermost self-renewing epidermis of primarily keratinocytes that becomes scaly and red in response to superficial inflammation from irritation or infection. Immediately subjacent is the dermis, which contains not only connective tissues and vasculature but also the skin appendages. These appendages are derived from buds of the epidermis that grow down into the dermis early in development and result in the eccrine and apocrine sweat glands, nails, and the pilosebaceous unit that is the target of inflammation in alopecia, acne vulgaris, and rosacea. Below the dermis, the subcutaneous tissue lies above muscular fascia in most areas of the body, and is composed of adipocytes as well as the larger trunks of vessels and nerves passing through.

KEYWORDS

- Alopecia
- Facial rashes
- Intertriginous rashes
- Leg rashes
- Acne

KEY POINTS

- The most common causes of alopecia are non-scarring and non-inflammatory and can be distinguished by the pattern of hair loss and the presence of ongoing shedding.
- Age of onset, distribution of the rash, presence or absence of comedones or systemic symptoms can help distinguish between the causes of facial rash.
- The utilization of the KOH prep and Wood’s lamp examination can aid in the diagnosis of intertriginous rashes.
- Stasis dermatitis and contact dermatitis on the legs are commonly mistaken for infection but history and examination findings can usually quickly exclude infection.

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Dermatologic diseases can all be thought of as perturbations of the components of these 3 layers of cells and structures. Loss of the barrier function, exuberant inflammation or autoinflammation, aberrant neurologic signals, infection, or metabolic disruptions in normal function account for virtually all of the more than 3000 dermatologic diagnoses. Identifying the specific causes can be challenging, and the causes of many dermatologic diseases remain unknown. Many diseases can appear similar to each other, and it is common for skin diseases to present uncommonly. Traditional texts and atlases of dermatology often present only 1 or 2 images of any skin disease, whereas newer Internet-based sources can produce hundreds of images of a single diagnosis to allow a better appreciation of how a disease can vary in its presentation. However, trying to match a patient’s skin to a picture or set of pictures can be time consuming and prone to error.

This review attempts to approach the subject as a clinician would: exploring the differential diagnosis for common complaints. The authors start with the hair and briefly describe how to approach the patient with hair loss to reach a diagnosis. Next, the 4 most common causes of hair loss, namely androgenetic alopecia, female pattern hair loss (FPHL), alopecia areata (AA), and telogen effluvium (TE), are discussed in depth. The discussion then turns to the patient with a facial rash. Differentiation of acne vulgaris from rosacea, periorificial dermatitis, and seborrheic dermatitis is covered, as is differentiation of erysipelas and cellulitis, and the malar rash of systemic lupus erythematosus (SLE). Axillary and inguinal rashes are presented in the section on intertriginous rash, and include candidal intertrigo, tinea, erythrasma, inverse psoriasis, and the common frictional and irritant contact dermatitis referred to as intertrigo. Finally, the authors provide quick and reliable signs and symptoms to differentiate infectious causes of an edematous red leg that may require immediate antibiotics or admission from subacute mimics such as contact and stasis dermatitis. A brief discussion of deep vein thrombosis (DVT) is also included.

ALOPECIA

One of the more common dermatologic complaints seen by dermatologists and primary care physicians alike is alopecia or hair loss. The term alopecia is usually used to refer to loss of hairs at the follicle, although often patients will complain of hair loss that is in fact due to breakage of the hair shaft resulting from congenital or acquired causes of hair-shaft fragility instead of true loss of the entire hair. These entities are usually easily differentiated by an initial examination of the scalp and a hair-pull test. The hair-pull test is done by grasping hairs within approximately 1 cm² of scalp and pulling firmly between the thumb and forefinger (violent pulling such as with the aid of a clamp is not recommended); more than 5 hairs per location is considered abnormal. Examination of the ends of the hair can further differentiate between broken hairs that will end abruptly or hairs that have come from the follicle and will have some remnant of a follicle. Normally only resting telogen or “club” hairs can be pulled, which have a small spherical follicular remnant at the end. Actively growing anagen hairs will keep part of the root sheath, which appears like a sheath on the end of the hair (Fig. 1). Anagen hairs should never normally be released with a firm pull unless there is an underlying disease process.

The potential causes of alopecia are many (Table 1), but the differential diagnoses can be focused by an examination of the scalp to determine if there is evidence of scarring or inflammation. Unlike some mammals that can regenerate the pilosebaceous unit, human hair follicles never regenerate if destroyed. Scarring can be subtle in early stages, but close examination shows a complete loss of the follicular os and
Fig. 1. Hair-shaft examination from hair-pull test. Note the anagen hair (left) has an attached rumpled inner root sheath still attached while the telogen hair (right) is often referred to as a club hair because of its shape and lack of attached root sheath. (From James WD, Berger TG, Elston DM. Andrews’ diseases of the skin: clinical dermatology. Philadelphia: Elsevier/Saunders; 2011; with permission.)

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Classification of alopecia diagnoses based on clinical findings</th>
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<tr>
<td></td>
<td>Scarring</td>
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<tr>
<td>Inflammatory</td>
<td>Lupus erythematous (discoid or subacute cutaneous)</td>
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<td>Lichen planopilaris</td>
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<td>Frontal fibrosing alopecia</td>
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<td>Chronic suppurative folliculitis</td>
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<td>Folliculitis decalvans</td>
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<td></td>
<td>Dissecting cellulitis of the scalp</td>
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<td></td>
<td>Sarcoidosis</td>
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<td>Favus</td>
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<td></td>
<td>Acne keloidalis nuchae</td>
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<tr>
<td>Noninflammatory</td>
<td>Central centrifugal scarring alopecia (follicular degeneration syndrome)</td>
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<td></td>
<td>Pseudopelade</td>
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<td>Inactive “burnt-out” scarring alopecia</td>
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<td>Scleroderma</td>
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replacement with smooth epidermis, occasionally with “doll’s hairs” clustering of multiple hairs through a single os. As this change is permanent, scarring alopecia must be treated quickly to stabilize the disease and prevent further loss. Referral to a dermatologist is strongly recommended if scarring is appreciated.

The presence of erythema and scale is clinically apparent in many causes of alopecia, and can be used to further narrow the differential diagnoses. A full discussion of alopecia is beyond the scope of this article; however, the 3 most common diagnoses are covered here, all of which are nonscarring and usually noninflammatory (Table 2).

**Androgenetic Alopecia/Female Pattern Hair Loss**

Male pattern baldness is very common, although the term androgenetic alopecia (AGA) is preferred, as it correctly suggests the causes as being androgen dependent and genetically predisposed. There is an estimated prevalence of nearly 50% in Caucasian men by age 50 years, increasing with age to plateau at about 80% prevalence in those older than 70, such that a lack of AGA in the elderly may be considered the abnormal state. An androgenic cause for AGA was recognized early as it is not found in eunuchs lacking androgens, in those without functional androgen receptors, or in those lacking 5α-reductase, which converts testosterone to dihydrotestosterone.
(DHT), the androgen most directly responsible for AGA. It is odd that DHT causes follicular miniaturization only in AGA-susceptible follicles on the crown, while the same hormone is responsible for development of secondary sexual hair in the axilla, groin, chest, back, and face. Transplantation of uninvolved follicles from the occiput to the crown results in hairs that do not undergo miniaturization, which is why hair transplants for AGA can be successful.

FPHL is also much more common than is generally believed, with a prevalence of 20% to 30% by age 50 years although, like AGA, it can start in the 20s. Though still controversial, a hormonal basis for FPHL has been more difficult to establish, hence the separate diagnostic term for the similar process in women. Although increased androgen levels can induce FPHL, androgen levels are not abnormal in most women with FPHL, nor do most have other evidence of androgen excess such as hirsutism or irregular menses. Antiandrogens are sometimes successful in treatment, however, suggesting a role in the etiopathogenesis of FPHL.

Genetics also play a strong role, but AGA and FPHL are likely polygenic and not well characterized. Not surprisingly, most candidate genes are involved in the production or binding of testosterone and DHT. Most commonly there is a family history of AGA, but phenotypic variation in families and variable penetrance owing to this likely polygenic inheritance often results in a patient feeling they are the “only one in the family” to be so affected, and should not be used as a reason to dismiss the diagnosis.

Contrary to popular belief and the clinical appearance, individuals with AGA do not have true alopecia but rather miniaturization of the affected hairs. In affected pilosebaceous units of AGA and FPHL, the hair-follicle growth phase, anagen, shortens drastically, resulting in miniaturized hairs appearing on the scalp, with thinner shafts, much shorter length (sometimes too short to reach the follicular os), and often a lighter color, giving the appearance of no hair at all. Patterns in AGA and FPHL are similar but distinctly different. AGA results in initial bitemporal recession of the frontal hairline or vertex thinning, often developing into complete apparent loss of the hair on the crown, but completely spares the horseshoe distribution of the inferior parietal and occipital scalp. By contrast, FPHL results in a more uniform apparent thinning of the entire crown owing to miniaturization of many, but not all of the hairs in the involved area. FPHL shares the same horseshoe distribution as in male AGA. Comparison of part width on the crown to the sides or occiput can be helpful in early cases of FPHL.

More recently, associations of AGA and FPHL with other comorbid diseases have become apparent. Metabolic syndrome is highly correlated with both AGA and FPHL (odds ratio = 10.5 and 10.7, respectively) as well as the presence of atherosclerotic plaques and cardiovascular disease in males with AGA and possibly in women with FPHL, although this is less established. The mechanisms by which these associations may occur are still unclear.

Though less common, conditions of androgen excess can also result in AGA or FPHL. In women, androgen excess should be suspected if alopecia is of a more male pattern and is accompanied by hirsutism, deepening of the voice, clitoral enlargement, hormonal acne, or irregular menses. Clinical or laboratory assessment for polycystic ovarian syndrome, congenital adrenal hyperplasia, or an androgen-secreting tumor should be considered in these cases. Sudden-onset AGA or FPHL in either sex should also prompt consideration of such tumors.

Diagnosis is by recognition of the common pattern of involvement and the presence of miniaturized hairs on close inspection, often with the aid of magnification. Eliciting a history of effluvium (hair shedding) rather than simply slow apparent disappearance of hair is important, as other causes of alopecia will unmask AGA or FPHL, leading to a similar initial appearance. For example, a person with previously subclinical
FPHL who has their patterned alopecia unmasked only when 10% of the entire scalp hair is lost for an unrelated reason may appear clinically to have FPHL until a hair-pull test shows effluvium from all areas of the scalp.

Any discussion of treatment should include acceptance, as AGA and FPHL are common. Techniques to disguise the appearance without actively altering the disease are also important, alone or in combination with medical therapy, including the “comb-over,” aerosolized powders or fibers to coat remaining hairs making them appear thicker, volumizing shampoos, artificial hairpieces, or hair transplantation.

There are several medical treatments in common use for AGA and FPHL, but it must be stressed at the time of initiation that none are a cure but rather are treatments that must be continued, or reversion to the initial state will occur.

Minoxidil is an antihypertensive medication that results in generalized hypertrichosis by an unknown mechanism if taken orally, but is much more commonly used as a topical preparation to encourage growth of hairs with thicker diameter and length. It is relatively inexpensive, is available without a prescription in the United States, and has a favorable side-effect profile in both men and women. The most common side effect is local irritation caused by propylene glycol in the solution formulation, but this is absent in the foam.10 As minoxidil lowers blood pressure when taken orally, topical application can be associated with compensatory symptoms (eg, tachycardia,
presyncopal symptoms), but in blinded trials hypotension occurs at the same rate as in the vehicle.\textsuperscript{10} Accidental application to other areas can result in localized hypertrichosis, but discontinuation usually normalizes this. When compared with twice-daily 5% solution, the 2% solution applied twice a day has a lower incidence of side effects, as does the once-daily application of 5% solution, though both at the expense of lesser efficacy.\textsuperscript{11,12} A temporary effluvium 1 to 3 months after initiation is common, as many follicles in telogen are stimulated to enter anagen, resulting in an effluvium of the hair shafts already residing in those follicles when treatment is begun. Educating patients to expect this paradoxic sign of medication efficacy will help adherence and prevent panicked phone calls. It will then take another few months for the new hairs to grow long enough that the hair appears denser.

Antiandrogens such as finasteride, spironolactone, flutamide, and cyproterone are also often used for AGA and FPHL, although finasteride is the only medicine approved by the Food and Drug Administration (FDA) for this indication, and only in males. Finasteride is a type II 5\textsubscript{\alpha}-reductase inhibitor, resulting in less conversion of testosterone to DHT. Finasteride is available in the United States as a 1-mg once-daily treatment of AGA in males, although often for cost-saving reasons it is prescribed as one-quarter tablet once daily of the 5-mg dose (resulting in a 1.25-mg final dose) produced to treat prostate disease. In a double-blind, placebo-controlled crossover study, hair count, patient self-assessment, and investigator assessment were significantly greater in the treatment cohorts. The most significant side effects are sexual, with decreased libido, erectile dysfunction, or decreased ejaculate volume.\textsuperscript{13} A recent meta-analysis of 12 studies of finasteride in men showed that patient self-report of improvement with long-term use was significantly more favorable than with placebo (relative risk $[RR] = 1.71$, 95% confidence interval $[CI] = 1.15–2.53$) with a number needed to treat of 3.4. Interestingly the relative risk of sexual side effects was actually higher than that for hair growth ($RR = 2.22$, 95% CI 1.03–4.78), although this complaint was uncommon even in placebo, so the number needed to harm was 82.1 and the number who discontinued the medication because of sexual side effects when compared with placebo was not significant.\textsuperscript{14} Recent concern in the lay press arose after a publication describing persisting sexual side effects in 71 men after discontinuation of finasteride; however, this descriptive case series was not part of a larger cohort, so an incidence estimate cannot be made. In addition, men never on finasteride complain of sexual side effects, so without a placebo group, causation by finasteride has yet to be established.\textsuperscript{15}

Initially there was concern that use of finasteride may delay early detection of prostate cancer, and early studies suggested an increased incidence of high-grade malignancies in those taking the 5-mg dose, prompting an FDA warning. However, subsequent analysis shows it is associated with a nearly 30% reduction in low-grade prostate cancer, with no effect on high-grade prostate cancer.\textsuperscript{16} Dutasteride is a similar inhibitor of both type I and type II 5\textsubscript{\alpha}-reductase and has also shown efficacy in AGA without significant adverse events when compared with placebo, although admittedly in a relatively small study.\textsuperscript{17} Use in AGA or FPHL is currently off-label. Finasteride and dutasteride are not recommended in women of child-bearing potential given the high risk of hormone-related birth defects, and are FDA pregnancy category X. Use in postmenopausal women to treat FPHL has been mixed.\textsuperscript{18}

Other antiandrogens are not typically used in males owing to the risk of feminization, although they are used in FPHL. Well-designed studies examining effectiveness of antiandrogens in FPHL have been lacking thus far to allow firm conclusions as to their effectiveness, but small case series sometimes support their use.\textsuperscript{19} A recent Cochrane review of hormonal and nonhormonal treatments found minoxidil was the only
medication for which evidence of treatment effect could be demonstrated in FPHL, possibly in part due to this lack of studies. Though used sometimes in FPHL, flutamide use is often limited because of concerns about hepatotoxicity, and cyproterone is not available in the United States. On the contrary, spironolactone is commonly used off-label to treat hormonal acne, hirsutism, and FPHL in doses from 50 to 200 mg daily. Spironolactone both decreases testosterone production and competitively blocks the androgen receptor. Hypotension and hyperkalemia can occur, but with normal renal function these are uncommon. At higher doses, irregular menses and breast tenderness occur, although this can often be mitigated by concurrent use of oral contraceptive pills. As expected by its effect on androgen activity, spironolactone is contraindicated in pregnancy. An FDA warning was issued because of an increased risk of malignancy based on animal studies, but it is unclear as to whether this is relevant in humans. A large retrospective matched cohort study of more than 1 million women aged 55 years and older showed no increased incidence of breast cancer in those who took spironolactone, but no such studies have been done in premenopausal women who might be expected to take the medication for years.

**Alopecia Areata**

AA is the most common inflammatory cause of alopecia, with an estimated prevalence of approximately 0.1%, while the lifetime risk approaches 2% based on those seeking medical attention. It is perhaps slightly more common in children than in adults. Its course is unpredictable, but typically self-resolving, so patients are often told that treatment is not necessary, and it is often viewed as strictly a cosmetic problem. However, its sudden onset and apparently random patchy areas can be very distressing to patients.

AA is an autoimmune process that appears to be a result of, or is associated with, loss of the immunologically privileged site of the hair bulb where pigment is produced. There is a predominantly T-cell rich infiltrate at the hair bulb that results in cessation of hair-shaft production but, importantly, it does not scar or permanently damage the follicle itself.

Most commonly, patients complain of 1 or a few patches of complete hair loss overlying otherwise normal skin while the rest of the scalp is unaffected (Fig. 3A). Early in its course, the involved skin can be subtly pink but not scaly. The beard is commonly affected in men, while involvement of the body hair without concomitant scalp involvement is either uncommon or uncommonly comes to medical attention. It is usually otherwise asymptomatic, and many people are unaware of the condition until a hairdresser or relative points it out. Areas of involvement start as a circular patch of near complete alopecia that gradually increases in diameter, often growing back centrally as it continues to expand peripherally. Often at the periphery of these patches are exclamation-point hairs, which are short hairs (<1 cm) named for the characteristic tapering diameter as they approach the follicular ostia (see Fig. 3B). New patches in other areas of the scalp may also occur and sometimes coalesce. Like another common autoimmune pigmentary disease, vitiligo, it mysteriously affects small areas of skin that enlarge rather than being generalized in most cases. Like vitiligo, it can often wax and wane in its course. There is often a history of white hairs growing back during recovery, even in patients without normal graying. Similarly, in those with graying hair the already white hairs are usually spared initially while the darker hairs fall out, leading to the myth of people’s hair turning white overnight when diffuse involvement occurs. It is the only type of alopecia that preferentially spares white hairs, so this is a pathognomonic finding. More extensive (and less responsive) patterns are uncommon and include the ophiasis pattern (loss of all but the crown), the sisaipho pattern (loss of
the crown only), AA totalis (loss of all scalp hair sparing the face and body), or AA uni-
niversalis (loss of all scalp, facial, and body hair). Diagnosis is by recognition of the common circular patterns without scaliness or
scarring of the underlying skin, presence of exclamation-point hairs, and apparent
sparking or regrowth of white hairs. The differential diagnosis includes secondary syph-
ilis, SLE, tinea capitis, and trichotillomania when localized and nonscarring patches
are seen. When diffuse, the differential includes trichotillomania, telogen effluvium
(see later discussion), and metabolic or drug-induced causes, and can be more diffi-
cult to diagnose without biopsy. Hair-pull tests are often positive near involved skin
and will show anagen hairs, which are otherwise not a normal finding.

AA affects only the hair, nails, and rarely the eyes, without any other systemic man-
ifestations. Comorbid conditions include thyroid disease in 8% to 28% of patients as
well as, less commonly, vitiligo, lupus, atopy, and other autoimmune conditions. A review of systems should be done to evaluate for these, although testing is not typi-
cally cost-effective in the absence of symptoms as they are not thought to be causal
or temporally associated.

Treatment can consist of the “tincture of time,” as approximately 80% of cases spon-
taneously resolve within a year, although resolution is less common in extensive
disease. Providing information as to what to expect by directing patients to resources

Fig. 3. Alopecia areata. Note the isolated area of smooth alopecia with trace pink to peach-
colored erythema of acute alopecia areata in (A) and the exclamation-point hairs (arrows) in
(B). ([A] Data from Restrepo R, Calonje E. Diseases of the hair. In: Calonje E, Bremm T, Lazar AJ,
permission; and [B] Adapted from Sperling LC, Sinclair RD, Shabravi-Caelen LE. Alopeacias. In:
Saunders; 2012; with permission.)
such as the National Alopecia Areata Foundation (www.naaf.org) can be as valuable as any treatment offered. Localized involvement often responds fairly well to topical or intralesional corticosteroids. High-potency topical corticosteroids are effective in approximately 25% of patients but can take up to 6 months to see regrowth, while monthly intralesional injection (usually triamcinolone) throughout affected patches into the deep dermis (to the base of the follicles) allows for more rapid regrowth, although both treatments need to continue until the disease has run its course. Aside from steroid atrophy and folliculitis, these are very safe options. When atrophy is seen and treatment is halted, it usually will resolve; if appropriately informed, many patients will tolerate mild atrophy if it occurs under a blanket of regrown hair. In more widespread disease, use of contact immunotherapy by application of a contact dermatitis sensitizer to involved skin will also allow regrowth, but referral to a dermatologist is recommended for this approach, as it is more complicated and can result in severe allergic or irritant contact dermatitis even in experienced hands. There is also a role for minoxidil as an adjunct topical therapy with any of the aforementioned treatments. Aside from systemic steroids, whose chronic use is not recommended for the months to years of expected disease, systemic agents have been disappointingly ineffective in most cases.

**Telogen Effluvium**

TE is a very common, usually temporary, shedding (effluvium) of hairs of the telogen-phase hair follicles. It is much more common in women than in men, and is the most common cause of effluvium in adult women. It usually starts abruptly, causing significant concern to the patient. Hair is easily pulled out at the root and patients will describe it collecting in their beds, on their brushes or combs, or in the shower drain. A decreased thickness of the ponytail is also a common complaint, as is a pile of shed hair offered to the provider as proof of a problem. Hair is lost uniformly throughout the scalp rather than in discrete circles as in AA, or just from the crown as in FPHL or AGA. As preexisting FPHL and AGA are common in the population, however, TE may unmask clinically subtle cases, resulting in a generalized effluvium that leaves the crown, vertex, or temples apparently more affected, as discussed in the section on AGA/FPHL.

The hair-follicle cycle is composed of a growth phase (anagen), an involution phase (catagen), and a resting phase (telogen), which occurs asynchronously among the 100,000 or so hair follicles on the scalp. After completing the telogen phase, the hair cycle restarts and produces a new anagen hair, releasing the old club telogen hair currently occupying that follicle. After a triggering event, an increased number of hairs leave anagen phase and enter catagen and then telogen phase in synchrony (anagen release), resulting in a sharp increase in shedding of club hairs approximately 3 months later as telogen ends.

Common triggers include childbirth (postpartum effluvium or telogen gravidarum), or any significant physical or emotional trigger such as trauma, major surgery, strict diets, or emotional stress such as divorce or death of a loved one. However, approximately one-third of cases have no identifiable trigger, making TE sometimes a diagnosis of exclusion.

Diagnosis is by recognition of a generalized effluvium that is sudden in onset, generally after a clear trigger. A hair-pull test will produce club hairs and will be positive in all areas of the scalp examined, rather than just the crown or in discrete patches. A skin biopsy can help distinguish TE from a diffuse form of AA, or from FPHL, AGA, and other less common causes of alopecia. An increased proportion of telogen-phase hairs is seen by histology with lack of inflammation or scar, confirming the diagnosis.
Treatment is reassurance that the patient will not lose all of the hair as the hairs are being pushed out by new ones growing in. TE is self-limited, so doing nothing is appropriate. Highly concerned patients can be instructed to collect and count all of their fallen hairs daily from the sources mentioned earlier and elsewhere in their environment. Patients can track the number of hairs and thus be reassured when it returns to a normal amount (about 50–100 hairs per day is a normal average number of hairs to shed).  

A chronic form of TE also exists whereby the condition persists for more than 6 months. This form may result from a chronic systemic medical or emotional disease, essentially an ongoing trigger. Exclusion of reversible causes is of course required to treat this condition, although it is commonly idiopathic. Again, patients can be reassured that their hair is regrowing constantly and will not all be lost, but it may not be possible to predict when it may normalize if a trigger is not identified, and may often persist for years.

**FACIAL RASHES**

Facial rash is a fairly common complaint in the primary care setting. Learning how to approach the evaluation of facial rashes is a valuable skill. This section discusses the common presentations of facial rashes and how to distinguish between clinical conditions, and discusses treatments for primary care providers to appropriately manage rashes and recognize clues to more severe illness that may require hospitalization or subspecialty management. The conditions addressed are acne vulgaris, acne rosacea, periorificial dermatitis, seborrheic dermatitis, erysipelas, cellulitis, and SLE (Table 3). In general, when evaluating a facial rash the first question to ask is whether the patient appears ill or complains of systemic symptoms. If the response is affirmative then erysipelas, cellulitis, or SLE moves to the top of the list. When considering the rashes of acne vulgaris, acne rosacea, periorificial dermatitis, or seborrheic dermatitis, age of onset of symptoms may help narrow the differential. The presence of symptoms during adolescence suggests acne vulgaris, whereas acneiform conditions developing sometime after the age of 30 years are more likely to be acne rosacea. Finally, the distribution of the rash as well as its characteristics (acneiform, eczematous, comedonal, and so forth) will further aid in proper diagnosis.

**Acne Vulgaris**

Acne vulgaris is the most common skin condition affecting young adults. Estimates of prevalence rates range from 35% to 90% in adolescents. Although most cases of acne vulgaris start in adolescence and resolve by the mid 20s, there is evidence that up to 50% of patients will have persistent symptoms into later adulthood. It is important for the primary care provider to recognize that this is a chronic, inflammatory condition that significantly affects quality of life and often results in anxiety, depression, and social withdrawal. Acute treatment followed by maintenance therapy can improve dermatologic and quality-of-life outcomes.

Understanding the pathogenesis of acne vulgaris helps to differentiate this from other acneiform facial rashes. It starts with androgen-induced increased sebum production in the pilosebaceous unit followed by abnormal keratinization, which occludes the follicle and leads to the development of a microcomedone. Bacterial colonization of the hair follicle with *Propionibacterium acnes* triggers a host immune response, leading to the development of inflammatory lesions. Understanding that the microcomedone is the base lesion for all types of acne vulgaris aids in the differentiation of acne vulgaris from acne rosacea and periorificial dermatitis, and
also helps explain why keratinolytics are necessary in the treatment of all types of acne vulgaris (comedonal and inflammatory). When evaluating a patient with acneiform lesions of the face, the most important thing to look for is the presence of comedones (closed comedones are known as whiteheads, open comedones as blackheads) (Fig. 4). These lesions will be present in acne vulgaris but not in acne rosacea or periorificial dermatitis, as the microcomedone is not involved in the pathogenesis of the latter conditions. In addition to affecting the face, acne vulgaris can involve the back, chest, shoulders, and torso, which are the areas with the highest sebaceous gland activity.

Acne vulgaris can range in severity from mild comedonal, to inflammatory (Fig. 5), to severe nodulocystic (see Fig. 5; Fig. 6); however, addressing the issue of abnormal keratinization with a topical keratinolytic is mandatory for the management of this chronic condition regardless of the type of acne. Setting appropriate expectations for improvement is also important. Treatments for acne vulgaris do not address the lesions that are already present, but rather prevent the development of new lesions. It takes approximately 8 weeks for a microcomedone to mature, so patients should

### Table 3
Differential diagnoses for facial rashes

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<th>Condition</th>
<th>History/Demographics/Risk Factors</th>
<th>Physical Examination Findings</th>
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<tr>
<td>Acne vulgaris</td>
<td>Starts in adolescence</td>
<td>Comedones (whiteheads or blackheads) should always be present, inflammatory papules</td>
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<td>Acne rosacea</td>
<td>Tends to present between ages 30 and 60 y, more common in light-skinned individuals or Celtic or Northern European descent</td>
<td>Flushing, nontransient erythema, telangiectasias, inflammatory papules, no comedones, sebaceous gland hypertrophy</td>
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<td>Periorificial dermatitis</td>
<td>More common in women ages 16–45 y, history of topical or inhaled corticosteroid exposure</td>
<td>1–2 mm clustered erythematos papules, papulovesicles or papulopustules with or without mild scale, located around mouth and/or the eyes, spares the vermilion border</td>
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<td>Seborrheic dermatitis</td>
<td>Peak incidence third to fourth decades, human immunodeficiency virus (HIV) associated with severe cases of seborrheic dermatitis</td>
<td>Greasy-looking, yellowish or erythematous scale affecting scalp, nasal ala, eyebrows, glabella, nasolabial fold</td>
</tr>
<tr>
<td>Erysipelas</td>
<td>Often complain of fevers and chills</td>
<td>Well-demarcated, shiny, erythematous, painful plaque with associated swelling and perifollicular edema</td>
</tr>
<tr>
<td>Systemic lupus erythematosus</td>
<td>Indolent and intermittent presentation of rash, most often affects young African American women</td>
<td>Flat or raised, painful or pruritic, no comedones, papules, or pustules, triggered by sun exposure, butterfly pattern sparing the upper lip and nasolabial fold. The nose acts as a sun shade to these areas</td>
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be advised that they may not see any improvement in their skin for several months.

Topical keratinolytics are available over the counter or by prescription. Over-the-counter options include tea-tree oil, benzoyl peroxide, and salicylic acid. Prescription agents include topical retinoids and topical azelaic acid. Recent recommendations

Fig. 4. Comedonal acne. Note the closed comedones in (A) and open comedones in (B), most of which are noninflammatory. ([A] From Brinster NK, Liu V, Diwan AH, et al. Dermatopathology: high-yield pathology. Philadelphia: Saunders; 2011; with permission; and [B] From Benner N, Sammons D. Overview of the treatment of acne vulgaris. Osteopathic Family Physician 2013;5(5):185–90; with permission.)

Fig. 5. Inflammatory acne. Note the erythematosus papules and pustules. (From Habif TP, Campbell JL, Chapman MS. Skin disease: diagnosis and treatment. Philadelphia: Elsevier/Saunders; 2011; with permission.)
from the Global Alliance Improve Outcomes in Acne Group recommend using topical retinoids as first-line agents when possible. This recommendation is based on the fact that topical retinoids are more efficacious than the over-the-counter options or azeleic acid, and that topical retinoids also improve penetration of other topical agents such as antibiotics if they become necessary.

Topical retinoids should only be applied at night because they can be deactivated by light and increase the risk of photosensitivity. Side effects include redness, dry skin, peeling, and burning. Starting at a lower potency and working up to the highest potency tolerated will improve efficacy and decrease the risk of side effects. Warning patients that they may have a pustular flare on initiating treatment is also important, as adherence rates are likely to decrease if patients are unaware that their skin may worsen before it improves. Cost and side-effect profile will influence the choice of topical retinoid. The least expensive option is tretinoin (Retin-A, Atralin, and so forth), the most potent is tazarotene (Tazorac), and the least likely to irritate the skin is adapalene (Differin). If patients cannot afford or cannot tolerate a topical retinoid, one of the other over-the-counter or prescription options should be considered. Tea-tree oil, salicylic acid, and benzoyl peroxide are similarly efficacious for the treatment of comedonal acne. Benzoyl peroxide or tea-tree oil can also be used as a single agent for the treatment of inflammatory acne. Tea-tree oil is generally well tolerated but may cause an allergic contact dermatitis. Salicylic acid is useful in patients with sensitive skin. Benzoyl peroxide may bleach clothes, towels, and sheets, so patients should be warned to wash off the product entirely or make sure it is dry or fully rubbed into the skin so that clothes or bedding are not ruined. Linens and towels made with benzoyl peroxide–resistant dyes are also available. Azeleic acid (Azelex, Finacea) is a prescription agent that can be used as a single agent for comedonal or inflammatory acne. It is generally well tolerated in patients with sensitive skin, but is more costly. It has the potential of causing hypopigmentation of the skin, which may be beneficial in patients with postinflammatory hyperpigmentation.

If patients have primarily inflammatory acne, an antibiotic may need to be added to the keratinolytic. Recent evidence has demonstrated the development of *Propionibacterium acnes* antibiotic resistance; therefore, limiting the use of topical and oral

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**Fig. 6.** Nodulocystic acne. Note the deeper papulonodules and cystic lesions along the jawline and scarring on the malar cheeks. (From Paller AS, Mancini AJ. Hurwitz clinical pediatric dermatology. Philadelphia: Elsevier/Saunders; 2011; with permission.)
antibiotics to patients who really need them and for the least amount of time possible is recommended.\textsuperscript{32,34,35} Ideally antibiotics would be limited to 3 months of therapy. Evidence suggests that if antibiotics are needed for longer than 2 months, patients should also be started on topical benzoyl peroxide or azeleic acid to minimize the development of resistance at sites of application.\textsuperscript{32,36} Benzoyl peroxide or azeleic acid can be applied continuously to prevent this resistance. In addition, oral and topical antibiotics should never be used as monotherapy for acne; they should always be combined with a keratinolytic.\textsuperscript{35} Once a patient’s inflammatory acne has improved, the antibiotics should be halted and the keratinolytic continued for maintenance therapy, with benzoyl peroxide or azeleic acid added for antimicrobial effect if needed.\textsuperscript{32}

The latest studies suggest that anti-inflammatory action of antibiotics is the most likely mechanism in the treatment of acne vulgaris and acne rosacea.\textsuperscript{36} With this information, the focus has shifted to using topical antibiotics or considering the use of sub–antimicrobial dose oral antibiotics. At this point, data demonstrate that sub–antimicrobial doses are effective in treating acne, but there is not enough known about whether these lower doses also decrease the risk of antibiotic resistance.\textsuperscript{35} Topical antibiotic options include clindamycin, erythromycin, dapsone, or sulfur/sulfacetamide. These products come alone or in some cases are combined with a retinoid or benzoyl peroxide, and studies have demonstrated that such combinations are more effective in the treatment of inflammatory acne than any of the agents used alone.\textsuperscript{36} Clindamycin or dapsone topicals can be stored at room temperature. Erythromycin–containing products must be refrigerated. Topical dapsone 5\% (Aczone) is a relatively new agent used in the treatment of acne. Despite its risk when used in the oral formulation, topical dapsone is safe in patients with glucose–6–phosphate dehydrogenase deficiency or sulfonamide allergies.\textsuperscript{36,37} There have been no direct comparisons of topical dapsone with topical clindamycin or erythromycin. The cost of dapsone in comparison with these other agents likely outweighs any benefits of the product at present.

Oral antibiotics may be needed for moderate to severe inflammatory acne. Tetracyclines such as doxycycline or minocycline are mainstays of therapy. There is no difference in efficacy between these 2 antibiotics\textsuperscript{36}; however, because of the side-effect profiles of both, doxycycline is generally recommended as the first-line agent. Side effects of doxycycline include gastrointestinal upset, pill esophagitis, and photosensitivity. Minocycline may cause vestibular symptoms, irreversible bluish-gray skin discoloration, and drug-induced lupus. Traditional dosing for both is 100 mg once or twice a day; however, there may be future recommendations for sub–antimicrobial dosing regimens such as 20 mg twice a day. There are variations in brand name, which are enteric coated (Doryx) or extended release (Oracea or Solodyn). For those who are allergic to tetracyclines, the macrolide azithromycin may be an option. Randomized controlled trials comparing azithromycin with doxycycline, minocycline, and tetracycline demonstrate that it is at least equally effective and in some cases even more effective.\textsuperscript{36} In these studies azithromycin was dosed in some form of pulsed dosed therapy. However, a recommended dosing regimen has yet to be determined. (Usual dosing options: 500 mg 3 times per week for 1 month followed by 500 mg twice weekly for 1 month, followed by once weekly for 1 month; or 500 mg on the first day followed by 4 consecutive days of 250 mg, repeated on the 1st and 15th of every month.)\textsuperscript{36} Azithromycin is a safe option during pregnancy and breastfeeding, whereas all the tetracyclines are contraindicated. Not enough data exist to determine whether cephalosporins or fluoroquinolones are effective in the treatment of acne.\textsuperscript{36} Finally, patients with moderate acne who do not respond to other therapies or those with severe/nodulocystic acne should be referred to Dermatology to discuss treatment with oral isotretinoin (formerly known by the brand Accutane).
Acne Rosacea

Like acne vulgaris, acne rosacea is a chronic inflammatory disorder with periods of exacerbation and remission. Unlike acne vulgaris, it tends to present in the 30s to 60s rather than during adolescence. It most commonly affects light-skinned individuals of Celtic and Northern European descent. Rosacea can affect the central face, nose, cheeks, eyelids, forehead, and chin. Symptoms include flushing, nontransient erythema, telangiectasias, papules, pustules, nodules, cysts, or sebaceous gland hypertrophy (eg, rhinophyma) (Fig. 7). A patient may have any one of these findings or a combination, and symptoms can range from mild to severe. Ocular involvement with blepharitis or conjunctivitis is present in more than 50% of patients, and may be found in the absence of other skin manifestations of rosacea. To differentiate rosacea from acne vulgaris one should look for the presence of comedones; acne vulgaris will have comedones whereas rosacea will not. The pathogenesis of rosacea is not well understood but seems to be a combination of factors including abnormalities in innate immunity, inflammatory reactions to cutaneous microorganisms, ultraviolet damage, and vascular dysfunction. Advising patients to avoid excessive sun exposure is important, as is setting clear expectations for the benefits of therapy. Topical and oral therapies work well for papules and pustules, but less so or not at all for flushing, redness, or telangiectasias. It may take 2 to 3 months before one sees any benefit from therapy, and because this is a chronic condition maintenance therapy is recommended.

Randomized controlled trials demonstrate equal efficacy for topical metronidazole 0.75% to 1% and azelaic acid 15% to 20%, although topical metronidazole is better tolerated and less expensive. Other options include topical sulfacetamide 10%/sulfur 5% or combination benzoyl peroxide/clindamycin; however, the data for efficacy are less robust. Data supporting the use of monotherapy with topical clindamycin or erythromycin are limited, and because of the potential risk of antibiotic resistance should be avoided. Only one small study of 50 patients evaluated the topical retinoid adapalene in comparison with topical metronidazole. Adapalene was better at reducing inflammatory lesions, although the metronidazole-treated patients had less erythema. There was no difference in the scores for erythema or telangiectasia.

Oral antibiotic therapies are similar to those used in the treatment of acne vulgaris, and include doxycycline, minocycline, and tetracycline. Oral antibiotics should be considered in patients with moderate to severe rosacea, ocular rosacea, and in those not responding to topical therapies. Whether traditional dosing versus sub-antimicrobial dosing should be used is not yet clear. The only systemic FDA-approved therapy for rosacea is once-daily sub–antimicrobial dose doxycycline (Oracea). Two double-blind, randomized, placebo-controlled trials demonstrated an average 61% reduction in inflammatory lesions, compared with 29% with placebo. Again, duration of use of antibiotics should be limited to the shortest time course possible. A few studies demonstrate added benefit when combining oral antibiotics with topical metronidazole. Maintenance therapy with a topical agent such as metronidazole or azelaic acid should be continued long term. The efficacy of oral isotretinoin in the treatment of rosacea has not been well established, but referral to Dermatology should be considered for patients with moderate to severe disease or the development of focal enlargement resulting from sebaceous gland hypertrophy (eg, rhinophyma) in an attempt to prevent poor cosmetic outcomes.

Unfortunately there are few pharmacologic options for the treatment of flushing. A variety of agents have been tried, including clonidine, β-blockers, antidepressants, gabapentin, topical oxymetazoline, and the newly approved topical brimonidine gel, but the data are limited and the side-effect risks are significant. Avoidance of triggers...
such as extremes of temperature, sunlight, spicy foods, and alcohol may be effective, but difficult to adhere to in most cases. Referral to Dermatology for pulsed-light therapy and laser therapy can be considered for the treatment of telangiectasias and nontransient erythema.43

**Periorificial Dermatitis**

Periorificial dermatitis, also called perioral dermatitis, typically presents with multiple small inflammatory papules around the mouth, nose, or eyes. Although the name suggests that the condition may have an eczematous appearance, it most closely resembles an acneiform or rosacea-like eruption with or without eczematous features. This condition can occur in individuals of all ethnic and racial backgrounds, with more than 90% of cases affecting women between the ages of 16 and 45 years.44,45 Pathogenesis of this condition is poorly understood; however, the clinical and histologic features of the lesions resemble those of rosacea. Exposure to external elements likely triggers these lesions, the strongest association being with topical or inhaled corticosteroids. Other triggers may include fluorinated toothpastes, heavy face creams and moisturizers, especially those with a petrolatum or paraffin base, and certain cosmetics such as lipsticks.44

The classic patient history may describe a mild papular or slightly scaly facial rash that initially improves with topical corticosteroid use, but either recurs or worsens with continued use or attempts to discontinue corticosteroid therapy. Periorificial dermatitis manifests with 1- to 2-mm clustered erythematous papules, papulovesicles, or papulopustules with or without mild scale. It most often affects the perioral region, but can also involve the nasolabial fold and area around the eyes or eyelids. The absence of comedones will rule out acne vulgaris. The presence of vermilion border sparing (a clear zone of 3–5 mm near the edge of the lip) around the perioral region argues for periorificial dermatitis and against acne rosacea (Fig. 8). Seborrheic dermatitis presents with erythema and scale most commonly around the nasal ala, scalp, eyebrows, and nasolabial folds. Small papules are not a feature of seborrheic dermatitis, nor is a perioral distribution.

The most important intervention is to stop the use of the inciting agent. Periorificial dermatitis may resolve on its own without therapy within a few months if topical corticosteroids or other triggers can be eliminated. Most patients will request therapy, however, as the initial flare of their rash can be distressing once the steroids or other triggers are stopped. Tapering from a higher-potency steroid to a lower-potency

![Fig. 8. Periorificial dermatitis. Note the papules surrounding an area of sparing around the vermilion border of the perioral area. (From Habif TP. Clinical dermatology: a color guide to diagnosis and therapy. 5th edition. Philadelphia: Elsevier; 2010; with permission.)](image)
steroid such as hydrocortisone 1% over a few months, and then discontinuing entirely, may reduce the likelihood of a significant flare, but this approach has not been well studied. For mild to moderate cases, topical calcineurin inhibitors (pimecrolimus or tacrolimus) should be considered. Most of the placebo-controlled randomized trial data support the use of pimecrolimus 1% cream twice a day, with most benefit shown in the first few weeks of therapy. Therapy should continue for about 4 weeks, then be either discontinued if effective or changed to a different therapy if ineffective. The data supporting the use of topical antibiotics such as erythromycin or metronidazole are limited, but if cost is an issue with pimecrolimus it might be worth trying one of these agents for up to 8 weeks for mild to moderate cases.

The use of an oral tetracycline has also demonstrated efficacy in small, randomized controlled trials. One trial of 120 patients comparing placebo with oral tetracycline 500 mg twice daily for 10 days then 250 mg twice daily for 10 days or topical erythromycin 1% demonstrated similar increased rates of improvement in the oral tetracycline and topical erythromycin groups compared with placebo. Another multicenter randomized trial of 108 patients compared oral tetracycline 250 mg twice daily with twice-daily application of metronidazole 1% cream for 8 weeks. Both groups showed improvement in lesion counts, although the oral antibiotic group improved faster. There are no studies examining the efficacy of doxycycline or minocycline; however, these regimens are often tried for 8 weeks because of their advantage of fewer restrictions on timing of administration. Small, uncontrolled studies or case reports have shown some benefit with azelaic acid 20% cream, topical tetracycline, topical adapalene 0.1% gel, topical clindamycin with or without hydrocortisone 1%, and topical sulfacetamide-sulfur plus oral tetracycline.

**Seborrheic Dermatitis**

Erythema accompanied by greasy-looking, yellowish scale in the eyebrows, glabella, lateral nasal areas, and nasolabial folds is characteristic of seborrheic dermatitis (Fig. 9). The mustache and beard (if present), scalp, external ear canals, chest, axilla, and groin may also be affected. Seborrheic dermatitis may begin in infancy (cradle cap) but typically resolves, often recurring later in life with the peak incidence during the third and fourth decades of life. Most individuals are healthy; however, there is a relationship between human immunodeficiency virus (HIV) infection and seborrheic dermatitis, with 35% of early HIV patients and up to 85% of patients with AIDS affected. Severe seborrheic dermatitis is more common with advancing HIV disease. If a patient presents with severe seborrheic dermatitis and risk factors for HIV infection, screening for HIV is appropriate. Other causes of immunosuppression such as lymphoma, or neurologic conditions such as Parkinson disease or unilateral involvement after a cerebrovascular accident, can occur as well. The cause of seborrheic dermatitis is unknown but is likely related to overgrowth of *Malassezia* species that thrive on the oils in sebum, possibly explaining why areas rich in sebaceous gland activity are predisposed to this condition. Seborrheic dermatitis can mimic rosacea, but rosacea is associated with telangiectasias and papulopustular lesions, has minimal or no scale, and more often affects the nose, malar, and perioral regions. However, seborrheic dermatitis and rosacea often occur concurrently.

Seborrheic dermatitis is a chronic, relapsing condition that flares with psychological stress, changes in weather, or lack of regular shampooing; therefore, acute treatment followed by maintenance therapy is required. For acute treatment, either topical low-potency corticosteroids or antifungals are effective. For facial symptoms, a low-potency steroid (group 6 or 7) or antifungal creams such as ketoconazole 2% or ciclopirox may be used once or twice a day until symptoms resolve. For maintenance
therapy for facial symptoms, intermittent use of topical antifungals (weekly ketocona-zole 2% cream or shampoo wash to the face) or emollient therapies is effective. Long-term use of topical steroids should be avoided for maintenance therapy to prevent telangiectasias and atrophy. Randomized controlled trials have demonstrated equal efficacy between pimecrolimus cream, topical steroids, and topical antifungals; however, topical calcineurin inhibitors are costly, associated with short-term side effects, and not FDA approved for the treatment of seborrheic dermatitis. For scalp symptoms, antifungal shampoos such as selenium sulfide 2.5%, ketoconazole 2%, or ciclopirox 1% are effective. The shampoo should be left on for 5 to 10 minutes before rinsing off. Daily or thrice-weekly use until symptoms are improved should then be followed by weekly maintenance therapy. Tachyphylaxis is common with these shampoos, so rotating to a different type every few months can help maintain remission. A high-potency steroid (groups 1–3) shampoo, lotion, or foam may be added daily to control severe itching, although treatment should be limited to 4 weeks. Fluocinolone aceto-nide 0.01% shampoo is approved by the FDA for the treatment of seborrheic dermatitis.

**Erysipelas and Cellulitis**

Erysipelas (also known as St Anthony’s fire) and cellulitis are skin infections most often caused by β-hemolytic streptococci (groups A, B, C, G, and F) followed by methicillin-sensitive *Staphylococcus aureus* (MSSA) or methicillin-resistant *S aureus* (MRSA) in high-risk populations. The lower extremities are most often involved, although facial infections can occur. Erysipelas affects the upper dermis and superficial lymphatics, and classically presents with a rapidly expanding, well-demarcated, shiny, erythematous, painful plaque associated with swelling and perifollicular edema (peau d’orange) often in a malar distribution (Fig. 10). Systemic symptoms such as fever, chills, and
malaise are often present.\textsuperscript{30} Cellulitis affects the deeper dermis and subcutaneous fat, and differs from erysipelas in that it is generally less well demarcated, will have little or no edema, and few if any systemic symptoms early in the course. Infection involving the ear or “Milian’s ear sign” is a finding unique to erysipelas and does not occur in cellulitis because the ear does not contain deeper dermis tissue (Fig. 11). Antecedent breaks in the skin allowing portals of entry are common for these infections when they involve the lower extremities but not when they occur on the face. Treatment with antibiotics such as dicloxacillin or cephalexin that cover both \( \beta \)-hemolytic streptococci and MSSA are necessary. If the patient is penicillin allergic, a broad-spectrum quinolone can be used. If there is concern for MRSA (purulent discharge or abscess), the addition of trimethoprim-sulfamethoxazole or doxycycline may be added. Clindamycin alone may treat both \textit{Streptococcus} species and community-acquired MRSA in some communities, although inducible resistance is common in MRSA. Linezolid alone should cover streptococci and MRSA, but this is a costly alternative. Parenteral antibiotics should be used if patients have systemic symptoms.

\textbf{Systemic Lupus Erythematosus}

The presence of a malar rash is a classic finding in SLE (Fig. 12). The rash can be raised or flat, and may be pruritic or painful. The absence of papules, pustules, and comedones helps to differentiate it from acne rosacea or acne vulgaris. The indolent and intermittent presentations of the SLE rash differentiate it from erysipelas and cellulitis. The malar rash of SLE is triggered by sun exposure and is described as a butterfly pattern. The butterfly appearance is due to the angle at which the sun’s ultraviolet rays land on the skin with the nose acting as a sunshade, thus sparing the upper lip and nasolabial fold. Seborrheic dermatitis commonly affects the nasolabial fold, hence aiding in the differentiation between these 2 conditions.\textsuperscript{30} Treatment of the SLE rash may
Fig. 11. Milian’s ear sign of erysipelas. (From Habif TP, Campbell JL, Chapman MS. Skin disease: diagnosis and treatment. Philadelphia: Elsevier/Saunders; 2011; with permission.)

Fig. 12. Malar rash of SLE. Rash predominantly involves the malar cheeks and nose but spares the upper lip and nasolabial folds. (From Doherty M, Ralston SH. Musculoskeletal disease. In: Colledge NR, Walker BR, Ralston SH, editors. Davidson’s principles and practice of medicine. 21st edition. Philadelphia: Elsevier; 2010. p. 1053–129; with permission.)
require systemic therapies targeting the underlying disorder along with topical steroids, although sun protection is a mandatory prophylactic measure.

INTERTRIGINOUS RASHES

In the primary care setting, patients often present with rashes in their intertriginous areas. The word intertrigo comes from the Latin inter (between) and terer (to rub), and refers to a condition involving friction and maceration caused by skin against skin. It often is used to refer to this irritant dermatitis without superinfection of any type, but the term may be also be used when infection is present. The most commonly involved sites include axilla, groin, anogenital region, web spaces of toes and fingers, and skin folds of the breast or pannus. These moist areas provide an ideal breeding ground for yeast, fungal, or bacterial superinfections. Other noninfectious inflammatory conditions may also develop in these areas and are often misdiagnosed. Because treatments differ depending on the etiology, some clinical pearls are presented here to help determine the cause and allow tailoring of appropriate therapy (Table 4).

<table>
<thead>
<tr>
<th>Table 4</th>
<th>Differential diagnoses for intertriginous rash</th>
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<tr>
<td><strong>Condition</strong></td>
<td><strong>History/Demographics/Risk Factors</strong></td>
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<tr>
<td>Candidiasis</td>
<td>Usually complain of severe pruritus</td>
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<td></td>
<td>Higher risk in moist skin folds, such as in obesity</td>
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<td></td>
<td>Higher risk in immunosuppressed states such as HIV or diabetes mellitus</td>
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<tr>
<td>Tinea cruris</td>
<td>More common in men</td>
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<tr>
<td></td>
<td>Often multiple sites infected simultaneously (tinea cruris, tinea pedis, tinea manus)</td>
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<tr>
<td>Erythrasma</td>
<td>Usually asymptomatic or only mild itching</td>
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<tr>
<td>Inverse psoriasis</td>
<td>One-third of patients will report family history of psoriasis</td>
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In addition to a clear history of risk factors and symptoms and the appearance of the rash, the regular use of a potassium hydroxide (KOH) prep and a Wood’s lamp can help in the diagnosis. A general approach to all types of intertrigo is to educate patients about keeping skin folds dry and avoiding friction. Drying the intertriginous areas thoroughly with a cool hairdryer if possible; wearing light, nonconstricting, absorbent clothing; and avoiding wool and synthetic fibers can be helpful. Absorptive powders or barrier creams may also help decrease moisture or limit friction. Cornstarch rather than talcum powder should be used as a drying agent in women in the genital area, as there has been an association reported between use of talcum powder and an increased risk of ovarian cancer.

KOH preps can easily be done in the primary care setting. Use a clean glass slide to collect scale that falls while scraping material from a lesion using the edge of another glass slide, or wet with an alcohol swab and then use a #15 blade to scrape. The highest-yield areas for hyphae are from the leading edge of the lesion, papule, pustule, or vesicle. Apply a coverslip to the slide to transport to the microscope area, then 1 to 2 drops of KOH to one edge of the coverslip: it will wick under the coverslip by capillary action. Gently heat the slide over an alcohol burner or with a lighter for a few seconds until just before it starts to boil. This technique breaks down cell-membrane components and makes it easier to see yeast or fungal elements. Repetitive gentle pressure to the top of the coverslip with a pen or probe will help flatten the specimen once it starts digesting, and will make examination easier. Examine the slide at 10× magnification and increase to 20× if needed to confirm the presence of the pseudohyphae or yeast forms of candida or the hyphae of dermatophytes. It is important to be aware that KOH preps can often yield false-negative results and that many structures (hair fibers, clothing fibers, KOH crystals, or the edges of folds of the sheets of cells) can mimic hyphae and cellular debris from digestion can mimic yeast forms (Fig. 13). Gram stains, if available, will highlight yeast forms.

Candidiasis
The warm moist environment of intertriginous areas provides an ideal location for the growth of candida. Obesity, hyperhidrosis, incontinence, and tight clothing lead to increased friction, and moisture in the area and immunosuppression secondary to either medical conditions or medications may promote fungal overgrowth. Refractory candidal intertrigo without a clear explanation may raise the possibility of an underlying condition such as malnutrition, endocrinopathy, malignancy, or HIV infection.

The classic description of candidal intertrigo is an erythematous, macerated plaque with erosions, delicate peripheral scale, and satellite papulopustules (Fig. 14). Patients generally describe severe pruritus or burning, and may develop painful skin breakdown. The diagnosis can be made clinically, but KOH prep or culture of skin scrapings can be helpful. Pruritus and satellite lesions are the most clinically helpful for diagnosis of candida. KOH prep may reveal oval budding yeasts with pseudohyphae (see Fig. 13B).

There are few data from controlled trials comparing the different topical antifungal agents for the treatment of candida. In general, twice-daily treatment until symptom resolution with any of the following agents is acceptable: nystatin, the azoles ( clotrimazole, ketoconazole, miconazole, econazole, sertaconazole), or hydroxypridone ( ciclopirox). Of note, all of these agents will also work for dermatophyte infections except for nystatin, which is only effective against candida. By contrast, the allylamines (terbinafine and naftifine) and benzylamines (butenafine) are the most effective topical agents against dermatophytes, but have less activity against yeasts. Some patients may benefit from twice-weekly applications in preventing recurrence. In general cream

Fig. 14. Candidal intertrigo. Erythematous, well-demarcated plaques with satellite papules and pustules. (From Habif TP. Clinical dermatology: a color guide to diagnosis and therapy. 5th edition. Philadelphia: Elsevier; 2010.)
formulations are more potent, although antifungal powders may have the added benefit of providing a drying effect. Consider using creams for treatment of acute infections and powders for maintenance therapy. In most cases, treatment with an antifungal will lead to rapid resolution of symptoms, but the addition of a low-potency topical steroid may help with the pruritus, pain, and burning. Moderate-potency and high-potency topical steroids should be avoided in intertriginous areas because of their increased potency and side effects when used under occlusion.

If topical therapy fails or the infection is severe, consider using oral antifungal agents. Oral azoles can be used for 2 to 6 weeks until symptoms have resolved. In adults, common dosing regimens include: fluconazole, 50 to 100 mg daily or 150 mg weekly; itraconazole, 200 mg twice daily; or ketoconazole, 200 mg daily. Griseofulvin has no activity against candida. The patient should be monitored for drug interactions with oral azoles.

Dermatophyte Infections

Tinea cruris occurs more often in men than in women. As opposed to candidal groin infections, which usually simultaneously involve the thigh and scrotum in men, tinea infections generally start on the inner thigh with a small red patch and then spread outward from the center, first down the leg and then into the pubic region. The scrotum is typically spared in tinea infections, which differs from candidal infections. Moreover, tinea cruris should not have papulopustular satellite lesions. The tinea lesion is usually slightly elevated, with an erythematous and sharply demarcated border and partial central clearing (Fig. 15). Using a hand lens may reveal very small vesicles at the

Fig. 15. Tinea cruris. Note the expanding annular red rim with partial central clearing along the inner thigh. (From Elewski BE, Hughey LC, Sobera JO, et al. Fungal diseases. In: Bolognia JL, Jorizzo JL, Schaffer JV, editors. Dermatology. 3rd edition. Philadelphia: Elsevier/Saunders; 2012. p. 1251–84; with permission.)
border. If tinea cruris is suspected, look for other sites of tinea infection such as the buttocks, feet, toenails, or hands, as multiple sites are often affected and patients can reinfect the groin area if other sites of infection are not also treated. A KOH prep from the active border of the lesion may show long, filamentous, and segmented hyphae (see Fig. 13A). In addition to the azoles already mentioned, allylamines (terbinafine and naftifine), benzylamines (butenafine), and hydroxypyridone (ciclopirox) are highly effective against most dermatophytes, whereas nystatin is not. In general, symptoms will resolve within 2 weeks with treatment. Encouraging patients to apply the creams to at least 3 cm beyond the advancing edge of the lesion, treating for 1 week after clinical clearing, and treating other sites of active infection may help to prevent recurrence. Treatment of onychomycosis may be necessary if patients continue to develop recurrent infection. For cases resistant to topical therapy, and severe or recurrent cases, oral treatment with griseofulvin, 250 mg 3 times daily for 14 days, or any of the previously mentioned oral azoles or terbinafine can be tried.

**Erythrasma**

Erythrasma is a superficial infection of the skin caused by *Corynebacterium minutissimum*, a gram-positive, non-spore-forming bacillus. Most patients will be asymptomatic and often present for treatment for cosmetic reasons. Some patients may complain of mild itching. The most common site of infection is the web spaces between the toes followed by the groin and axilla. In the interdigital areas, macerated, scaly plaques are common, and closely resemble the findings of tinea pedis. In the groin or axilla, erythrasma may present with well-demarcated red patches or plaques that eventually turn a lighter brownish color. Fine-scale and “cigarette-paper” wrinkles are often apparent (Fig. 16A). Assuming that the patient has not recently scrubbed the affected area in an attempt to get it clean, a Wood’s lamp examination may reveal a coral-red fluorescence that develops after 5 to 10 seconds, owing to the production of porphyrins by the *C. minutissimum* (see Fig. 16B). A KOH prep may also be performed because concomitant infection with tinea or candida can occur.

Most cases can be treated with topical antibiotics or antifungals. There are few data available to guide therapy recommendations. Small studies support the use of clindamycin 1% or erythromycin 2%, 2 to 3 times daily for 1 to 2 weeks. Randomized

![Fig. 16. Erythrasma. (A) A slightly brownish, evenly colored patch is often seen without the erythema or annular character of other rashes in this area. (B) Porphyrins fluoresce coral-red on Wood’s lamp examination. (From Millett CR, Halpern AV, Reboli AC, et al. Bacterial diseases. In: Bolognia JL, Jorizzo JL, Schaffer JV, editors. Dermatology. 3rd edition. Philadelphia: Elsevier/Saunders; 2012. p. 1187–220; with permission.)](image-url)
controlled trials with small numbers of patients have demonstrated benefit with topical azoles twice daily (miconazole, tioconazole, econazole) or once daily (oxiconazole) for 7 to 60 days. Although no clinical trials support its use, benzoyl peroxide 5% gel has been reportedly effective based on clinical experience. Small studies show that Whitefield’s ointment (containing 12% benzoic acid and 6% salicylic acid) applied twice a day for 1 week is effective.

If patients have multisite disease or interdigital involvement, oral antimicrobials plus topical agents should be considered. One randomized, placebo-controlled trial comparing erythromycin (1 g daily × 14 days), clarithromycin (single dose 1 g), 2% fusidic acid (applied twice a day × 14 days [not available in the United States]), placebo cream, and placebo tablet showed the following complete response rates at 14 days based on a reflection score of 0 on Wood’s lamp examination: fusidic acid (97%), clarithromycin (67%), erythromycin (53%), placebo cream (13%), placebo tablet (3%).

Several other small, randomized controlled trials and case reports demonstrate efficacy of oral erythromycin. Erythromycin is usually prescribed at a dose of 250 mg 4 times daily for 14 days, but is associated with gastrointestinal upset and drug interactions. One study of tetracycline, 250 mg 4 times daily showed benefit, although it may be less effective than erythromycin. When considering the diagnosis and treatment of erythrasma multiple sites must be examined, as recurrence may occur if all sites are not treated. Data supporting prophylactic therapy with topical agents to prevent recurrence are not available.

**Inverse Psoriasis**

Plaque psoriasis is the most common variant of psoriasis, and typically develops in young adulthood. These plaques are generally raised and erythematous with a thick silvery scale, and develop on the extensor surfaces such as elbows and knees, umbilicus, scalp, back, and gluteal fold. Inverse psoriasis is a variant, which presents in flexural and intertriginous areas and is often misdiagnosed as a fungal or bacterial infection. Family history can be helpful because approximately one-third of patients with psoriasis will have a first-degree relative with this condition. Inverse psoriasis plaques are uniformly and evenly erythematous and well demarcated, but generally lack scale in comparison with plaque psoriasis (Fig. 17), likely because of the friction in these areas rubbing off the scale or the moist areas where it occurs. It also tends to

![Fig. 17. Inverse psoriasis of the breast in an obese woman. (From Berth-Jones J. Psoriasis. Medicine 2009;35:240; with permission.)](image-url)
be shinier than erythrasma, and should lack the pruritic papulopustular and satellite lesions of candida and the central clearing of a dermatophyte infection. Closely examining the fingernails for pitting, subungual keratosis, the oil-drop sign, and onycholysis can also be helpful, as 80% to 90% of patients with psoriasis will develop nail disease over their lifetime (Fig. 18). Negative KOH and Wood’s lamp examinations may also point toward inverse psoriasis as the cause. Inverse psoriasis is often diagnosed after multiple failed treatment attempts with antifungals or antibacterials. The condition can usually be diagnosed clinically, but biopsy may be necessary. Short-term therapy (2–4 weeks) with low- to moderate-potency topical steroids can be used to initiate therapy; however, long-term use of daily steroids in intertriginous areas should be avoided to prevent the development of atrophy, striae, and telangiectasias. Tachyphylaxis can also develop with continuous long-term use of topical steroids. If necessary, maintenance therapy can involve the use of topical vitamin D analogues (calcipotriene or calcitriol), topical calcineurin inhibitors (pimecrolimus or tacrolimus) or pulse-dosed topical steroids 1 to 2 times per week. Systemic therapies administered by a dermatologist may be needed for severe or refractory cases, although this is uncommon.

THE RED LEG

Patients often present to primary care providers with red rashes on the lower extremities. Differentiating between diagnostic possibilities is urgently needed to decide how a patient should be treated, as such rashes may require admission to a hospital or merely some compression stockings. This section discusses the clinical presentations of cellulitis and erysipelas, stasis dermatitis, allergic contact dermatitis, and DVT, and pearls for quickly differentiating between these possibilities through history taking and physical examination (Table 5).

Although ruling out infection may seem like the most pressing clinical priority, it is estimated that inappropriate treatment of cellulitis may account for more than a billion dollars in the Medicare system alone, in addition to the difficult to capture costs of adverse antibiotic reactions and complications of hospitalization. Key elements to consider in an initial assessment include the acuity of onset, differentiating pain versus pruritus as the sensory complaint, and the distribution of the rash, as well as...
predisposing factors such as preexisting leg edema, recent hospitalizations, or injuries. Treatments for each are also be briefly discussed.

Infectious entities tend to develop quickly in hours to days, whereas stasis dermatitis has a more insidious onset over the course of weeks. Pruritus is a predominant feature of dermatitis, whether from stasis or contact sensitization, whereas pain is predominant from the tissue destruction of infection or from DVT. Preexisting leg edema might suggest stasis dermatitis as a leading contender, whereas recent prolonged immobilization or surgery would push DVT higher in the differential. If symptoms are rapidly progressing over minutes to hours and are accompanied by pain out of proportion to examination and beyond the obvious area of involvement or a dusky gray to black color centrally, necrotizing fasciitis should be considered. Necrotizing fasciitis is a surgical emergency that is not medically treatable, and is not covered further herein.65

<table>
<thead>
<tr>
<th>Condition</th>
<th>History/Demographics/Risk Factors</th>
<th>Physical Examination Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cellulitis and erysipelas</td>
<td>Abrupt onset (hours to days)</td>
<td>Painful expanding area of redness</td>
</tr>
<tr>
<td></td>
<td>Higher risk with older age or obesity or chronic edema</td>
<td>Unilateral</td>
</tr>
<tr>
<td></td>
<td>Usually a portal of entry can be found</td>
<td>Crosses anatomic boundaries and may be discontinuous in its spread</td>
</tr>
<tr>
<td>Stasis dermatitis</td>
<td>Subacute onset (many days to weeks)</td>
<td>Scaly erythematous patches or plaques nearly continuous from the upper calf and shin to the top of the ankles, but usually not involving the joints or feet</td>
</tr>
<tr>
<td></td>
<td>Pruritic rather than painful</td>
<td>Often bilateral</td>
</tr>
<tr>
<td></td>
<td>Edema is always present</td>
<td></td>
</tr>
<tr>
<td>Contact dermatitis</td>
<td>Irritant contact dermatitis is nonimmune mediated and appears in hours</td>
<td>Shape usually matches an external exposure and is geometric rather than following anatomy, suggesting an “outside job”</td>
</tr>
<tr>
<td></td>
<td>Allergic contact dermatitis is idiosyncratic and may take days to appear</td>
<td>Very pruritic if allergic</td>
</tr>
<tr>
<td></td>
<td>Exposure history may include metals, topical antibiotics, topical agents with fragrance or preservatives, rubber products, latex, leather</td>
<td>Marked epidermal change with vesiculation often</td>
</tr>
<tr>
<td>Deep vein thrombosis (DVT)</td>
<td>History of injury</td>
<td>Acute unilateral edema and tenderness</td>
</tr>
<tr>
<td></td>
<td>History of immobility</td>
<td>Erythema is usually absent or localized only to the proximal medial thigh</td>
</tr>
<tr>
<td></td>
<td>History of prior DVT is a risk factor</td>
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</table>

Cellulitis and Erysipelas

Already covered in the portion of this article on facial rashes, cellulitis and erysipelas in the lower extremities has some characteristic differences in both history and physical examination. Predisposing factors for both types of lower extremity infections include obesity, older age, edema, venous insufficiency, and greater saphenous vein stripping/harvesting, while lymphedema specifically is a significant risk factor for erysipelas.66,67
Most cellulitis and erysipelas occurs from common skin-colonizing bacteria, \(\beta\)-hemolytic streptococci and *Staphylococcus aureus*, which enter through breaks in the integument from abrasions, arthropod bites, or maceration between the toes, the latter being commonly from tinea pedis.\(^{67,68}\) As a result, a history of such predisposing events or conditions can be helpful in suspecting the diagnosis. Incidence rates nearly double with each 10-year increment of age, with a prevalence of about 200 per 100,000 person-years, although estimates vary widely.\(^{69}\)

Patients typically present complaining of a painful expanding area of redness on the affected limb, usually over the course of a few days. Occasionally, systemic symptoms may precede the rash, possibly deriving from the significant inflammatory response to invasive bacterial soft tissue infection.\(^{66}\) Patients with cellulitis present with a localized and sometimes discontinuous area of erythema, edema, warmth, and tenderness, similar to the classic hallmarks of inflammation originally described by Celsus as *rubor*, *tumor*, *calor*, and *dolor* 2000 years ago (Fig. 19).\(^{70}\) Lower extremity cellulitis is nearly always unilateral and 95% occurs below the knee.\(^{69}\) The erythema usually blanches early in the process, but owing to extravasation of erythrocytes from inflammation and the increased intravascular hydrostatic pressures of the lower extremities, non-blanching erythema will often develop; this is often centered around the site of initiation but may spread proximally along lymphatics as red streaks. Bullae may occur as a result of either production of bacterial toxin or acute edema. Suppuration in the form of pustules or frank abscess may also occur, the latter of which should raise suspicion for *Staphylococcus aureus* and MRSA.

Because of its more superficial location and involvement of lymphatics, erysipelas is typically a bright red erythema that is sharply marginated and has a more defined superficial peau d’orange edema as described previously. Fever and adenopathy of the draining lymph nodes may also be present, although these are probably less common in the outpatient setting. Laboratory studies will show leukocytosis in most cases.\(^{66}\)

Treatment of cellulitis and erysipelas is usually empiric as most cases are not culturable, most instances being caused by \(\beta\)-hemolytic streptococci, with only about 10% caused by *S aureus*. Whereas MRSA is a common cause of furuncles, it is relatively uncommon in nonsuppurative cellulitis. As a result, empiric treatment need only cover \(\beta\)-hemolytic streptococci and MSSA if suppuration is not present, as illustrated by a study of 179 patients with nonculturable cellulitis, which showed that 96% improved with \(\beta\)-lactam antibiotics despite a high prevalence of MRSA at the institution.\(^{71}\) Addition of prednisolone to antibiotics can hasten symptom improvement and shorten hospital stays in patients with cellulitis.\(^{72}\) Antibiotic treatments are generally as those described earlier for facial rashes.

### Stasis Dermatitis

Erythema and scale can occur in any area of the body that has acutely worsening edema, although it is by far most common on the lower legs as a result of venous disease. Here, either venous obstruction or loss of venous valve competence commonly results in venous congestion in the leg and resulting edema. When chronic, the veins become more visible with enlargement, such as with varicose veins (bluish veins over 3 mm diameter that protrude from the skin surface) or corona phlebectasia (purplish 1-mm diameter branching vessels around the sides of the ankle and foot, resembling a broad cascading waterfall). The higher intravascular pressure is also associated with erythrocyte extravasation and accumulation of hemosiderin, giving the legs and dorsal foot a speckled, rusty brown color.\(^{65}\)

When this chronic edema acutely worsens, the skin can become red and scaly, even with serous drainage and crust (dried serum), and can mimic cellulitis or erysipelas,
although there are several distinguishing factors that help differentiate it from infection. The history is typically one of slow development over weeks rather than hours to days. Although there may be some burning pain, pruritus is usually the predominant complaint, as it is a dermatitis rather than pathogen-mediated tissue damage. On examination, scale (white flaky skin) is prominent, unlike in infection. Erythema involves only the leg, respecting the anatomic boundaries of the ankle and knee, typically only on the lower half of the shin and calf (Fig. 20). Although infection may also occur here, it is more likely to spread into adjacent anatomic areas. Most importantly, stasis dermatitis is typically bilateral, whereas “bilateral lower extremity cellulitis” is the Sasquatch of Dermatology: believed by many to exist, but rarely actually seen.

Eczema craquelé (asteatotic eczema) is a rash similar to and often concurrent with stasis dermatitis. As its name suggests, it appears as if the skin is cracking apart, showing a pattern reminiscent of sun-baked desert clay or glazed pottery (Fig. 21).
In fact, these apparent fissures typically extend only through the stratum corneum. This condition arises from acute edema and is less dependent on presence of venous disease specifically, although a prerequisite is profound xerosis, as seen in the elderly or those abusing their skin with cleaning products in an attempt to disinfect themselves.\textsuperscript{73,74}

\textbf{Fig. 20.} Stasis dermatitis. Typical distribution from the mid shin and calf to the ankle of both legs. (\textit{From} Habif TP, Campbell JL, Chapman MS, et al. Skin disease: diagnosis and treatment. Philadelphia: Elsevier/Saunders; 2011; with permission.)

\textbf{Fig. 21.} Asteatotic eczema (eczema craquelé). Note the “dry riverbed” pattern to the superficial cracking of the epidermis. (\textit{From} Norbert Reider N, Fritsch PO. Other eczematous eruptions. In: Bologna JL, Jorizzo JL, Schaffer JV, editors. Dermatology. 3rd edition. Philadelphia: Elsevier/Saunders; 2012. p. 219–31; with permission.)
Treatment of the acute inflammation is usually achievable with a moderate-potency topical corticosteroid such as triamcinolone 0.1% ointment applied twice daily to the affected areas, although systemic treatment with a short course of prednisone can be helpful in severe cases. Both stasis dermatitis and eczema craquelé require correcting the edema and xerosis for complete resolution and long-term control. Emollients and elevation can be instituted immediately, although in an outpatient setting, elevation of a leg above the level of the heart is impractical for long periods. As a result, external compression is usually desired through compressive wraps such as an Unna boot, a short-stretch bandage, or a multilayer compression device (of which several different brands are available). These devices are typically applied in the office but can be inconvenient and costly for the patient, as they cannot get wet, are too bulky for normal shoes, and require at least weekly changes in a physician’s office. As a result, most ambulatory patients transition to a graduated compression stocking or adjustable elastic compression (eg, CircAid) as soon as they are able.

Graduated compression stockings differ from uniformly compressive thromboembolism deterrent (TED) hose used in immobile patients in that they have higher compression around the foot and ankle, which lessens toward the upper calf. Veins with competent valves use the pumping action of leg-muscle contraction during walking to return blood against gravity to the heart. In the absence of valves, graded compression stockings accomplish the same unidirectional movement by being more permissive to venous flow only in the proximal direction. These stockings still rely on muscle use when patients are upright, so patients should be encouraged to be mobile. Treatment of clinically significant edema may require higher levels of compression stockings (>30 mm Hg) than most patients can comfortably wear or even put on, although there is little consensus as to which level of compression may be needed for different conditions. Stockings of 20 to 30 mm Hg are often a compromise in efficacy and patient adherence. Alternatively, wearing 2 lower-level compression stockings on top of one another achieves a roughly additive compression (eg, wearing two 15–20 mm Hg stockings on the same leg is roughly equivalent to a single 30–40 mm Hg stocking), and may be easier for patients to use with a trade-off of increased material bulk and heat. Compression stockings are commonly available at medical supply stores, online stores, and, more recently, at athletic and yoga stores, in more attractive designs for better adherence. Effects of poor arterial perfusion can be compounded by external compression, of course, so compression is ill advised if the ankle-brachial index is less than 0.8.

Contact Dermatitis

An acute dermatitis caused by either an irritation or allergic cause is also common on the legs. Typically, the clue as to a contact dermatitis is that the shape of involvement will be geometric rather than anatomic such that it appears to be an “outside job.” Irritant contact dermatitis develops within hours after exposure to a chemical that has direct toxic effects on the skin. It is not an immunologic response and therefore is an expected reaction in any person exposed to such an environmental stimulus. Common examples include detergents or soaps left in contact with the skin, or adhesives in bandages (which may less commonly cause allergic contact dermatitis as well). Erythema, scale, and crust occur with vesiculation or bullae in more profound responses. The predominant symptom is typically one of pain or burning rather than pruritus.

Allergic contact dermatitis is an immunologic response to an allergen, and therefore is more unique to an individual in that a sensitized person will contract a rash any time they are exposed to this product, whereas a nonsensitized person would have no
reaction at all. Common causes include metals such as nickel and cobalt, topical antibiotics such as neomycin or bacitracin, fragrances, lanolin, textile dyes in socks, latex in compression stockings, potassium dichromate used in leather tanning, or numerous chemicals used in the production of rubber or neoprene used in footwear. As with irritant contact dermatitis, erythema, scale and crust, and vesiculation and bullae can occur, but the predominant symptom is primarily one of pruritus. Onset is typically delayed by 1 or 2 days, and relies on a type IV hypersensitivity response rather than the immediate toxic effects seen in irritant dermatitis.

The acute onset, erythema, and pain of either reaction may mimic cellulitis, but the pattern of involvement will stay localized only to the area of contact, and the marked epidermal change (scale and serous exudate and crust) and pruritus help to suggest a dermatitic process. The shape of involvement will also give clues as to the cause (Fig. 22).

Treatment is, of course, complete removal of the causative agent, followed by topical corticosteroids. A moderate-potency steroid such as triamcinolone is often sufficient, but a week-long course of an ultrapotent steroid such as clobetasol and even systemic prednisone may be needed for severe cases.

**Deep Vein Thrombosis**

Though not necessarily a problem of the skin, acute DVT is often in the differential for an edematous painful and red leg. Whereas edema and pain are typical in DVT, erythema is not in fact a typical symptom. Erythema was only one of the minor criteria for DVT in the original Wells score, yet did not hold up after regression analyses and is absent from the currently used Revised Wells score. Venous congestion may leave a purplish hue to the leg, but erythema only occurs when overlying a DVT of the proximal medial thigh where the femoral vein is close enough to the skin that the inflammation is visible externally. However, chronic edema of DVT may result in secondary stasis dermatitis or eczema craquelé.

**TREATMENT**

Although systemic agents are heavily used in dermatology, often patients and providers alike prefer topical management. Whether using corticosteroids, antimicrobials,
or comedolytics, topical vehicles allow a high concentration of a medication where it is needed while minimizing systemic side effects. The normal epidermis functions as a barrier to these medications, however, and the vehicle can affect not only medication efficacy but also patient adherence. Fig. 23 shows some of the characteristics of the most common vehicles for which ease of application is in general inversely related to efficacy. All medications containing water require the use of preservatives, which can cause allergic contact dermatitis, so nonaqueous ointments are preferable when this is a concern. Greasier medications such as ointments also penetrate the hydrophobic epidermis more readily and thus are more potent.82

All topical agents are most easily applied to moist skin just after showering, although patients may not like the slimy feeling that results on the skin. Advising patients to only apply the minimum necessary to feel something has “just barely” gotten on all the affected area is important, as overapplication is not generally more efficacious but results in excess medicine on clothes and bedsheets, limiting adherence quickly. The concept of the fingertip unit (FTU) of topical medication (a single line of cream or ointment from a standard tube that extends along the distal phalanx of the patient’s index finger) can be useful in advising how much to apply. A single FTU is enough for one hand, 2 FTU enough for a foot, 3 FTU for an arm, and 6 FTU for a leg.83,84

Corticosteroids are among the most commonly prescribed class of topical medications. In the United States these are grouped by their varying efficacy from class 1 (superpotent) to class 7 (least potent). Because the vehicle can affect efficacy by increasing absorption, any given steroid may belong to more than one class depending on its vehicle; for example, fluticasone 0.005% ointment is class 3, whereas the more concentrated 0.05% cream and lotion are class 5. Similarly, flurandrenolide 0.05% lotion is a class 5, whereas tape impregnated with flurandrenolide is a class 1 owing to its occlusive effects. Concern about side effects from overuse by both clinicians and patients results in ineffective treatment being more of a problem than overuse. Side effects of topical corticosteroids include striae distensae, bruising, telangiectasias, skin fragility, pigmentary changes, and suppression of the hypothalamic-pituitary-adrenal axis (though the latter is not clinically significant without extensive areas of application over long periods).82 A conservative rule of thumb is that a steroid can be used for as many weeks as its class without concern for side effects; for example, a class 2 could be used for 2 weeks while a class 6 could be used for 6 weeks.

Fig. 23. Characteristics of vehicles.
SUMMARY

When assessing a patient with a new dermatologic condition, developing a differential diagnosis is essential to ensure the condition is not elusive as a workup and potential therapy are considered. Subsequent narrowing of that differential based on history and physical examination can allow a more targeted approach to diagnostic testing and triage, and hasten an effective treatment and resolution.

The authors hope to have provided useful historical and clinical clues to aid in the rapid differentiation of the more common diagnoses for alopecias and rashes of the face, intertriginous areas, and legs.

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