Common Skin Condition in Black Skin

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The clinical features and management of many common cutaneous conditions, including acne, atopic dermatitis, and rosacea, are consistent across skin types, but some findings associated with these entities, most importantly skin inflammation, can present uniquely in skin of color and lead to misdiagnosis and inappropriate treatment. This chapter provides an overview of commonly encountered skin diseases, highlighting the variations in presentation that can be seen in skin of color. The aim of this chapter is to aid the practitioner in recognizing common skin diseases and selecting appropriate treatments for a wide variety of skin types.

2.1 Dermatitis

Dermatitis is a general term referring to superficial inflammation of the skin, typically associated with erythema and scaling. Other characteristics of dermatitis that are not always present can include crusting, blistering, edema, and pruritus. There are multiple subtypes of dermatitis, which are defined by their unique clinical presentations and underlying causes.

2.1.1 Atopic Dermatitis

Atopic dermatitis (AD) is one of the most common chronic inflammatory skin diseases. It typically presents during infancy or early childhood, but onset can occur at any age. The underlying cause of AD is complex and only partially understood. It appears to be driven by predisposing genetic factors, environmental exposures, and inappropriate breakdown of the skin's normal protective barrier. In combination, these factors result in activation of multiple inflammatory signaling pathways, infiltration of inflammatory cells into the skin, and development of the scaly, pruritic, inflamed patches, plaques, and nodules that are characteristic of AD. AD is often seen in association with asthma and allergic rhinitis.

2.1.1.1 Presentation in Black Skin

The strongest risk factor for development of AD is the presence of the *FLG* gene mutation, a mutation that results in a disrupted epidermal skin barrier. African Americans are six times less likely than European Americans to carry this mutation, but they nevertheless have a higher prevalence of AD.

The erythema of AD, which presents as bright red or pink in lighter skin types, often appears as violaceous, brown, or ashen grey in skin of color. Though more subtle in brown or black skin, red or pink erythema can usually be seen at the border of active AD lesions, and in combination with the presence of scale and induration, can help to distinguish active AD lesions from resolving AD lesions with hyperpigmentation. Clinical signs that appear to be more common in skin of color include severe pruritus, hyperlinearity of the palms, lichenification, extensive xerosis, and infra-orbital Dennie-Morgan lines (Figs. 2.1 and 2.2). The presentation of AD also may vary, as less flexural surface involvement and more extensor distribution is seen in African Americans than in European Americans. Papular atopic dermatitis, a variant of AD characterized by small, scaly papules on the trunk and extremities, often centered around hair follicles, is also more common in skin of color (Figs. 2.3–2.37).

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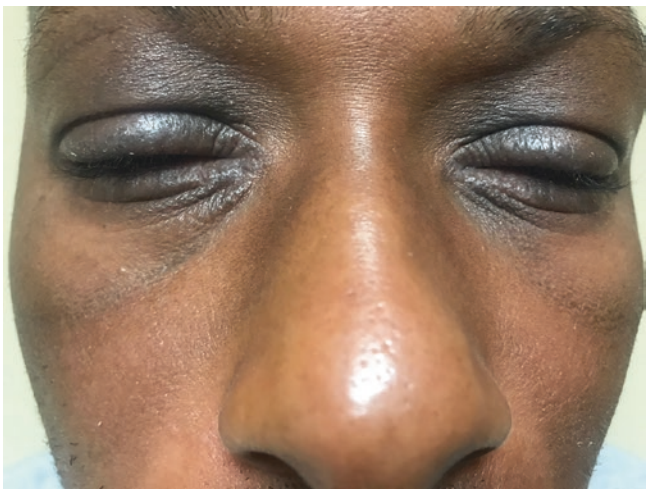
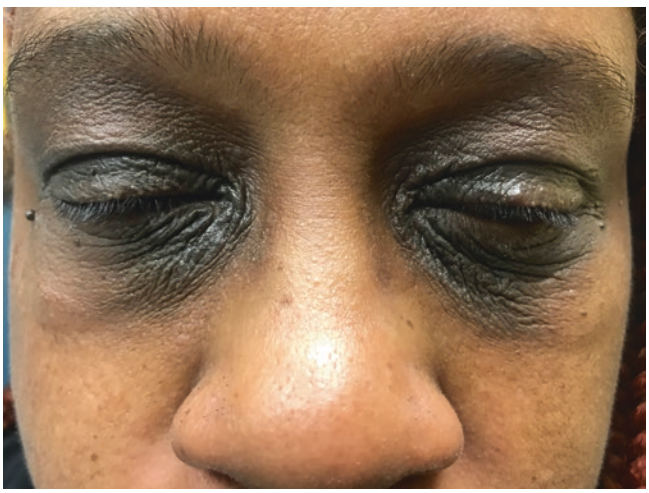


Fig. 2.1 Dennie-Morgan lines



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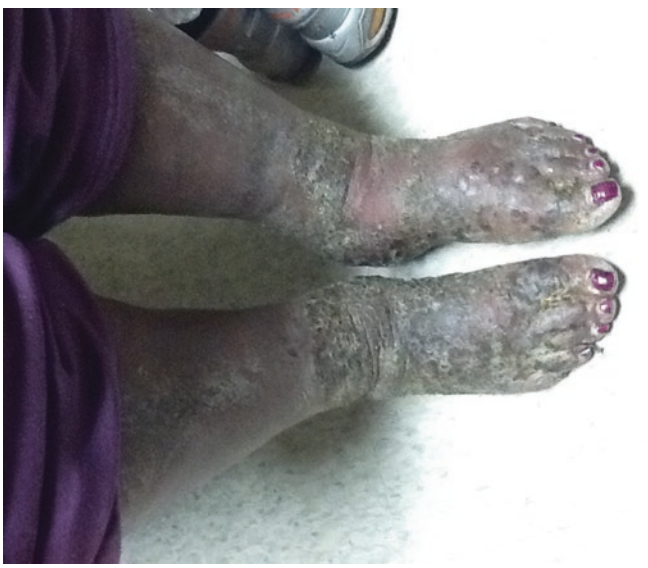
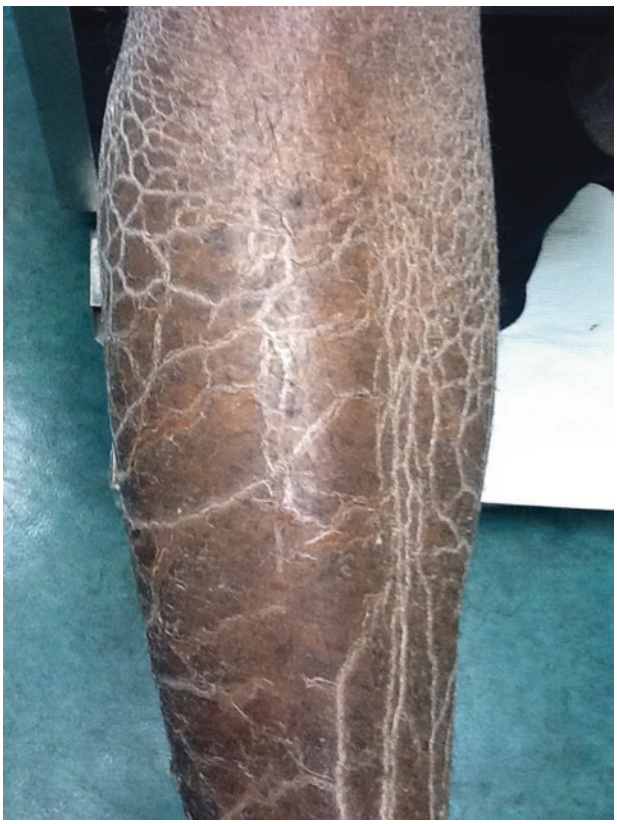


Fig. 2.3 Atopic dermatitis



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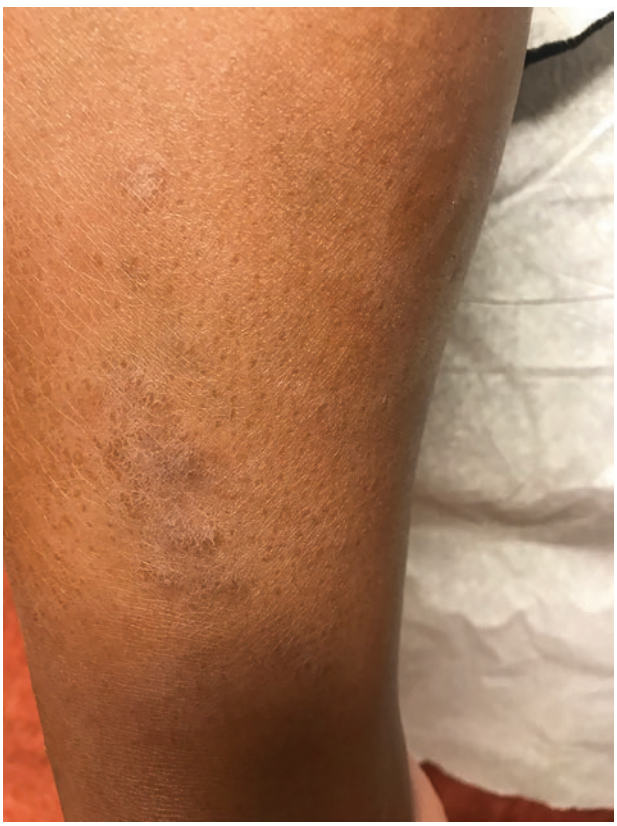

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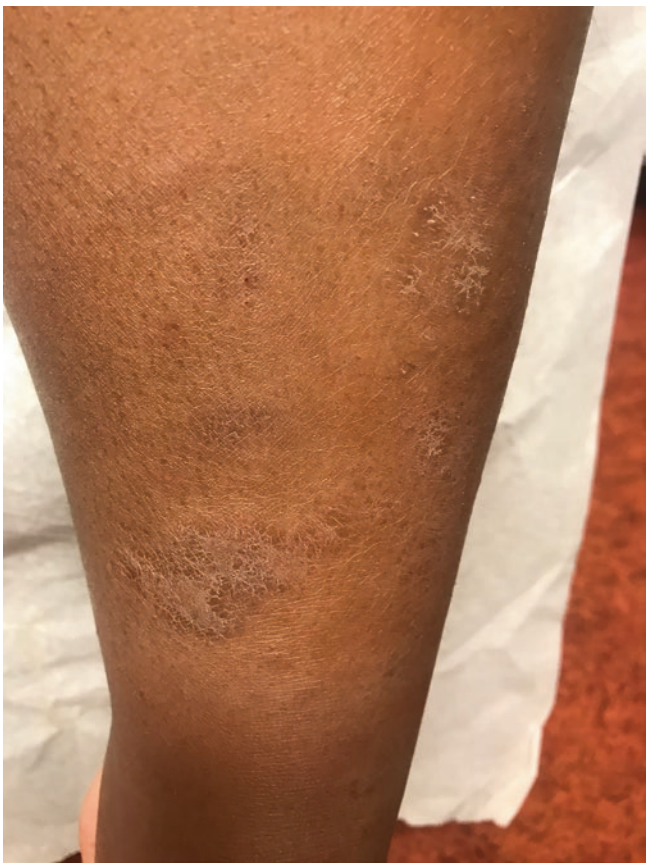

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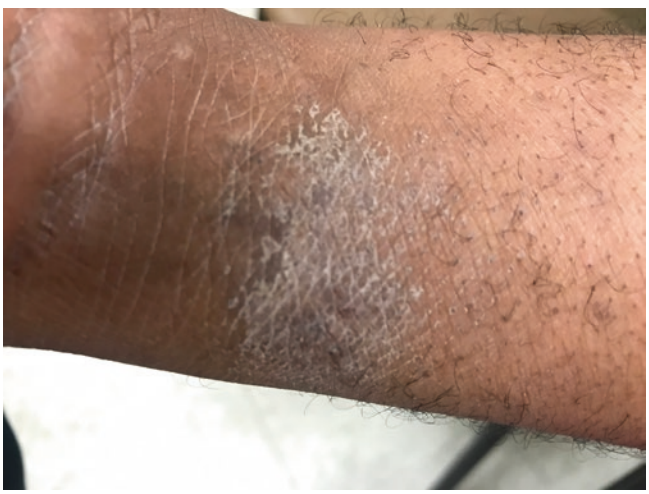

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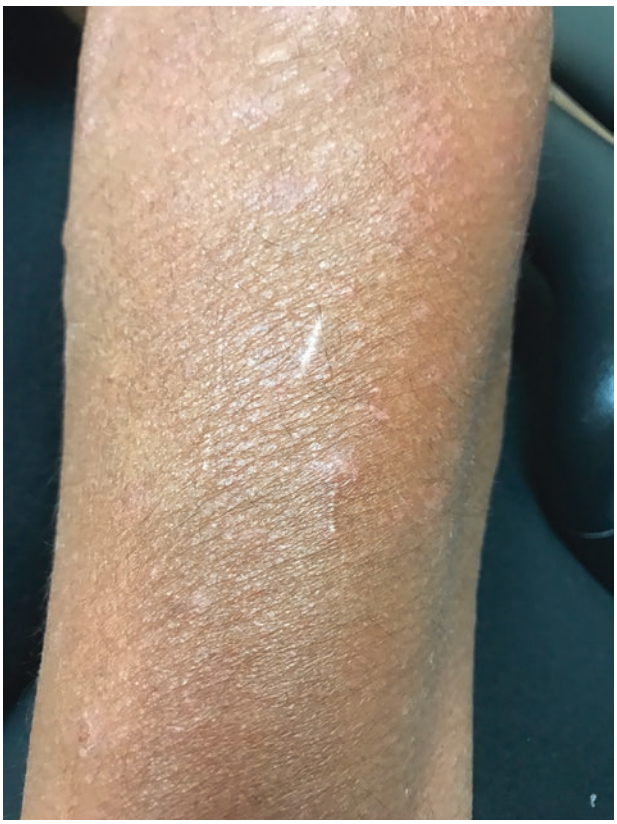

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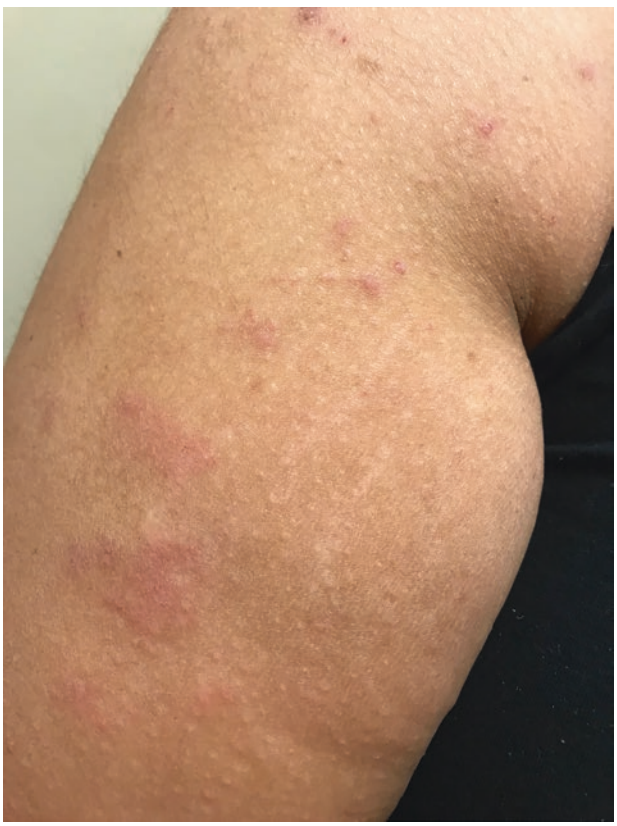

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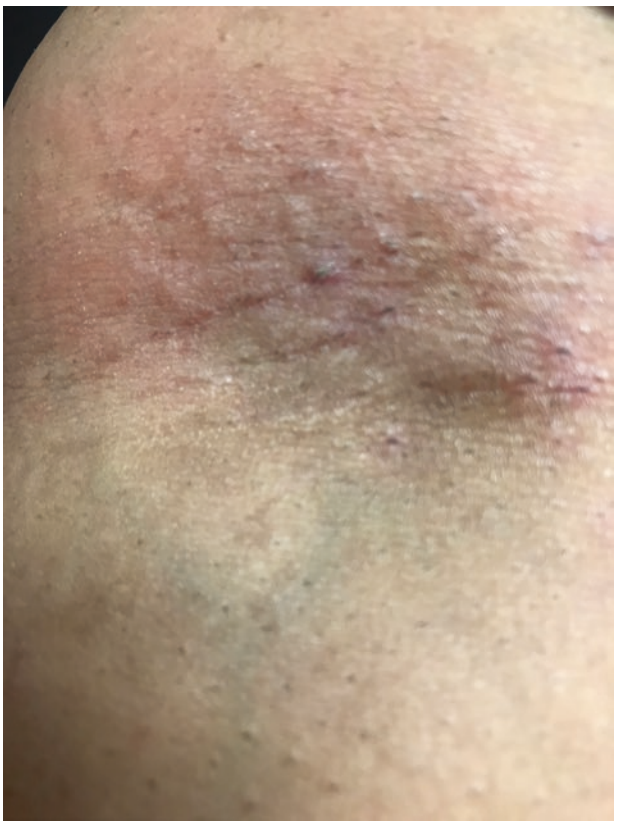

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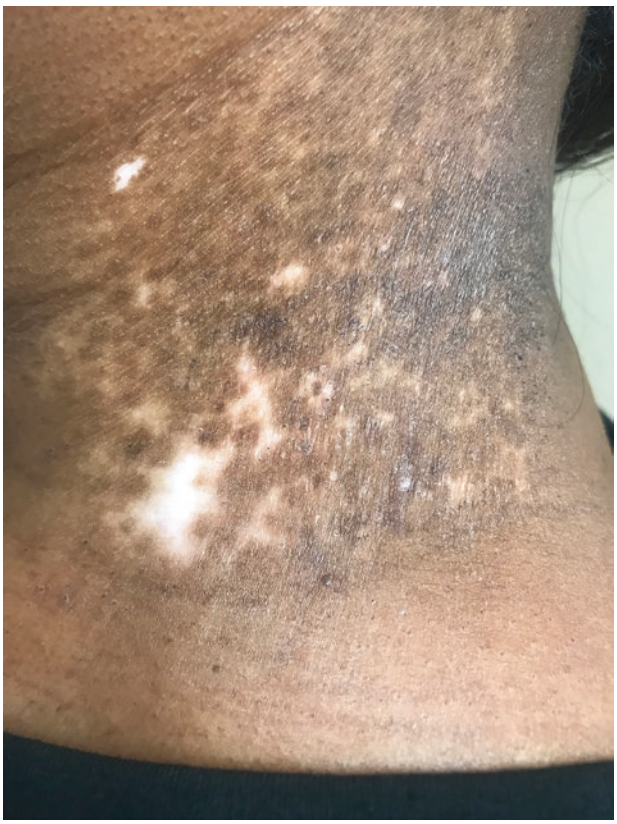

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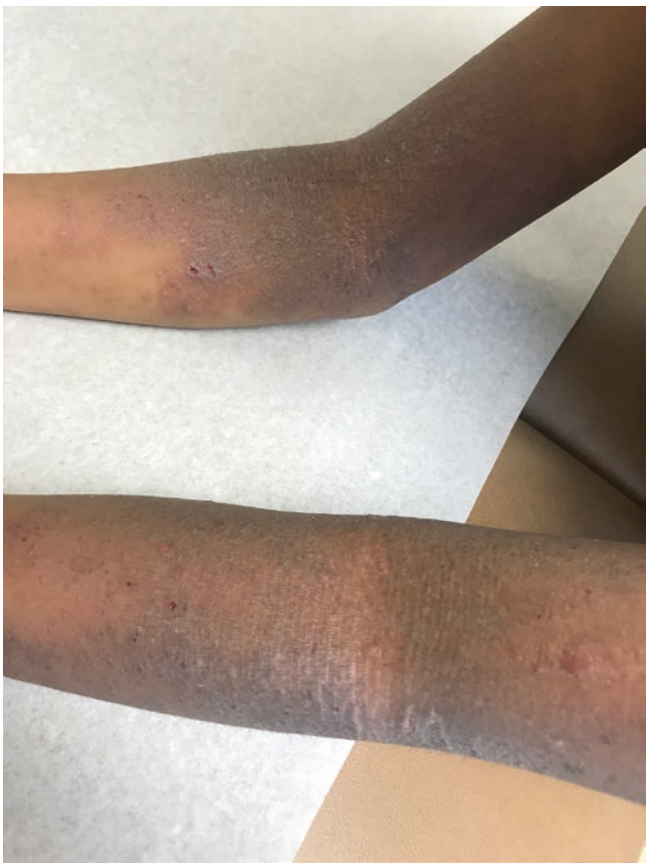

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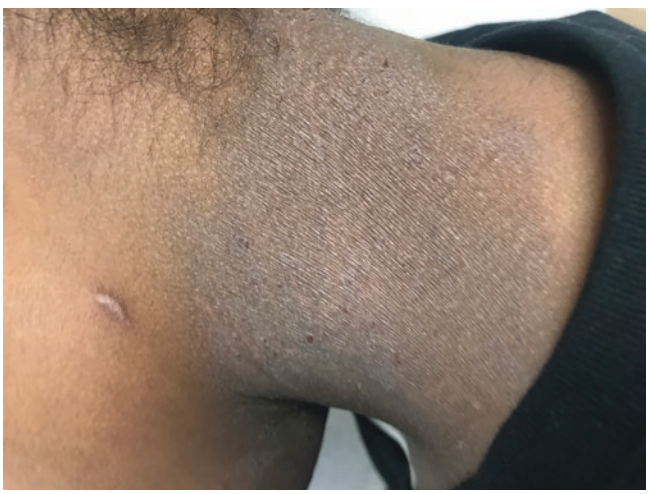

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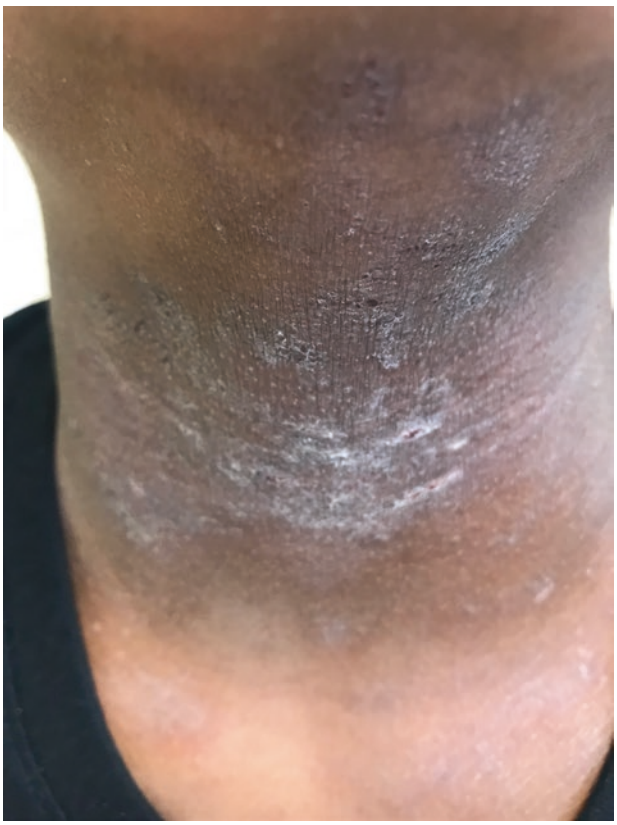

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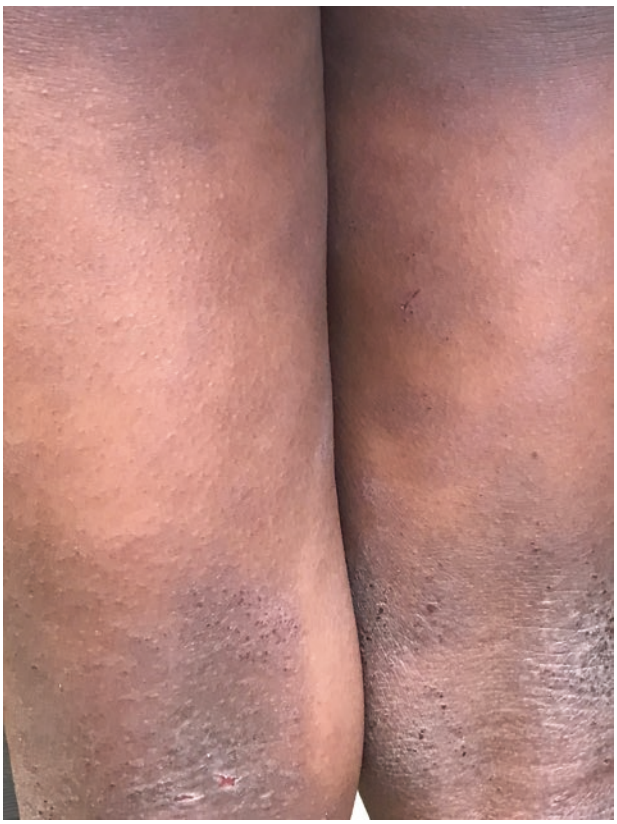

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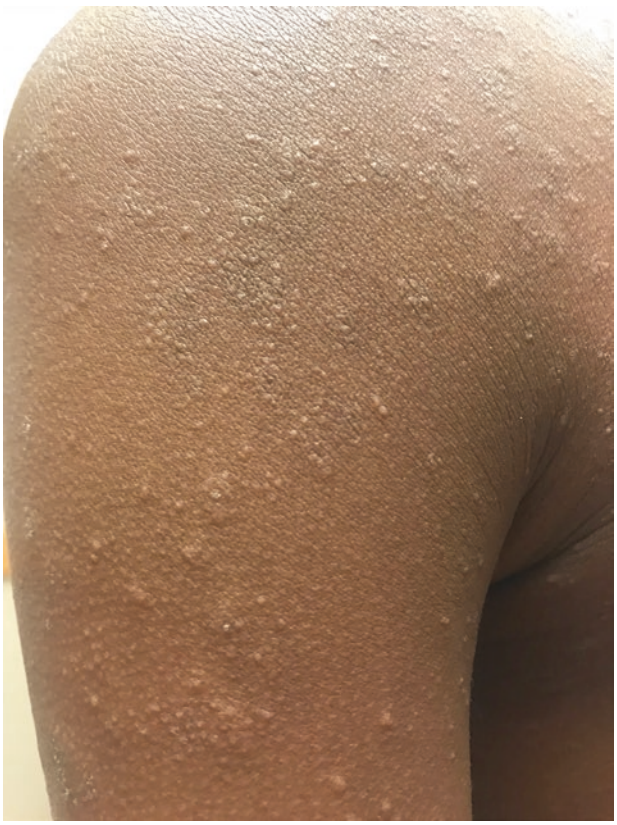

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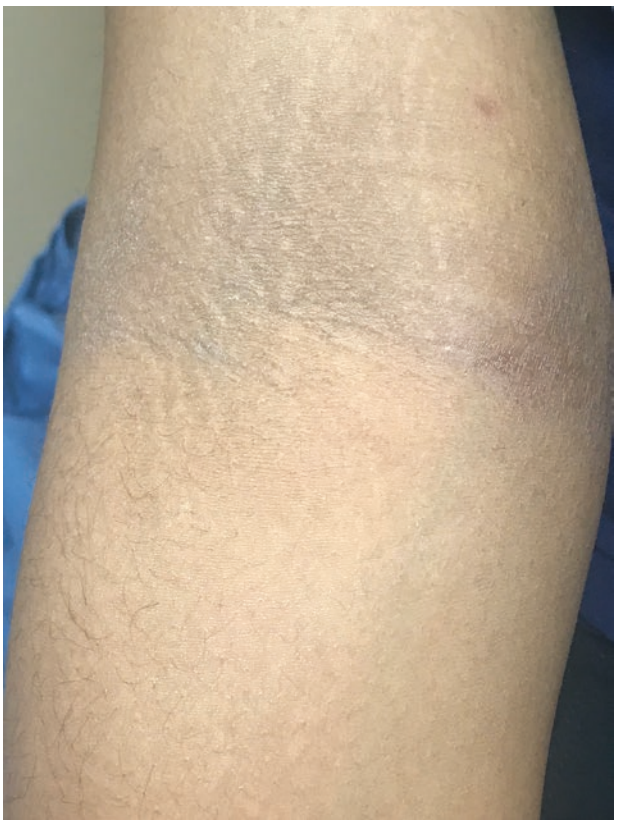

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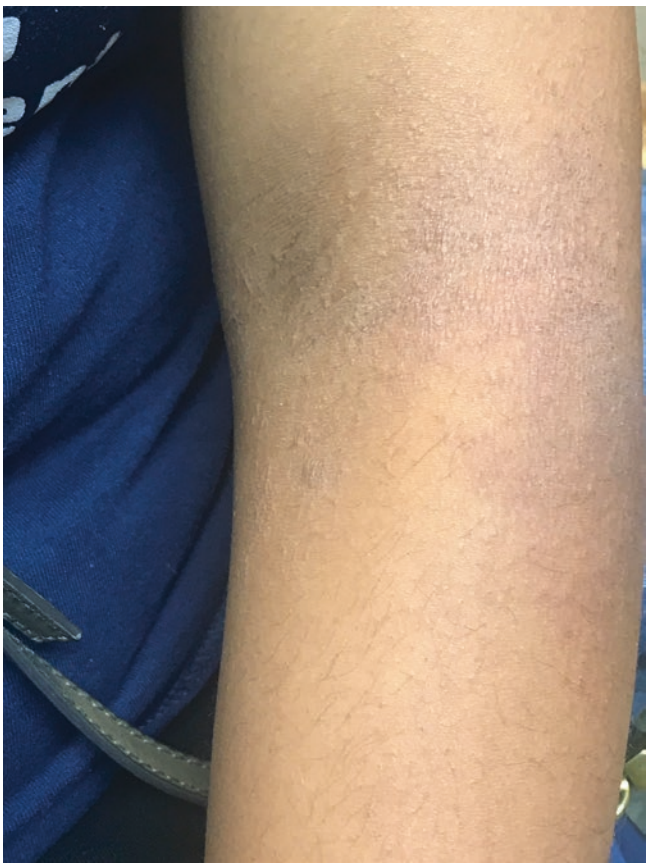

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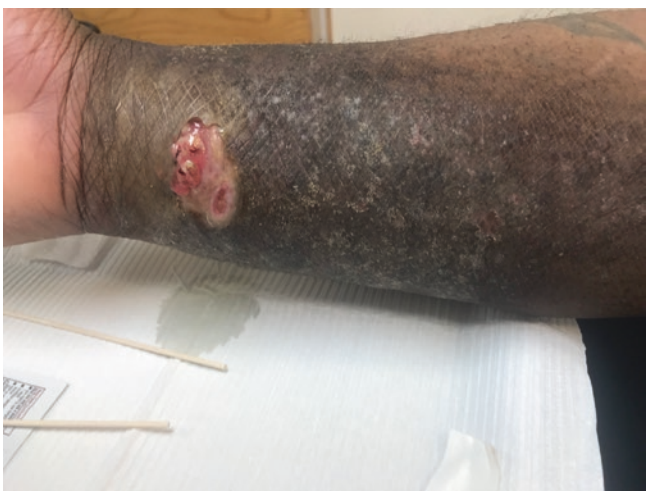

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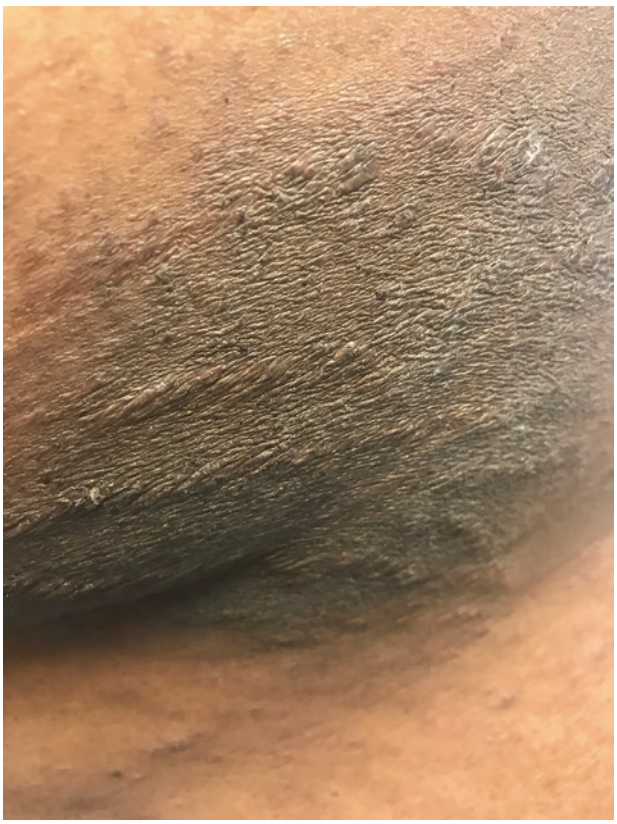

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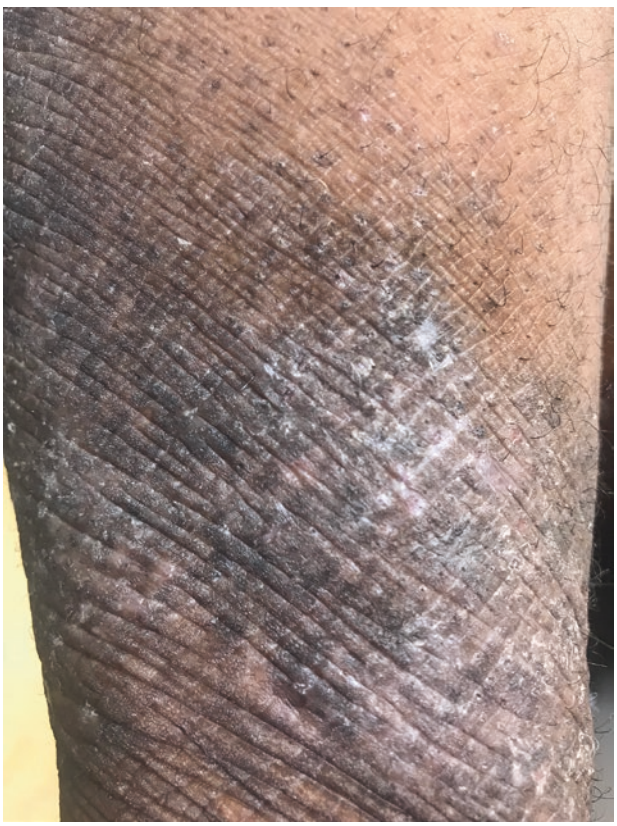

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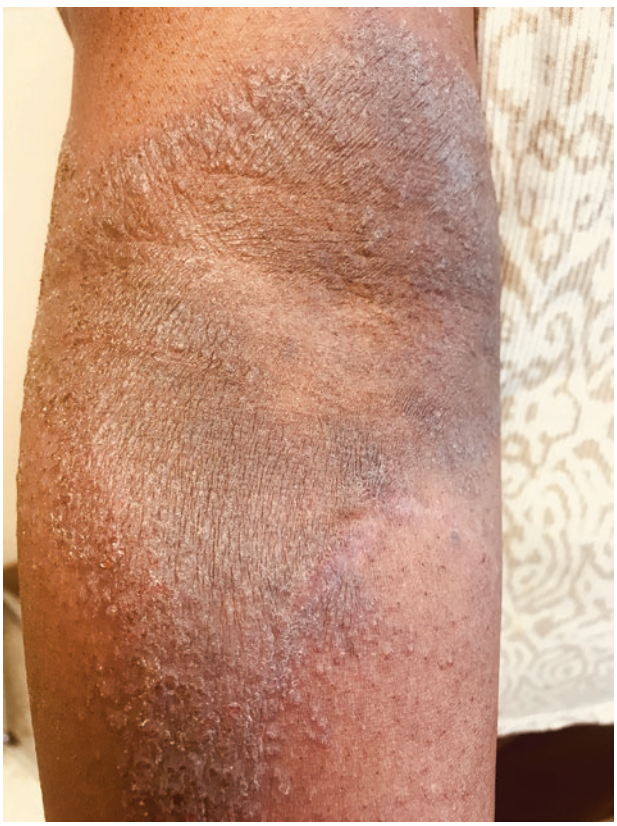
Fig. 2.22 Atopic dermatitis



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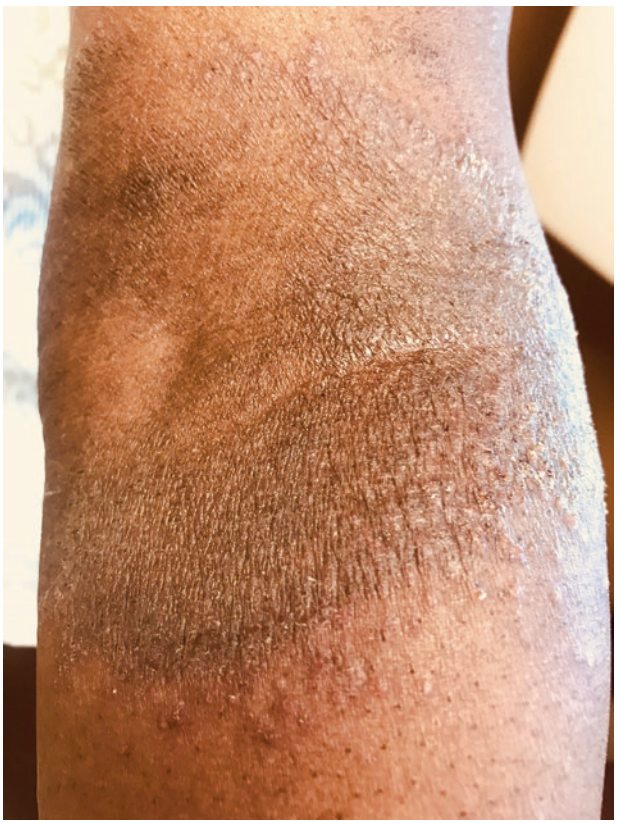

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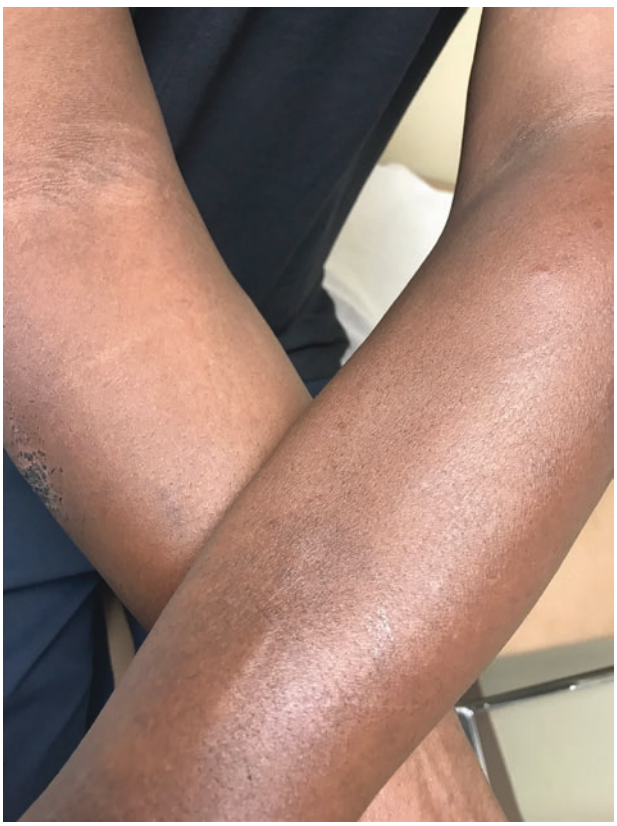

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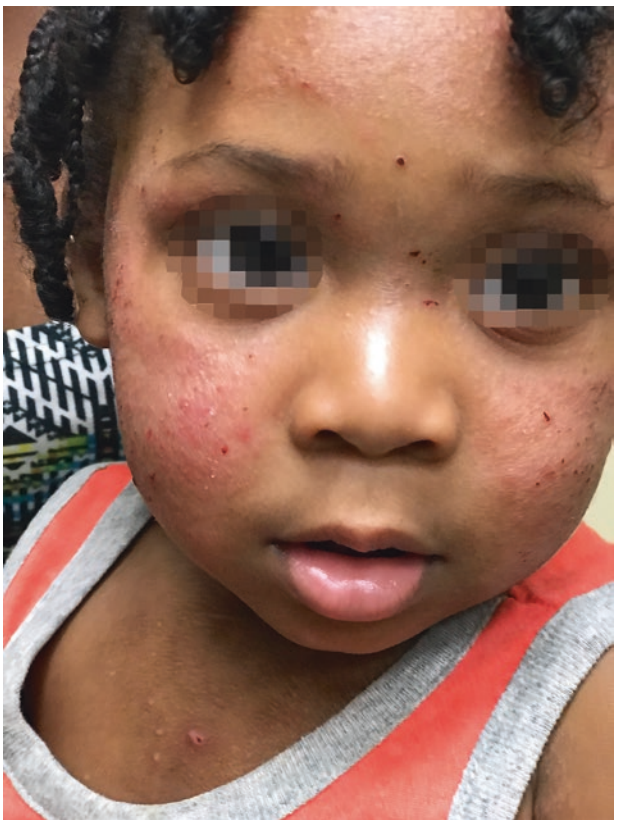

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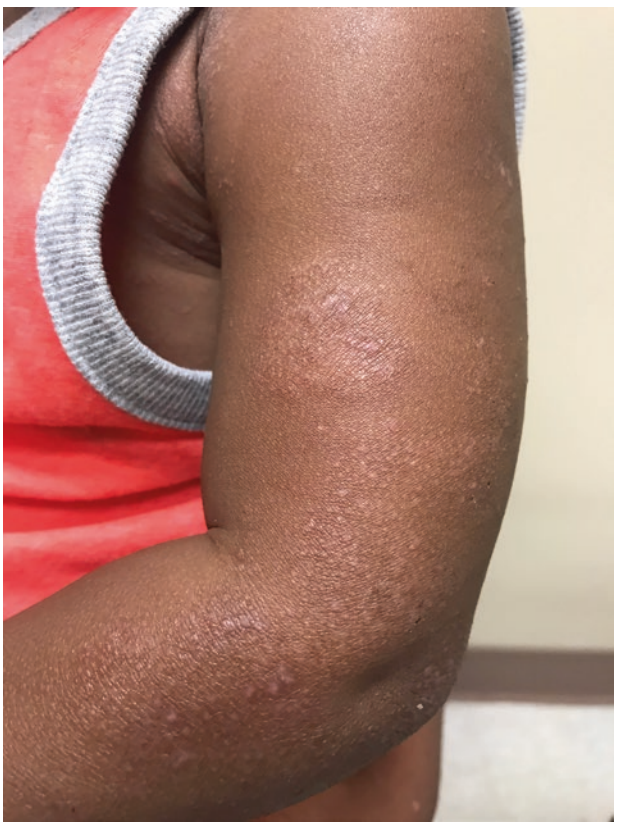

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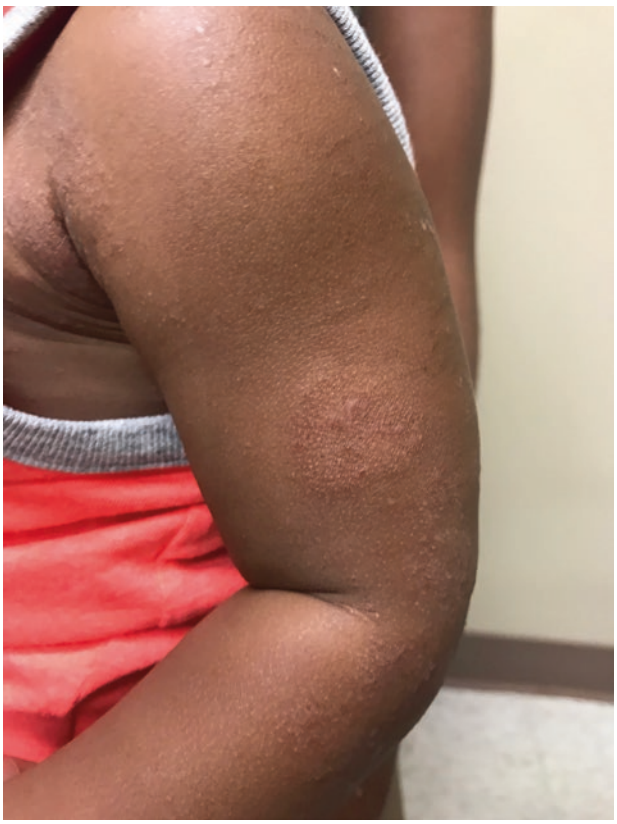

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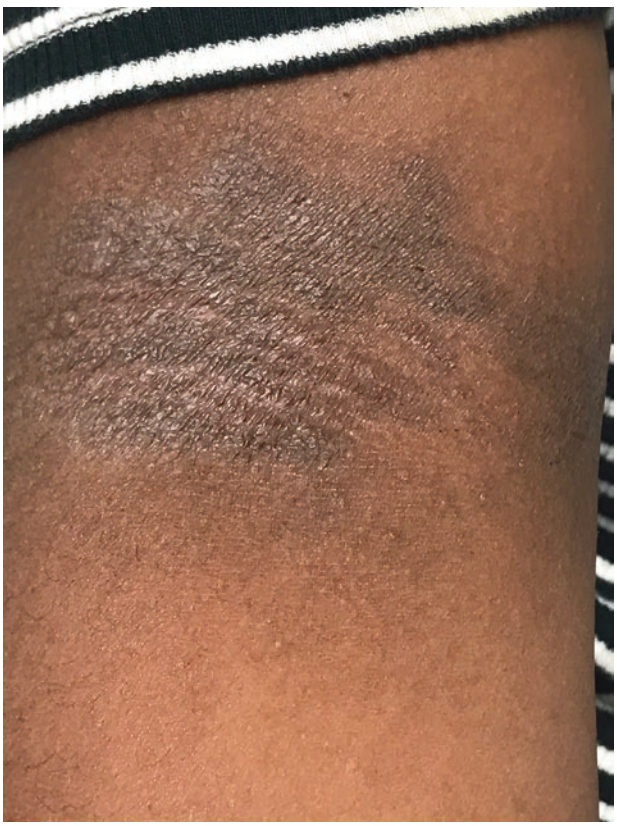

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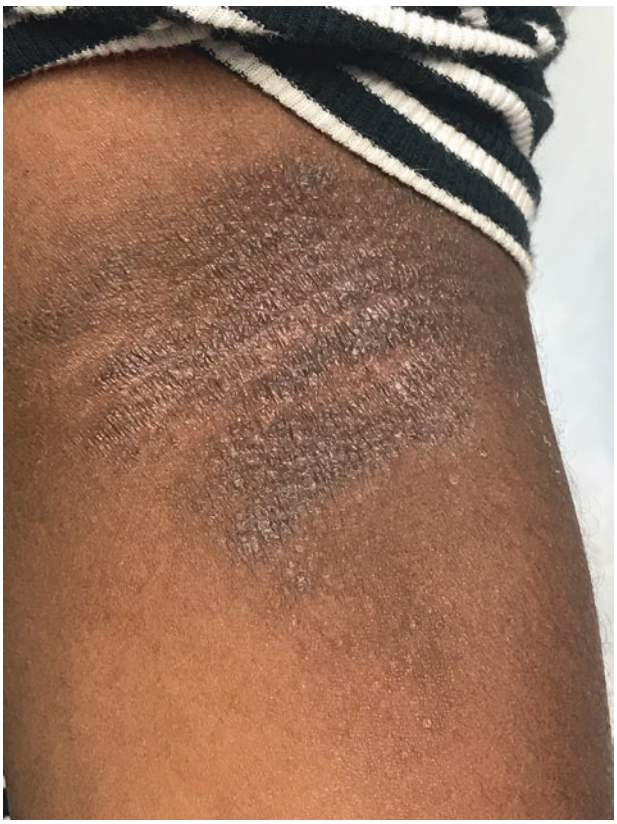


Fig. 2.30 Atopic dermatitis

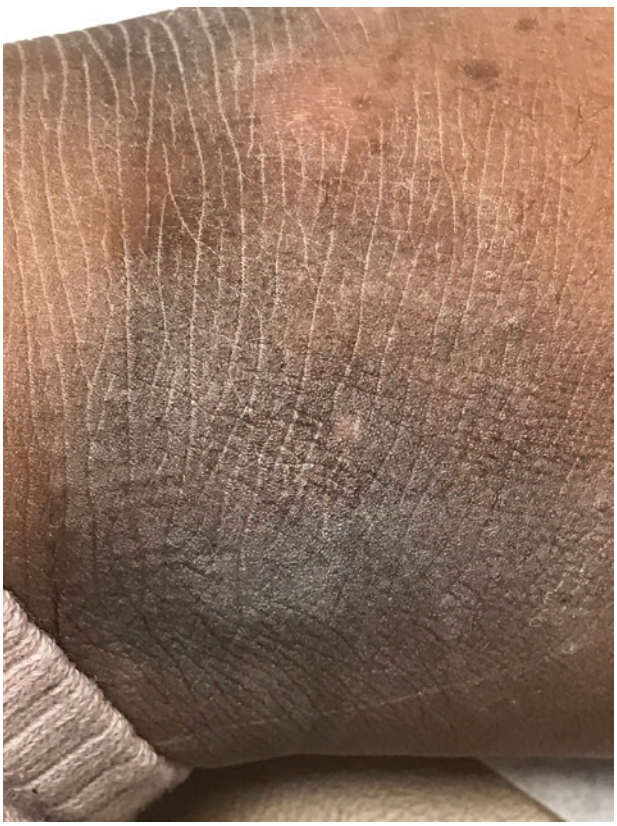
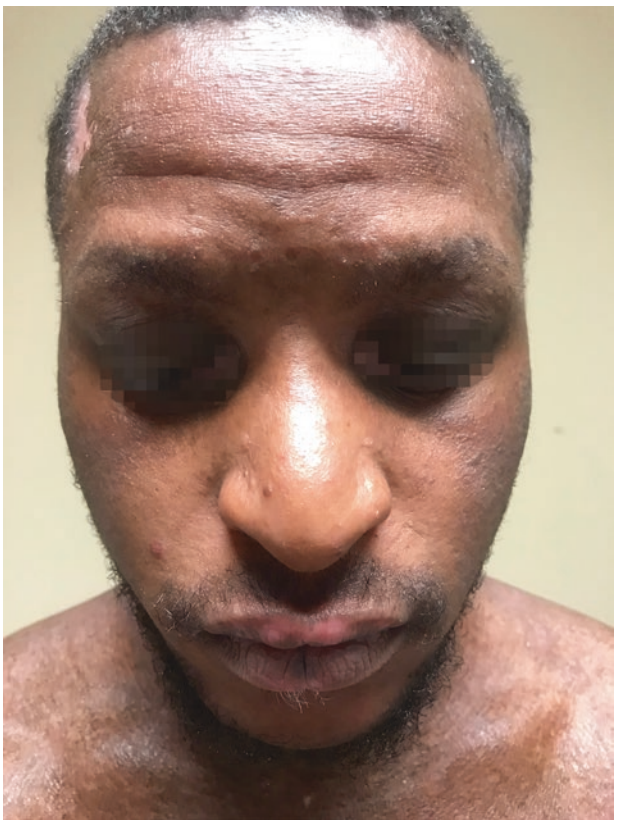


Fig. 2.31 Atopic dermatitis

Fig. 2.32 Atopic dermatitis

Fig. 2.33 Atopic dermatitis



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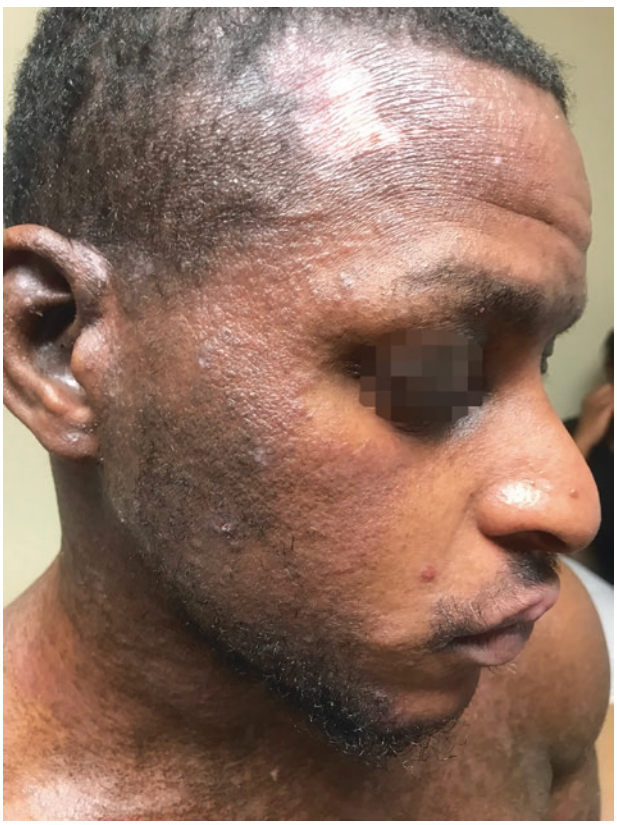

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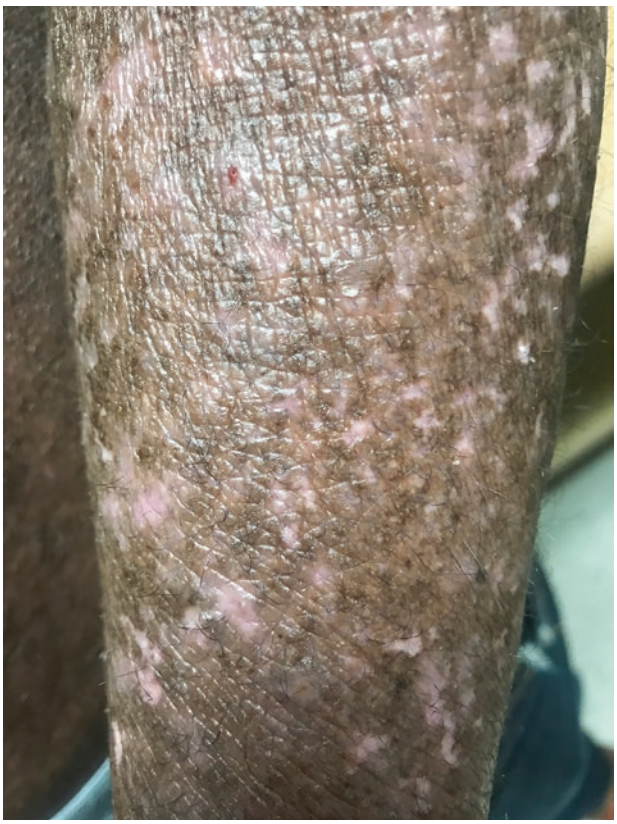

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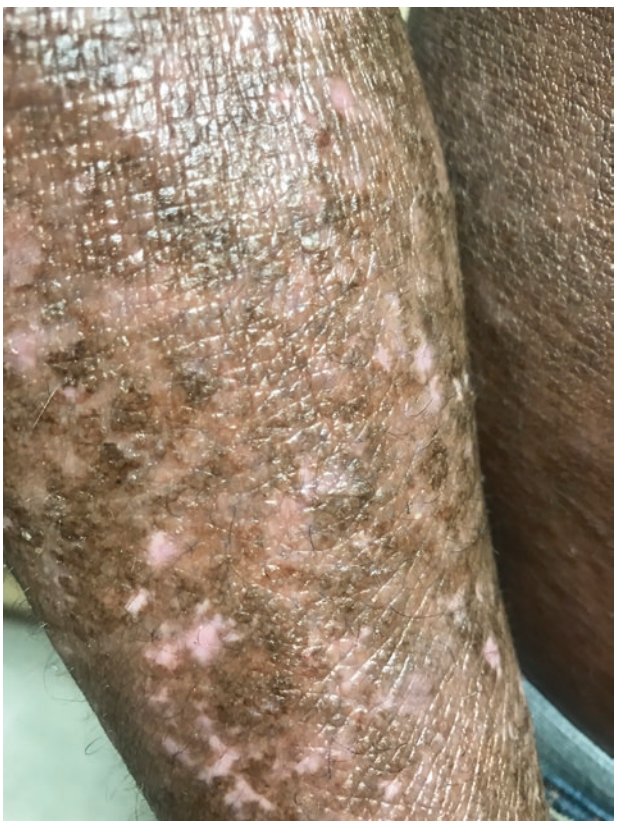


Fig. 2.37 Atopic dermatitis

2.1.1.2 Management

First-line therapy includes aggressive use of emollients, avoidance of environmental triggers, use of topical steroids or topical calcineurin inhibitors, and for mild disease, the topical PDE4 inhibitor crisaborole. Phototherapy, systemic immunosuppressants, and targeted biologic medications can be used for refractory, widespread, or severe disease.

2.1.2 Contact Dermatitis

Exposure of the skin to environmental agents can result in superficial skin inflammation, scaling, and blistering that is known as *contact dermatitis*. Contact dermatitis is subdivided into two types: Irritant contact dermatitis (ICD) develops from the direct toxic effect of a chemical on the skin and is non-immunologic in nature. Allergic contact dermatitis (ACD) occurs when the skin is exposed to an allergen that triggers a cell-mediated, delayed (type IV) hypersensitivity reaction in the skin. ICD accounts for about 80% of all cases of contact dermatitis, compared with 20% for ACD. ICD and ACD are not mutually exclusive, however, and may occur simultaneously in some patients.

2.1.2.1 Presentation in Black Skin

The characteristic clinical features of contact dermatitis in all skin types are pruritic, erythematous, scaly papules, patches, plaques, and occasionally vesicles. These develop in geographic patterns that correspond to the area exposed to the causative agent. In darker skin types, erythema may be less red, and more purple or brown in color. Because inflammation from contact dermatitis is often intense, post-inflammatory hyper- and hypo-pigmentation can be prominent and persistent, especially in skin of color. Though the susceptibility of skin of color to contact dermatitis in comparison to Caucasian skin has been studied extensively, the data are largely conflicting and do not show any clear differences between skin types. However, differing cultural practices may result in different patterns of exposure to environmental allergens and irritants. Inquiring about culturally specific exposures, e.g. use of hair relaxers in patients of African descent or black henna tattoos in Hindu or Middle Eastern patients, can help to confirm a diagnosis of contact dermatitis (Figs. 2.38–2.42).

2.1.2.2 Management

Identification and avoidance of the causative agent is the most important step in management. Elimination trials for suspected allergens and irritants and patch testing for potential allergic exposures can be useful in identification of the offending agent. First-line treatment consists of aggressive emollient use to restore the damaged skin barrier, application of topical steroids, and if necessary, antihistamines to control pruritus and histamine-induced inflammation in the skin. Short courses (up to 2–3 weeks) of systemic corticosteroids

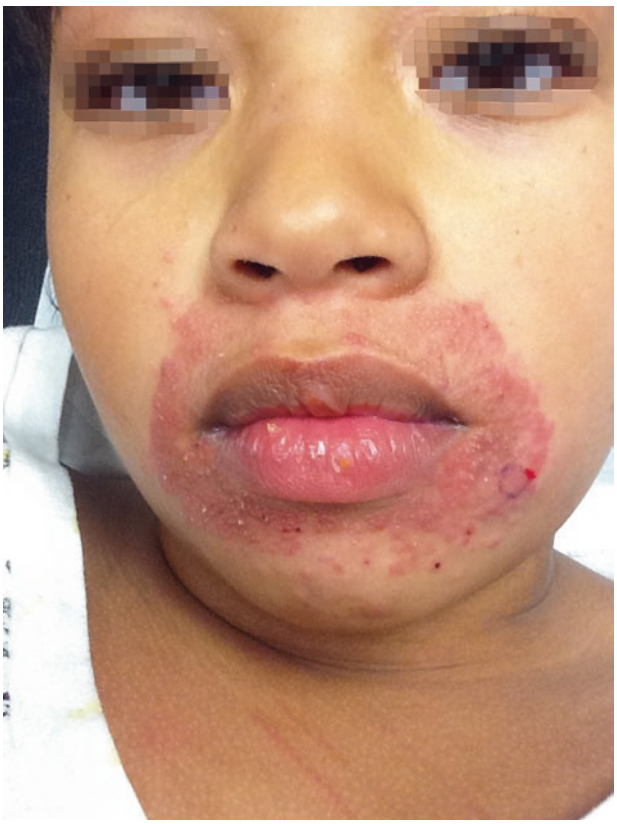


Fig. 2.38 Contact dermatitis secondary to oranges

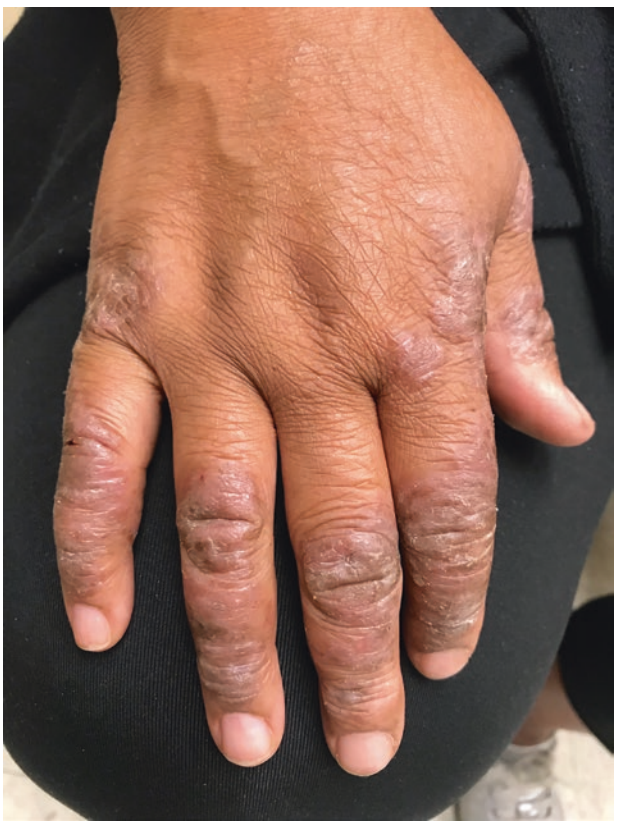


Fig. 2.39 Contact dermatitis secondary to gloves

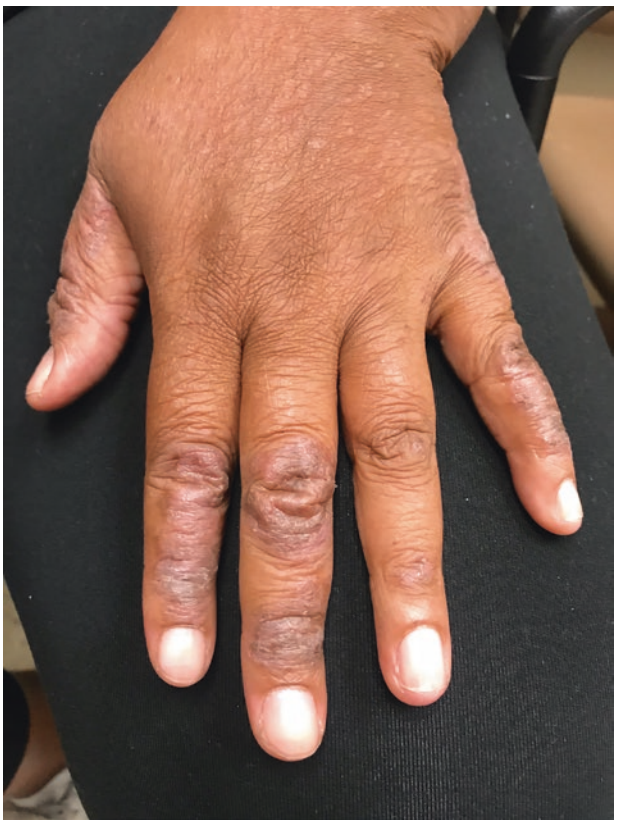


Fig. 2.40 Contact dermatitis secondary to gloves

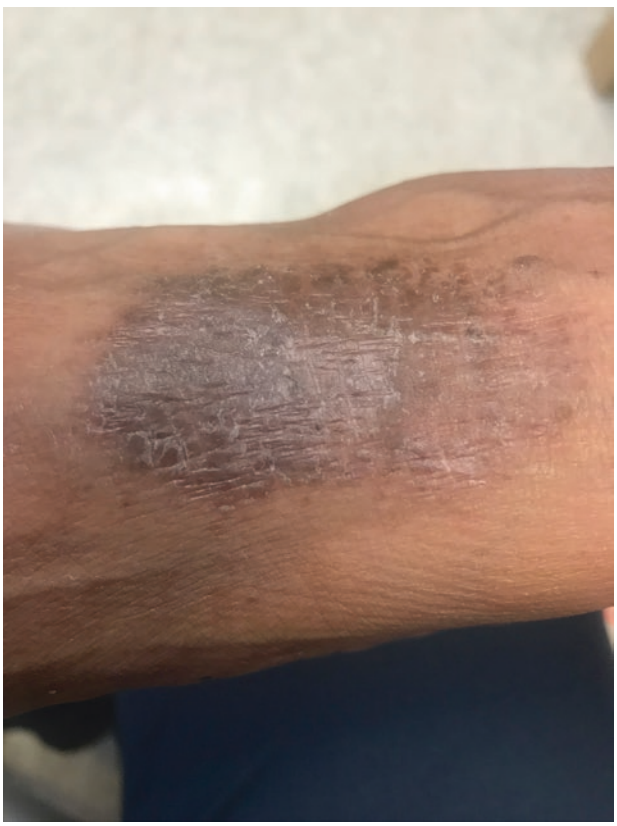


Fig. 2.41 Contact dermatitis secondary to nickel

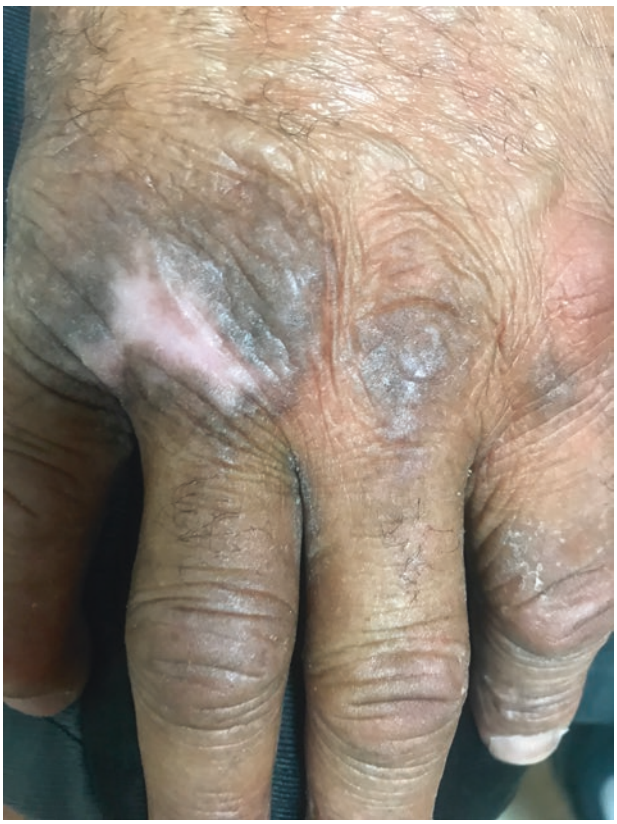


Fig. 2.42 Chronic contact dermatitis secondary to perfumed soaps

can be used for widespread or severe acute disease and phototherapy can be considered for persistent, extensive, or chronic disease. Hyper-and hypo-pigmentation, if present, will resolve over time once the underlying dermatitis has been treated. Hydroquinone and other topical skin lightening agents can be used for refractory hyper-pigmentation.

2.1.3 Nummular Dermatitis

Nummular dermatitis is characterized by well-demarcated, coin-shaped, pruritic, scaly patches and plaques with no central clearing. Nummular dermatitis lesions appear primarily on the extremities and are often chronic. Men are more likely to be affected than women, and though it is most common in adults, it can present in childhood. The etiology of nummular dermatitis is unknown.

2.1.3.1 Presentation in Black Skin

The patches and plaques of nummular dermatitis are more likely to appear brown or violaceous in skin of color, with less prominent, bright red erythema than in lighter skin types. Active nummular dermatitis lesions can be differentiated from post-inflammatory hyperpigmentation because of the presence of induration, red/pink erythema (often most prominent at lesion borders), and scale (Figs. 2.43–2.54).

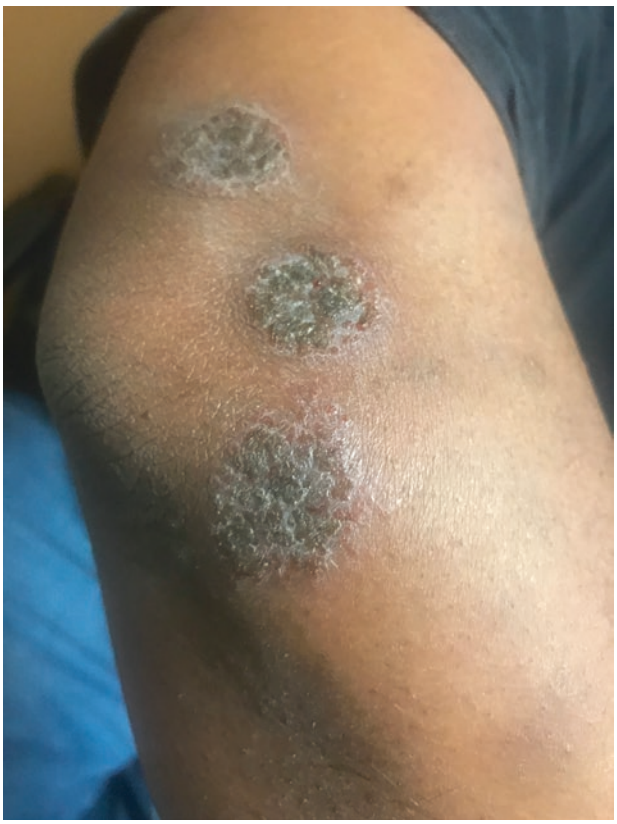


Fig. 2.43 Nummular dermatitis

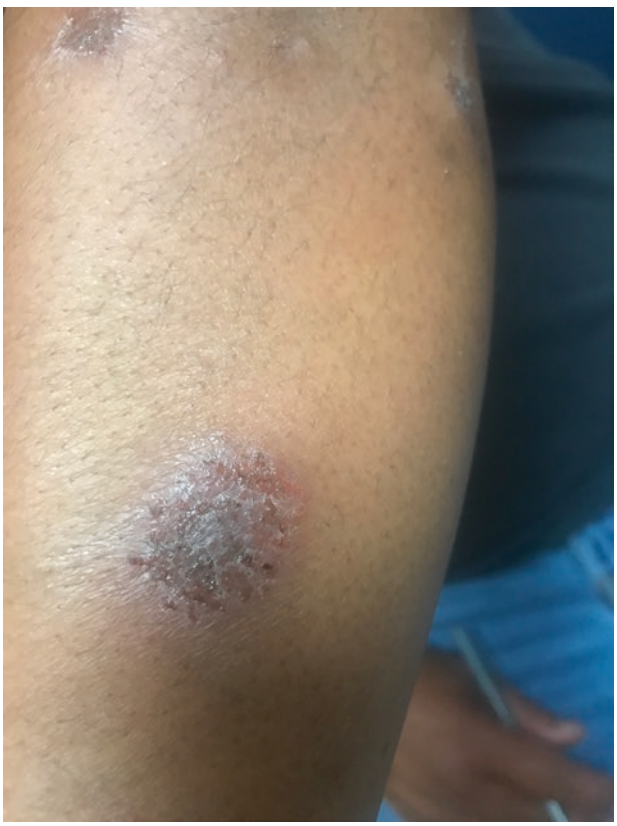


Fig. 2.44 Nummular dermatitis

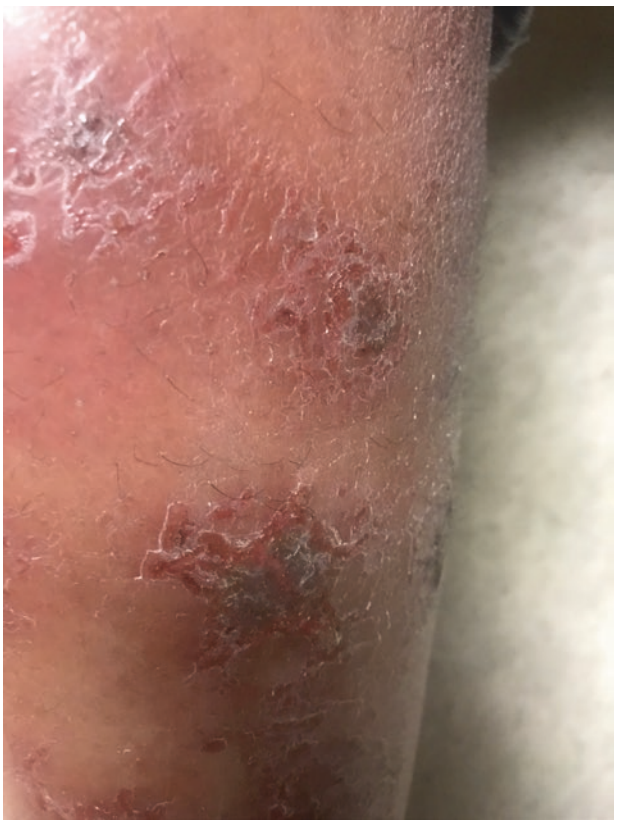


Fig. 2.45 Nummular dermatitis

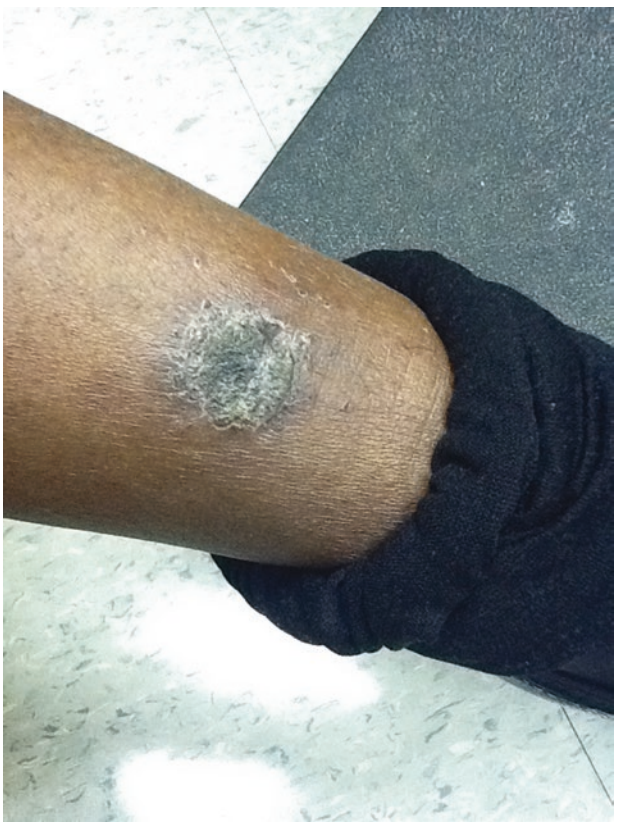
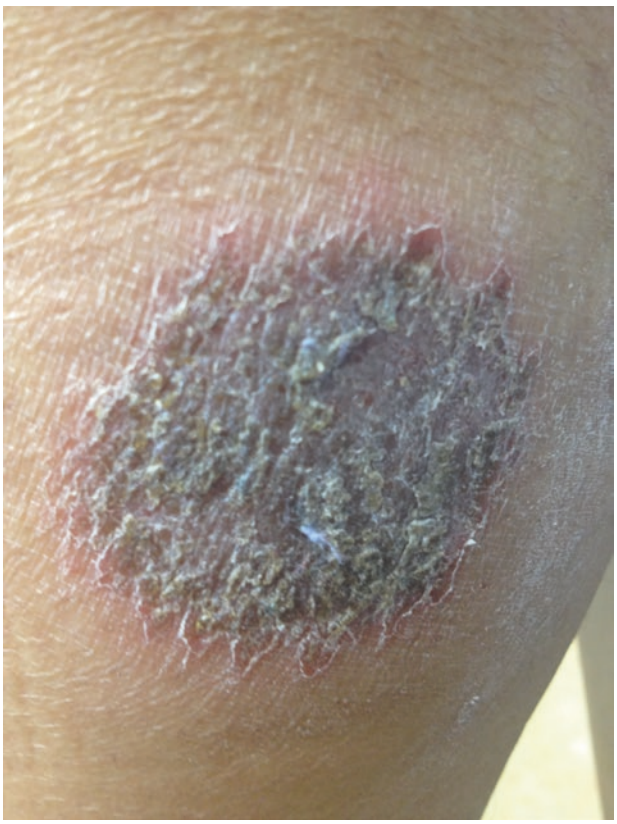


Fig. 2.46 Nummular dermatitis



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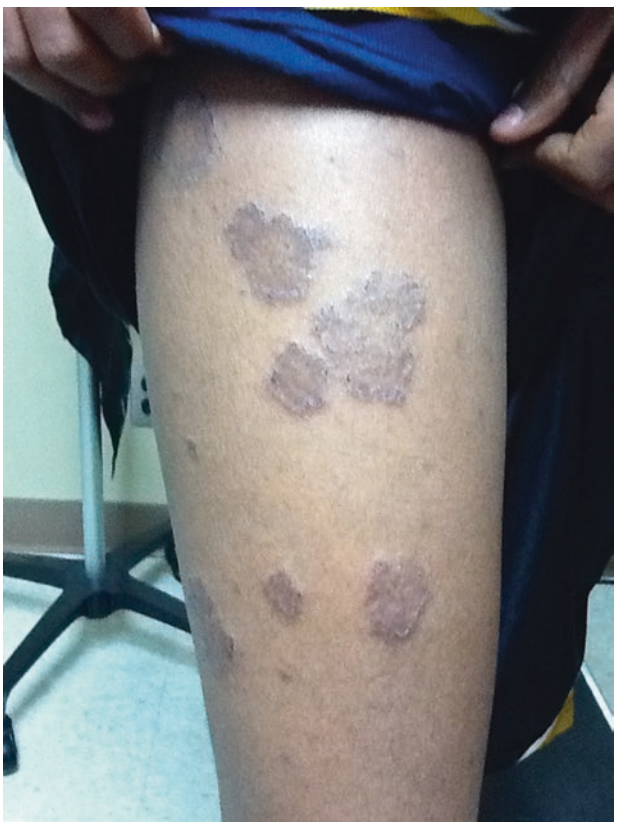
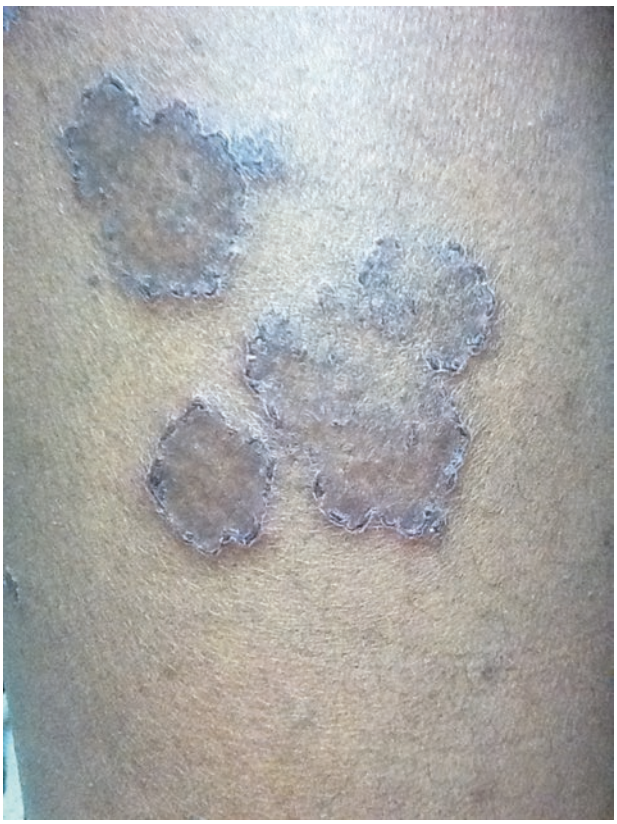


Fig. 2.48 Nummular dermatitis



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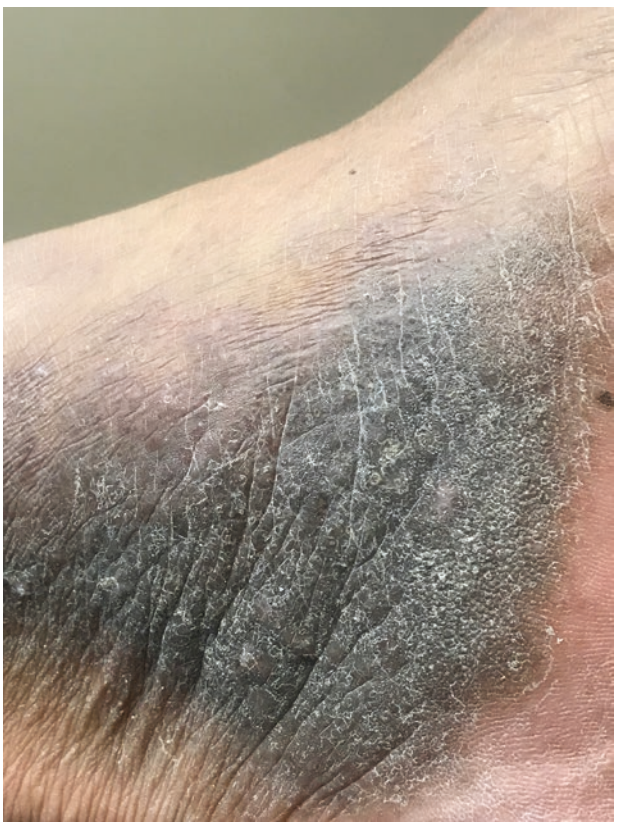

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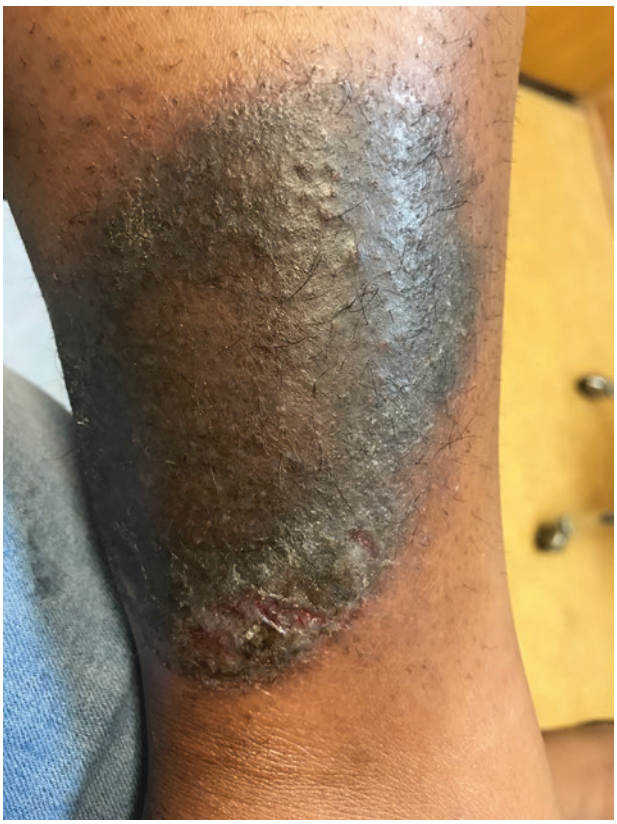

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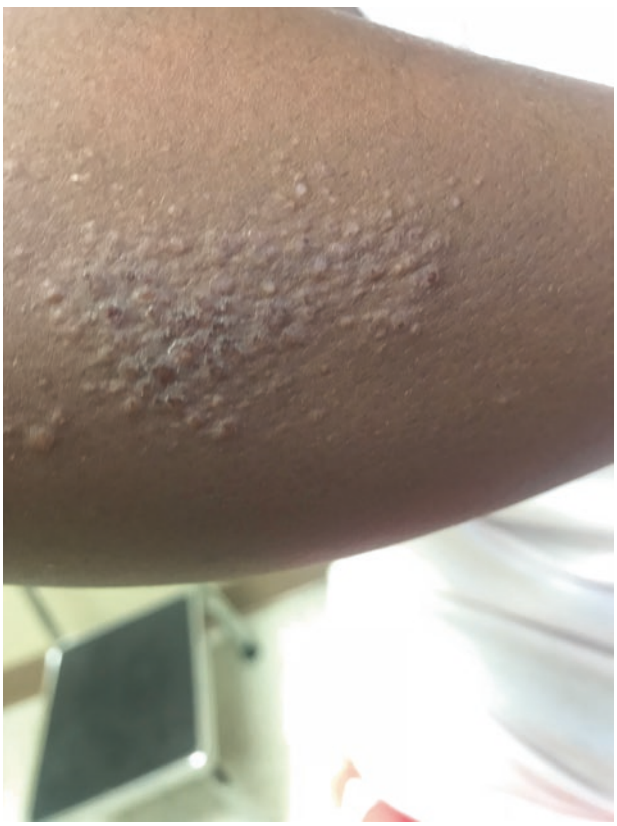

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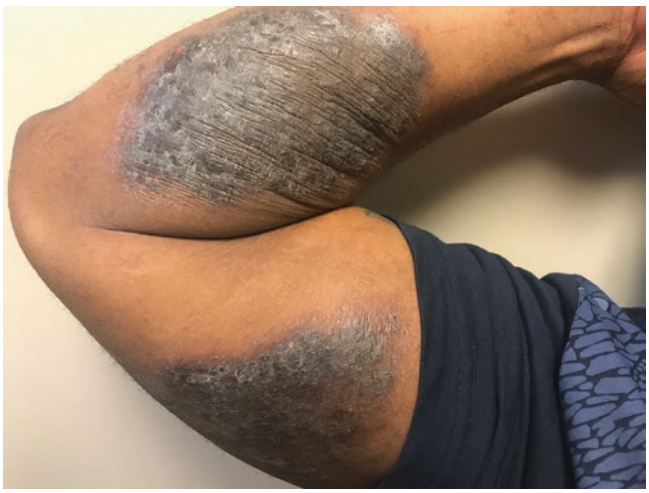

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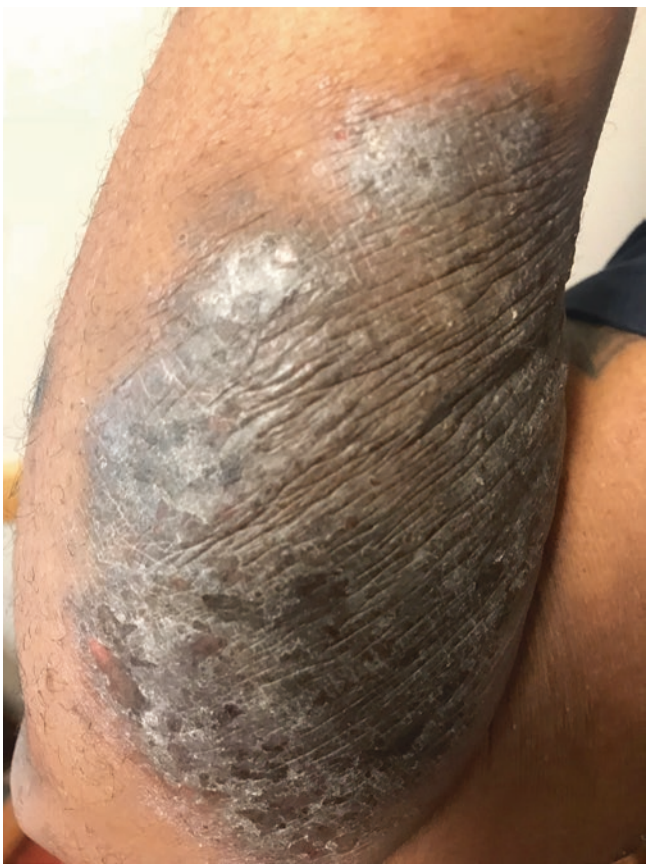


Fig. 2.54 Nummular dermatitis

2.1.3.2 Management

Frequent emollient use, topical steroids, and topical calcineurin inhibitors can be used to treat this chronic condition. Phototherapy is an option for disease refractory to topical treatment.

2.1.4 Seborrheic Dermatitis

This type of dermatitis is discussed fully in Chap. 8.

2.2 Psoriasis Vulgaris

Psoriasis vulgaris is a common, immune-mediated chronic skin disease that affects approximately 1–3% of the world's population. Psoriasis incidence is highest in young adulthood (age 20–30 years) and middle age (age 50–60), though it can develop at any age. The most common type, representing 90% of cases, is plaque-type psoriasis, which presents as well-demarcated, erythematous papules and plaques with white-silver adherent scale. Other common subtypes of psoriasis vulgaris include guttate psoriasis, erythrodermic psoriasis, inverse (intertriginous) psoriasis, and pustular psoriasis.

Both environmental factors and genetic predisposition underlie the pathogenesis of psoriasis, leading to upregulation of the Th17 immune pathway and the increased proliferation of keratinocytes associated with the development of clinical disease. Psoriatic arthritis, a destructive inflammatory arthritis, develops in up to 30% of patients with cutaneous psoriasis; it can precede onset of skin manifestations.

2.2.1 Presentation in Black Skin

In the United States, the prevalence of psoriasis in African Americans (2.0%) and Hispanics (1.6%) is less than in Caucasians (3.7%). This pattern is also seen worldwide, with whites having higher prevalence rates than nonwhite ethnic groups. The genetic polymorphisms associated with psoriasis vary significantly among ethnic groups.

Clinically, the erythema of psoriasis lesions in skin of color may be less pink or red, and more brown or violet in hue. Plaque-type psoriasis lesions have been described as scalier, thicker, and covering a larger body surface area in patients of African descent, and dyspigmentation (both hyperpigmentation and hypopigmentation) is common (Figs. 2.55–2.92). Some studies have indicated higher rates of intertriginous psoriasis in whites than in Asian and African populations.

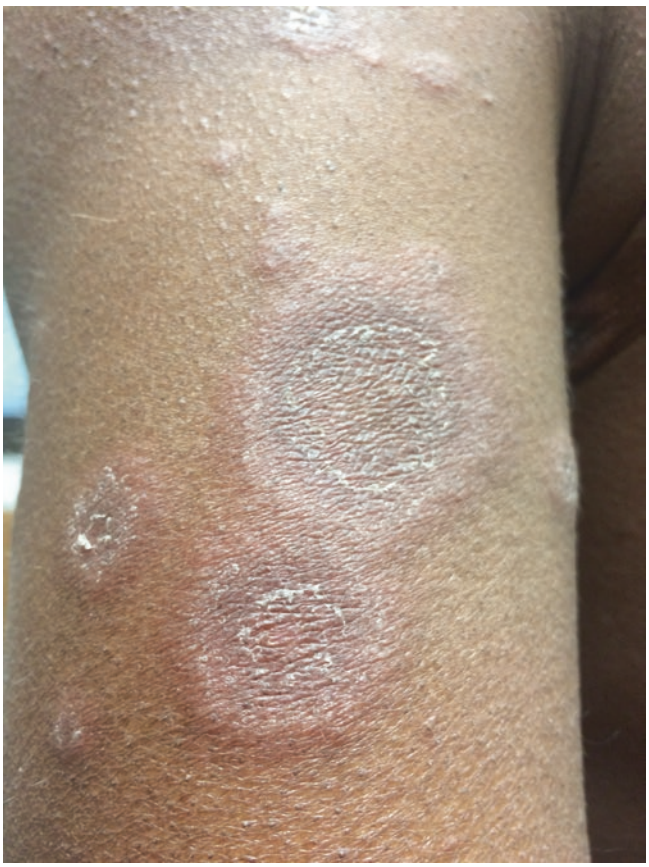
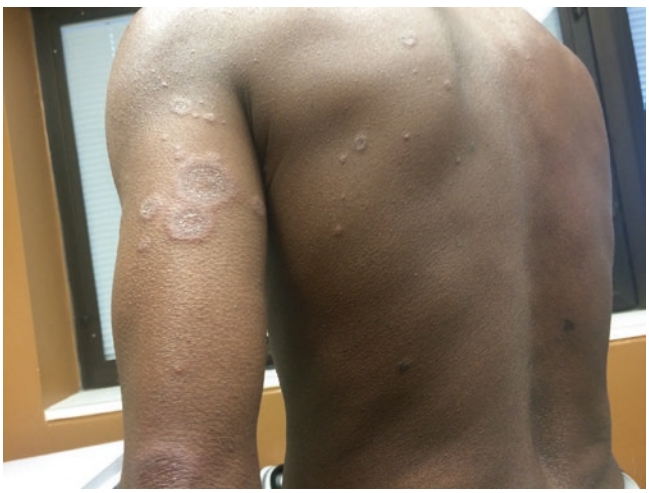


Fig. 2.55 Psoriasis



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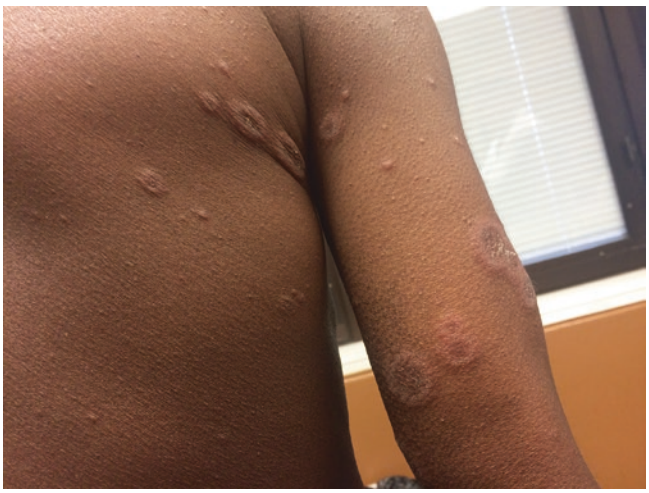

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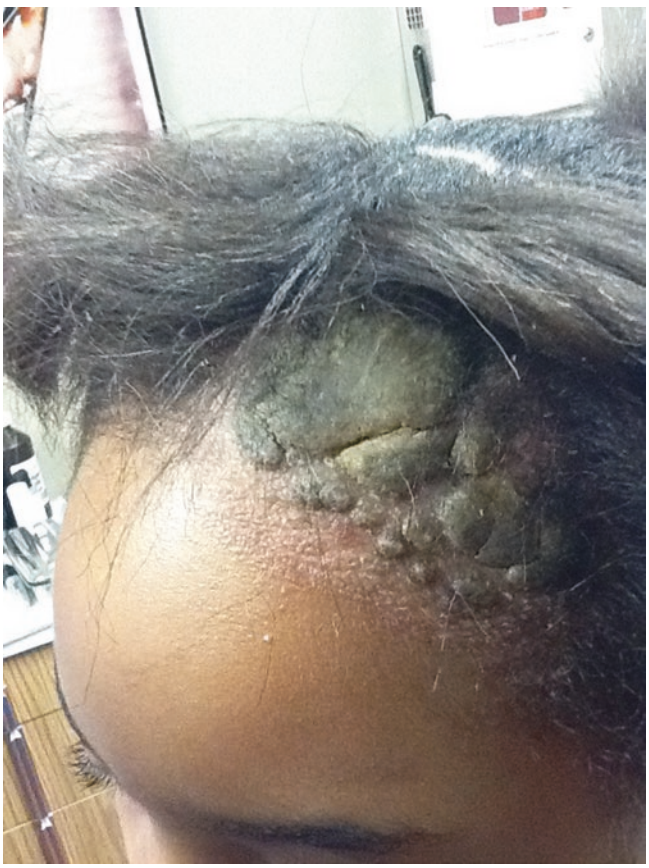

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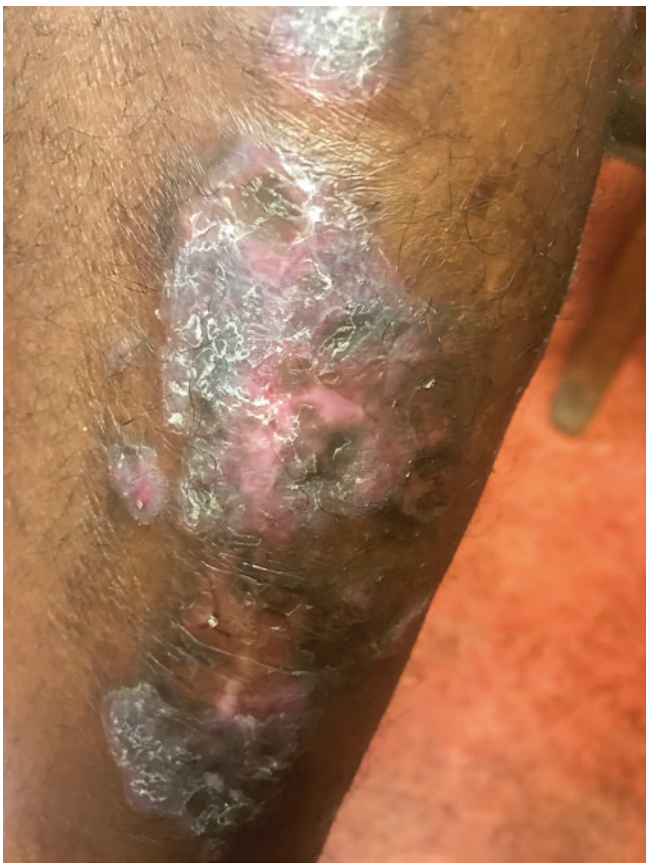

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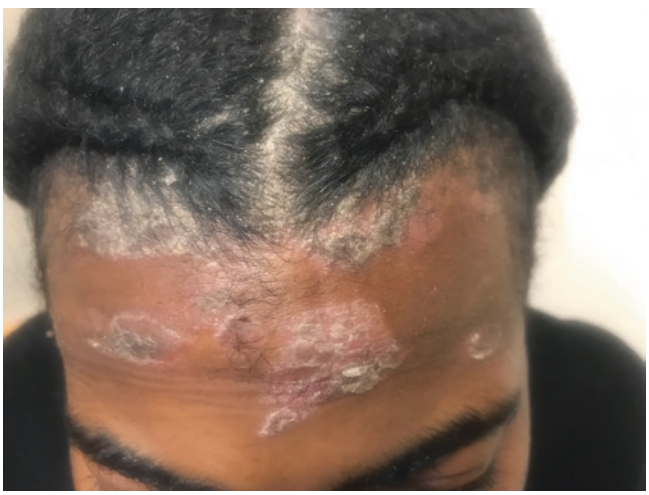


Fig. 2.60 Psoriasis

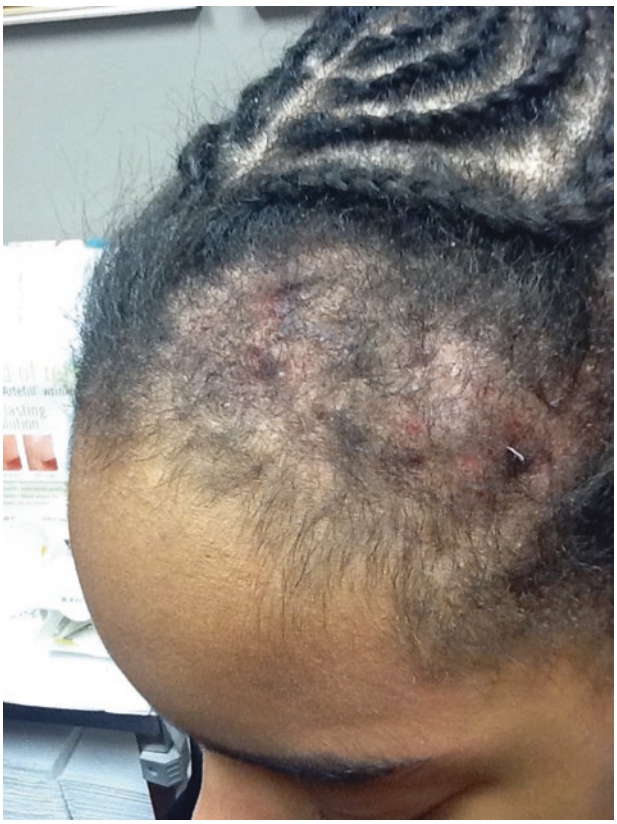


Fig. 2.61 Psoriasis

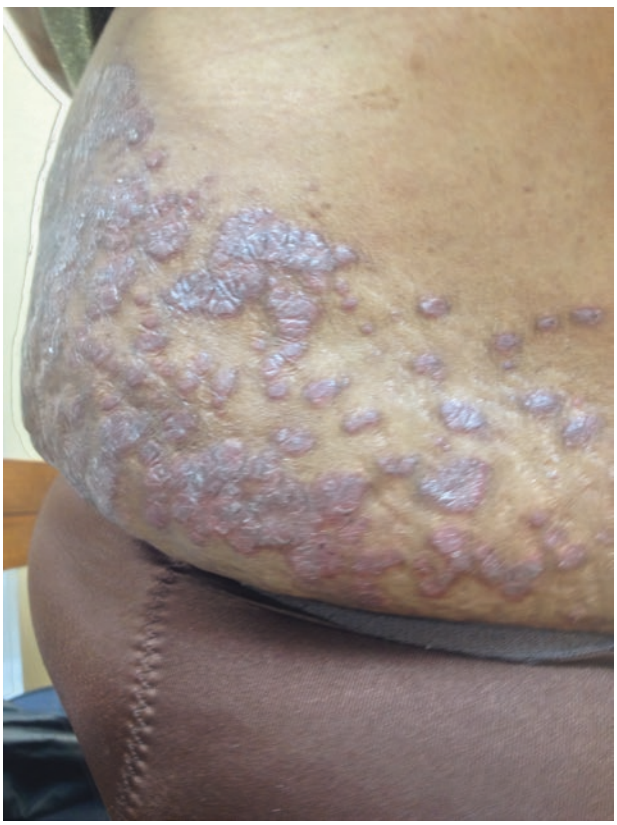
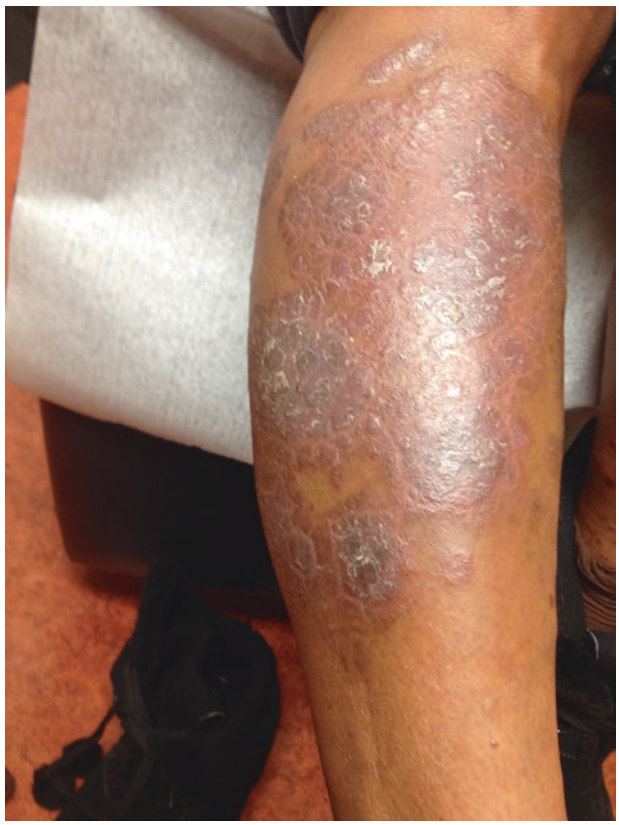


Fig. 2.62 Psoriasis



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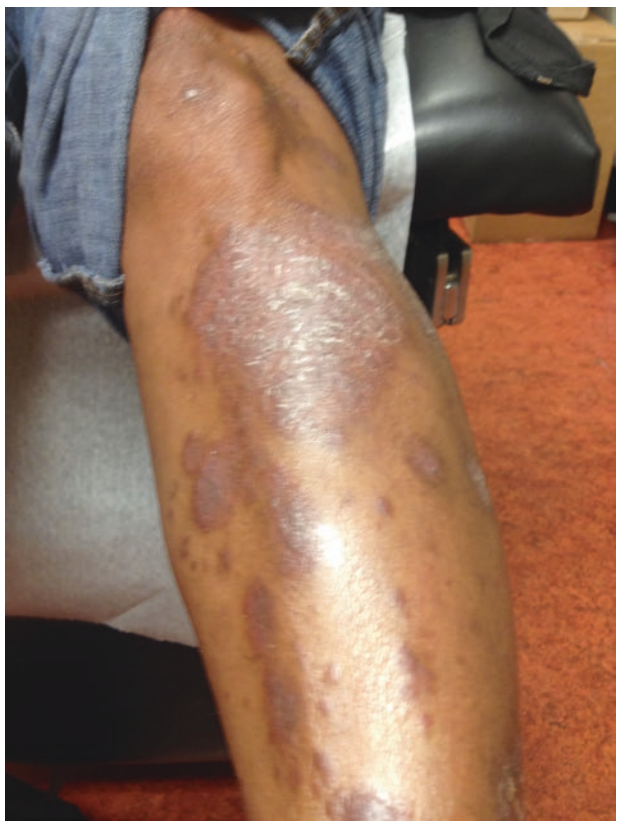

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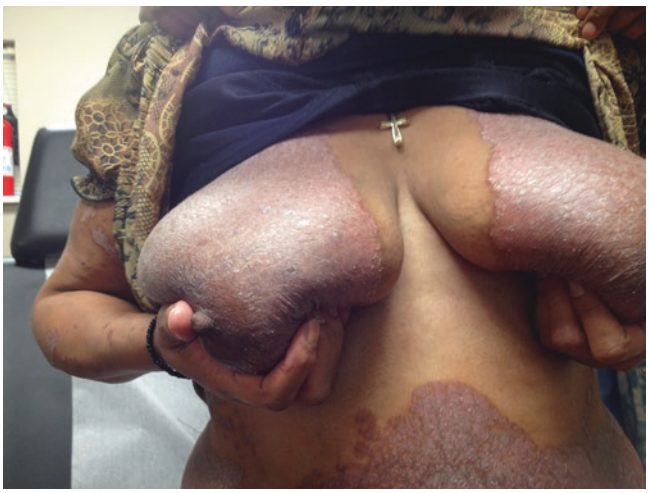


Fig. 2.65 Psoriasis

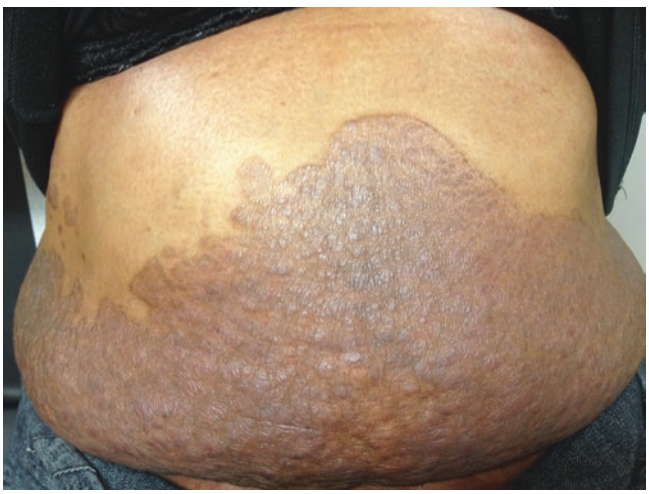


Fig. 2.66 Psoriasis

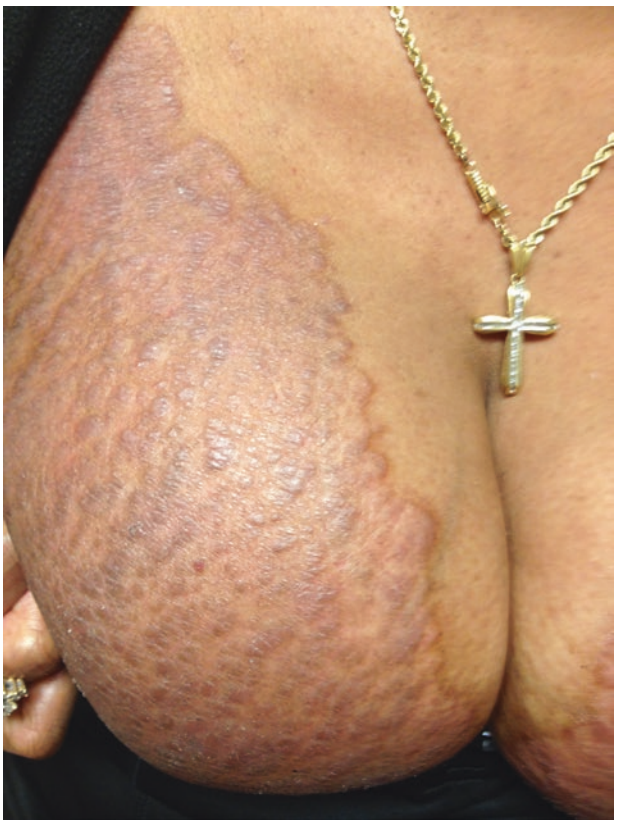
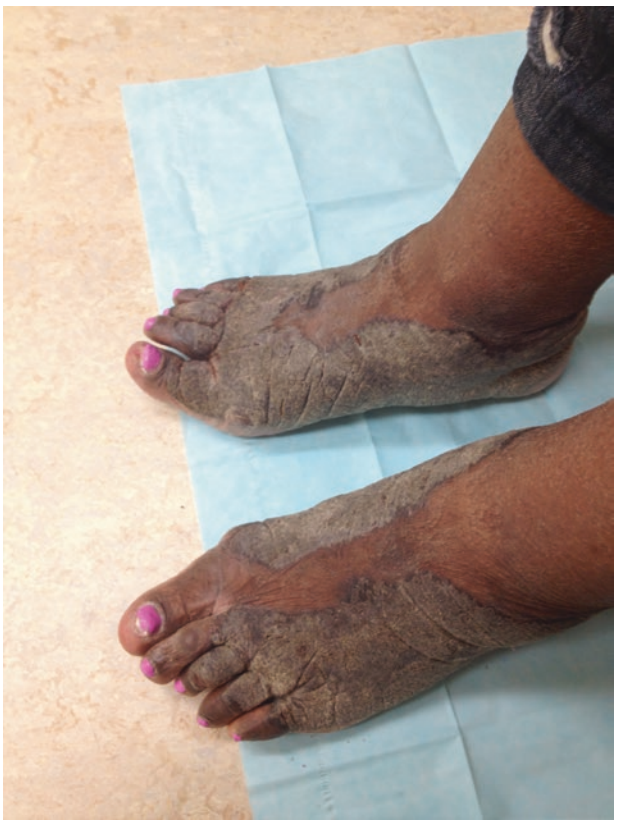


Fig. 2.67 Psoriasis



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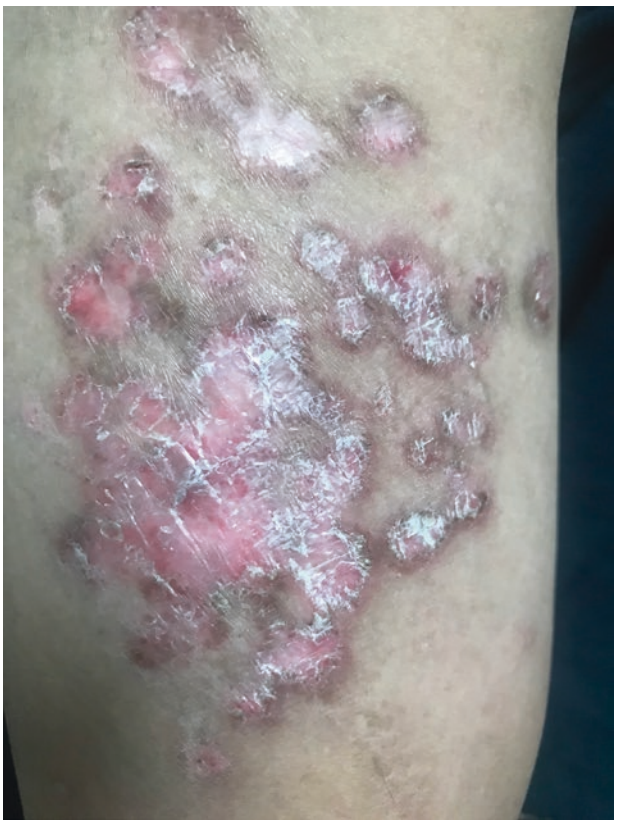

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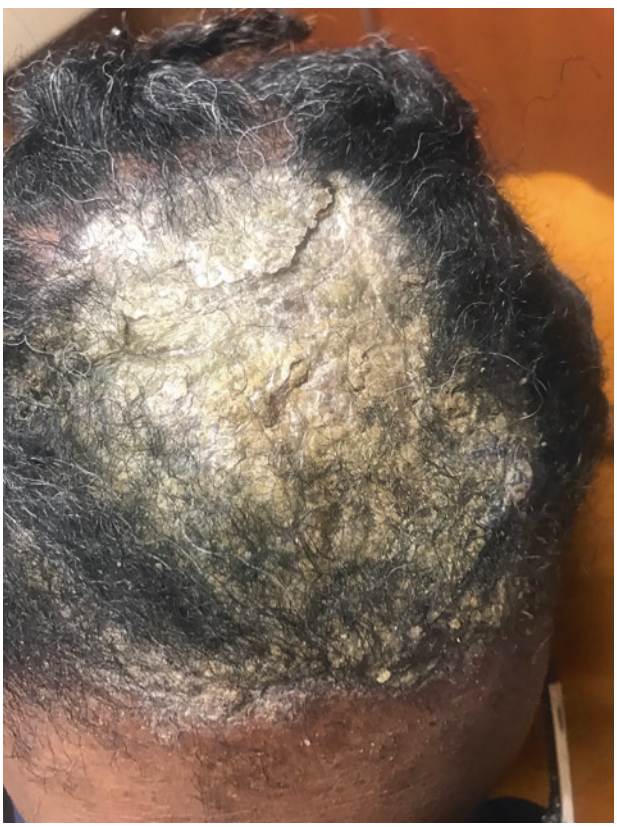

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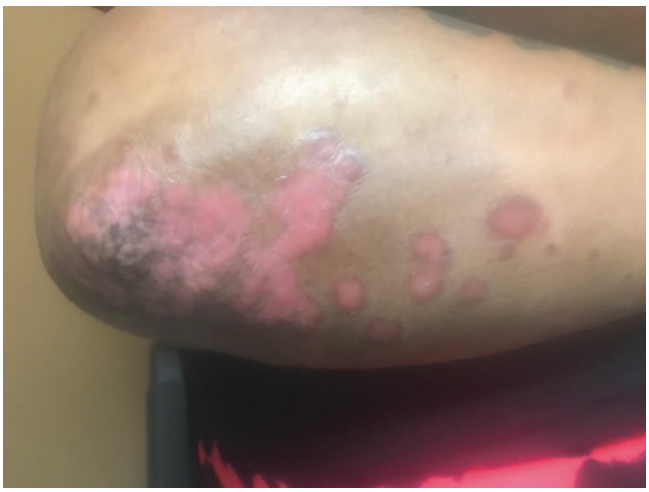

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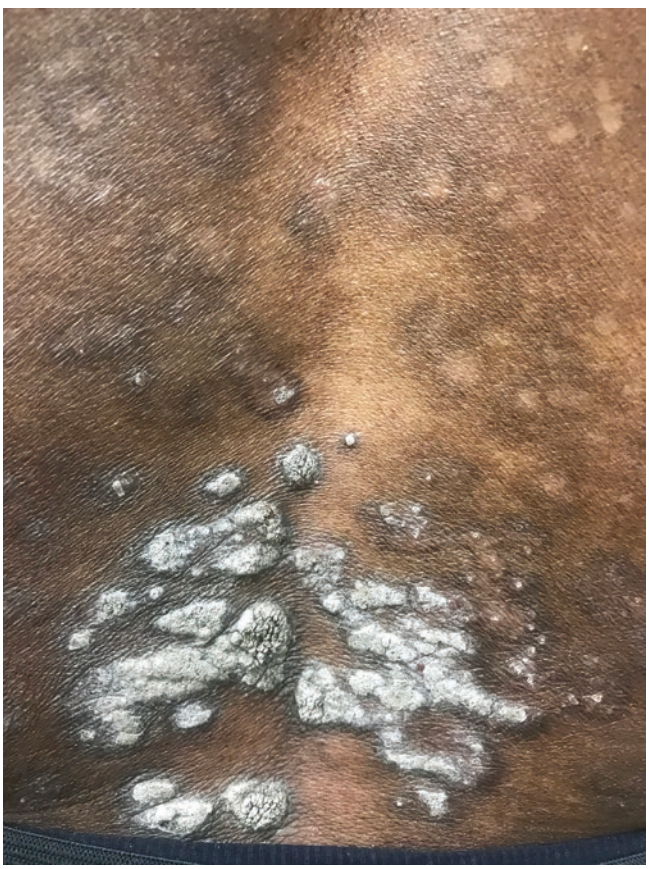

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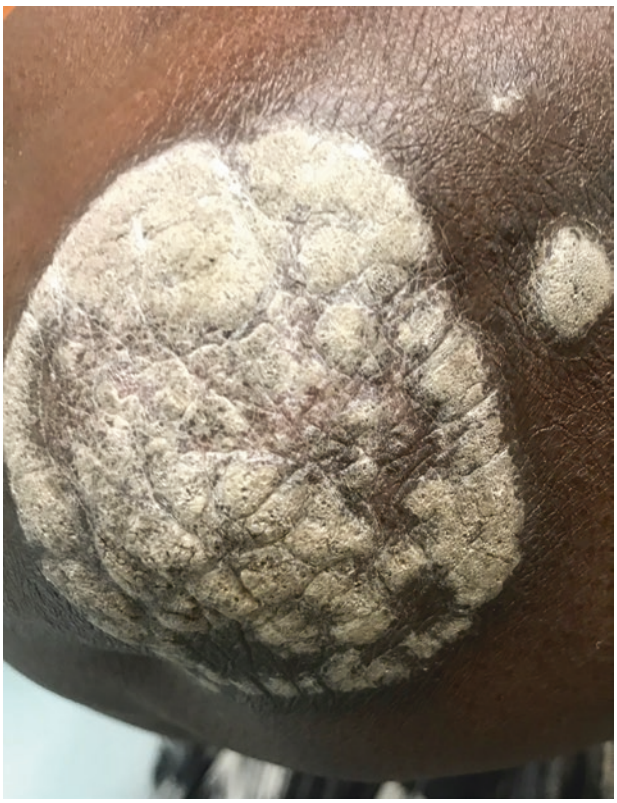

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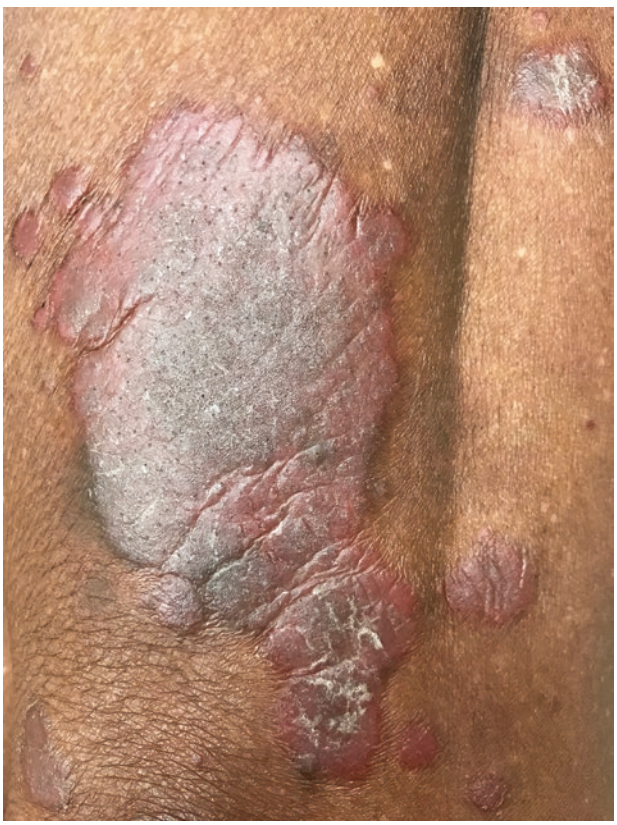

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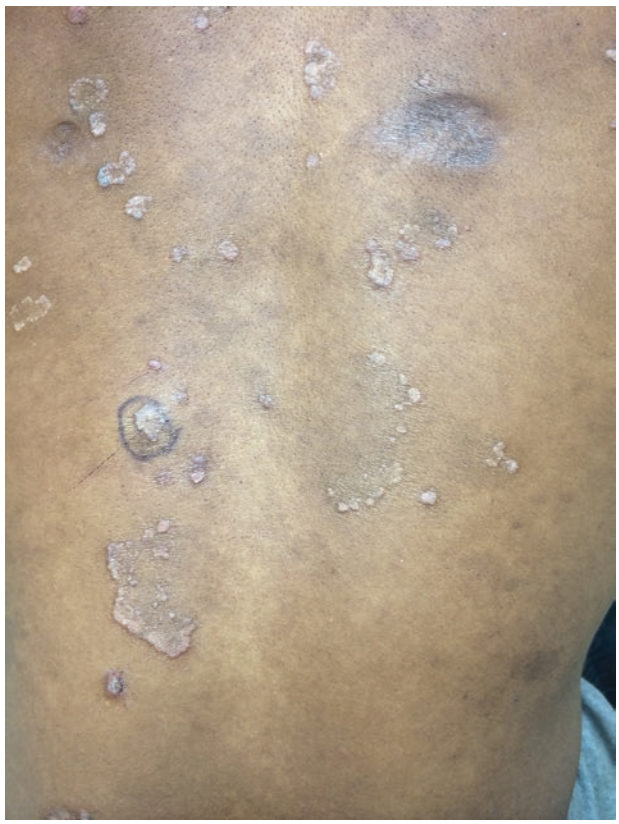

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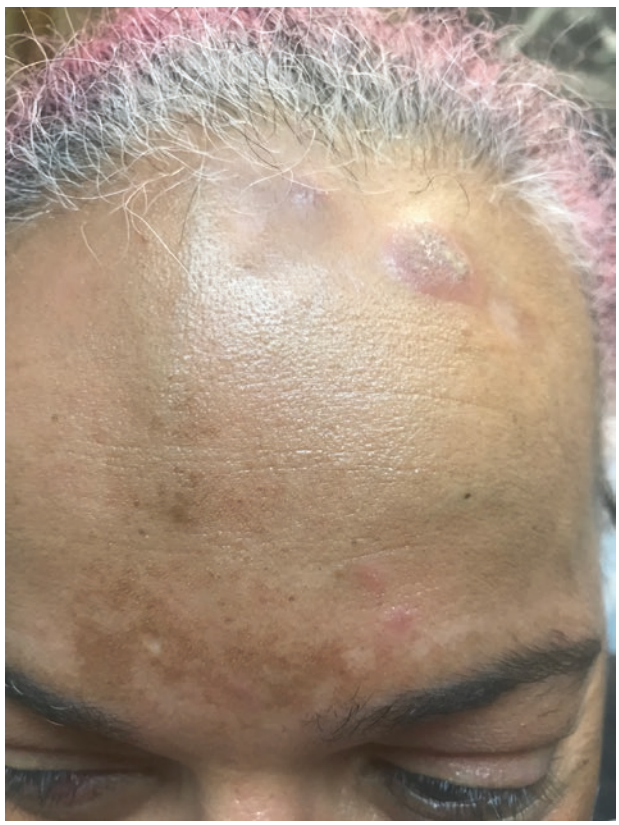

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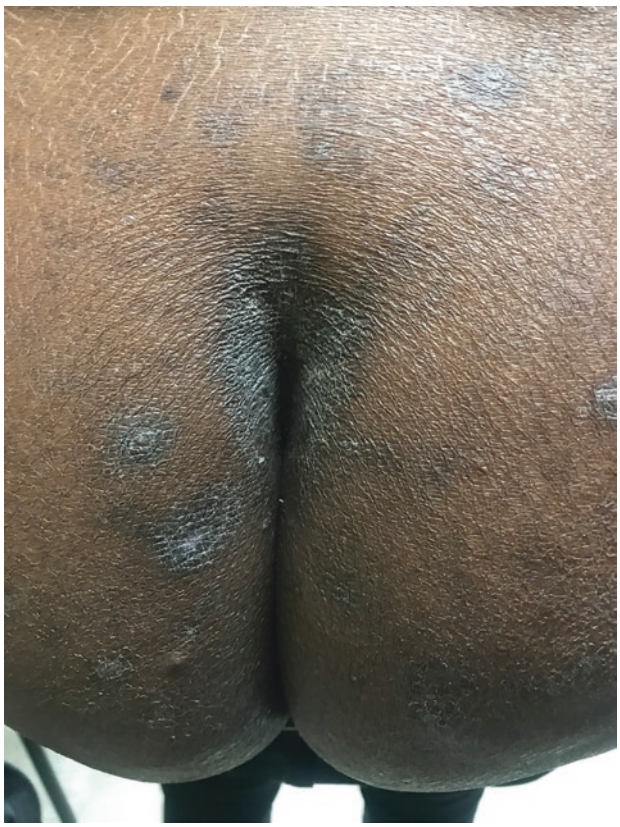

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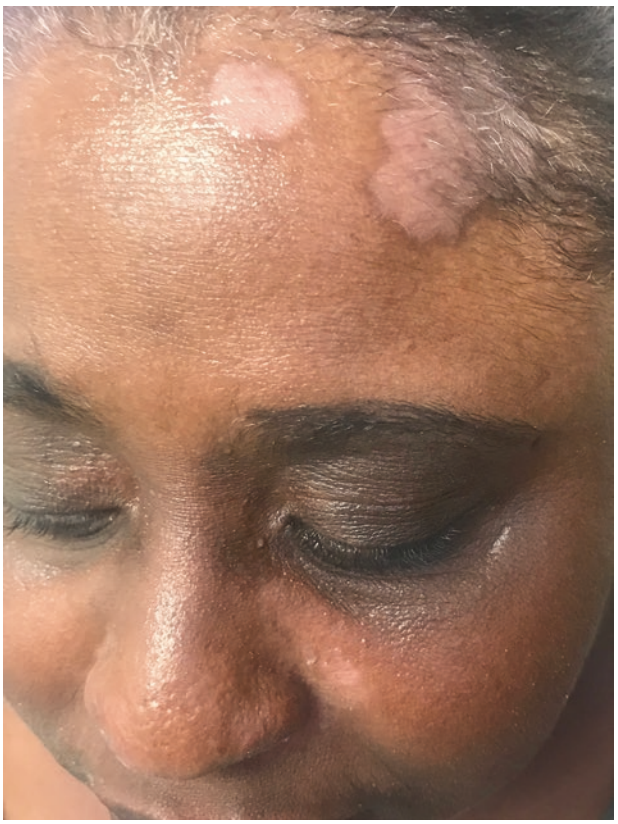

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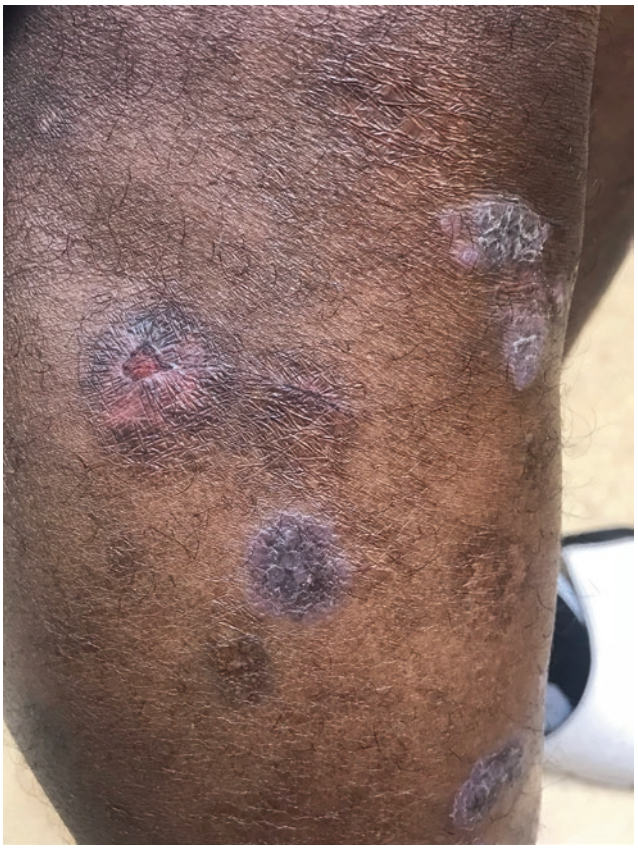

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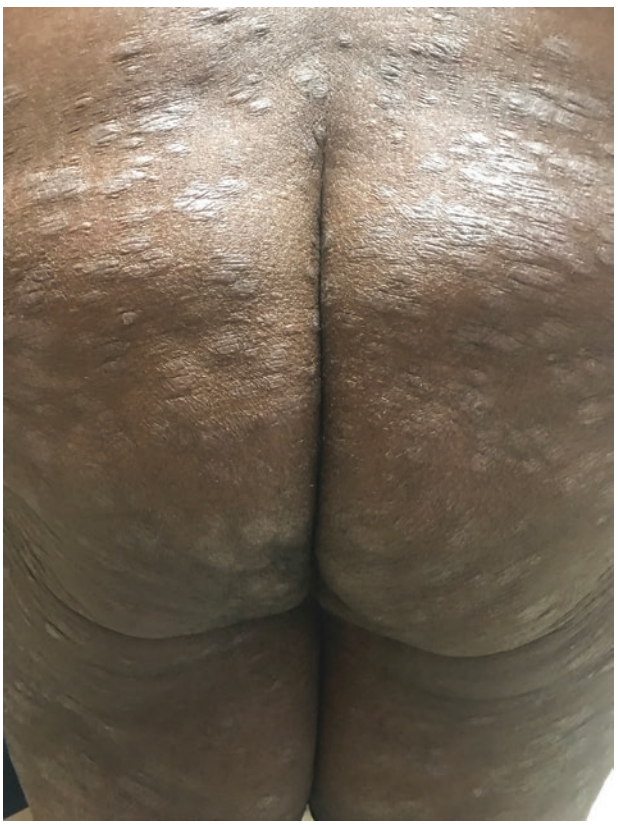

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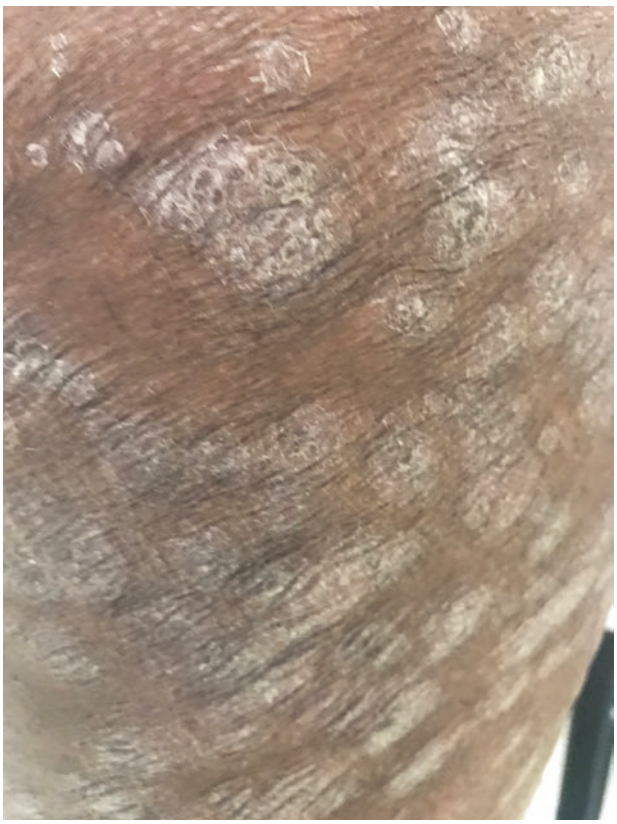

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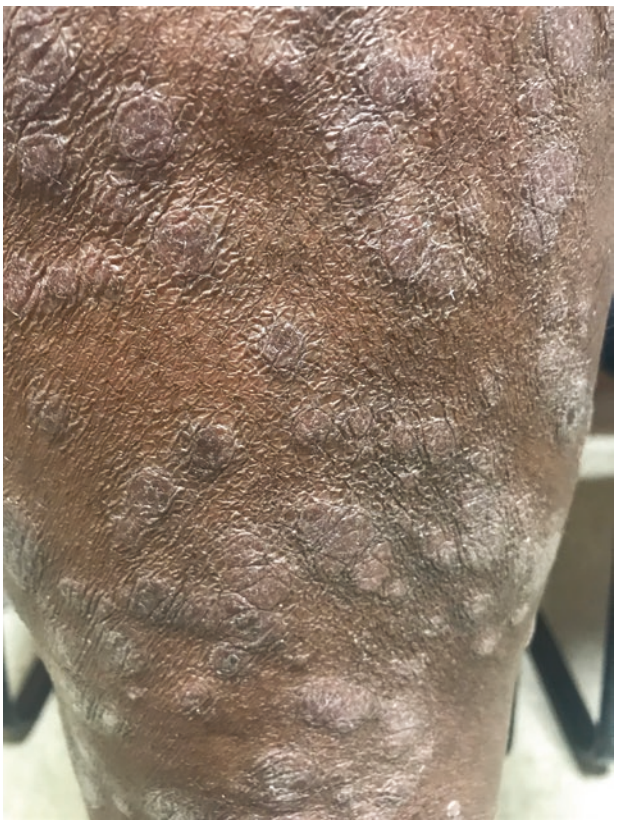

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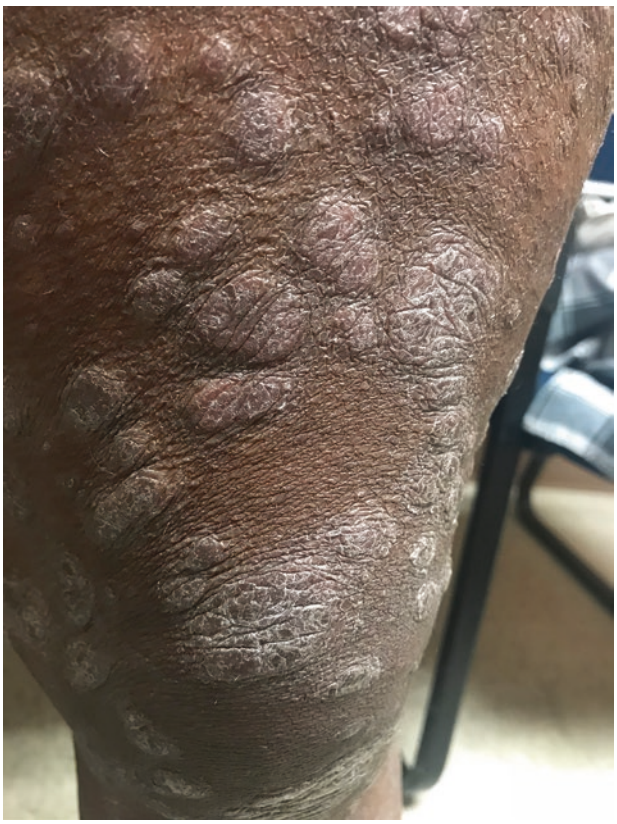

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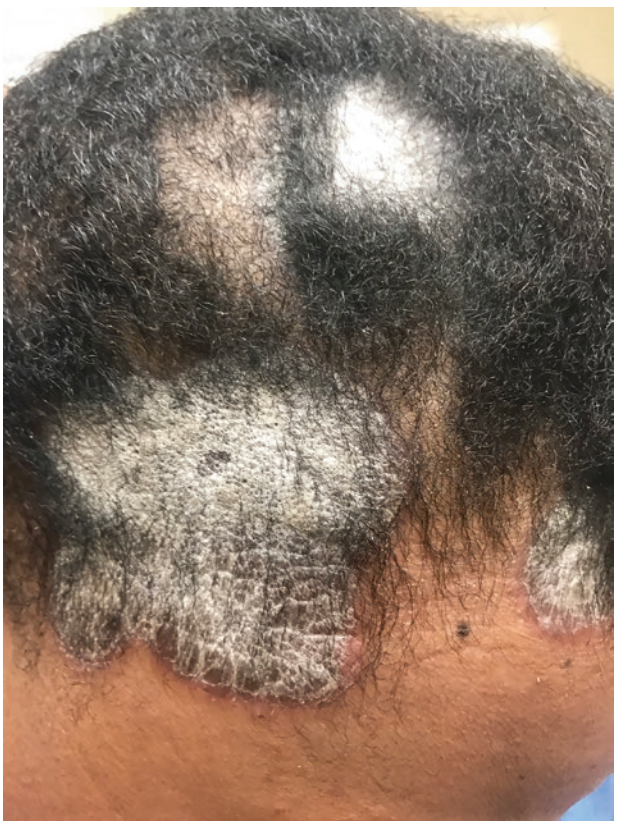


Fig. 2.84 Psoriasis

Fig. 2.85 Psoriasis

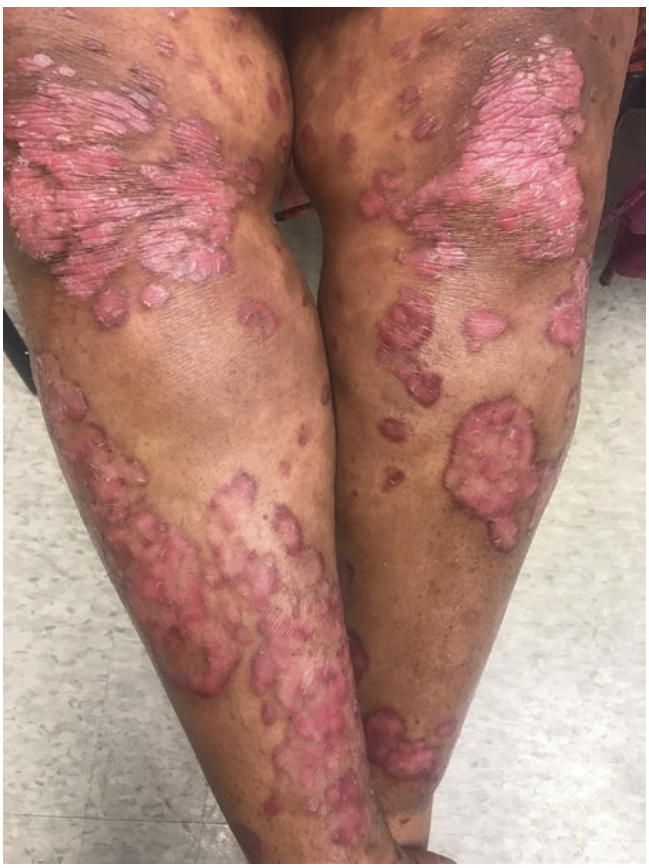


Fig. 2.86 Psoriasis

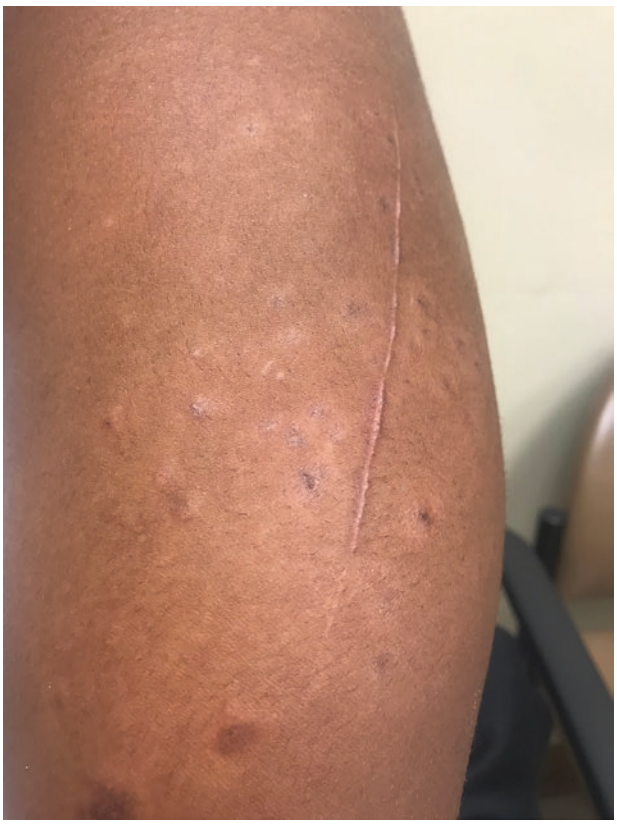


Fig. 2.87 Psoriasis

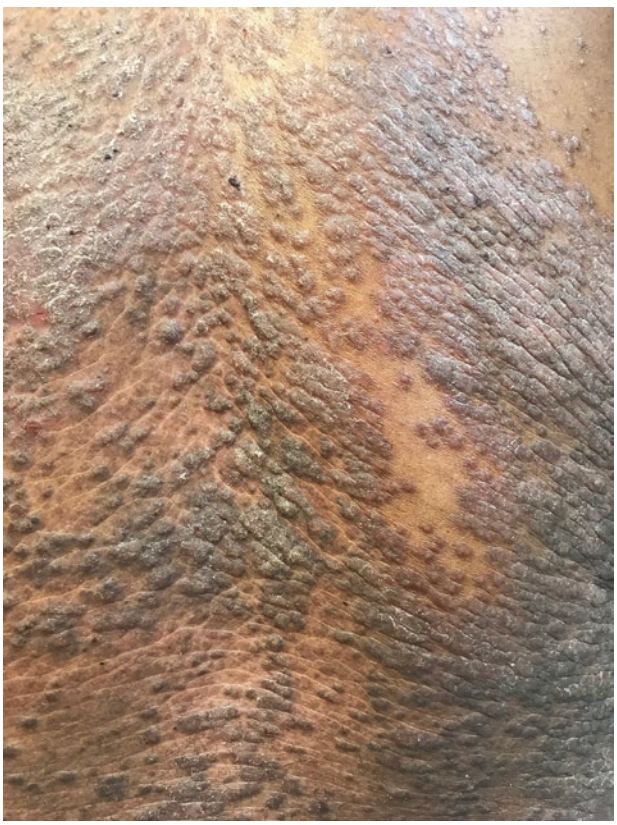
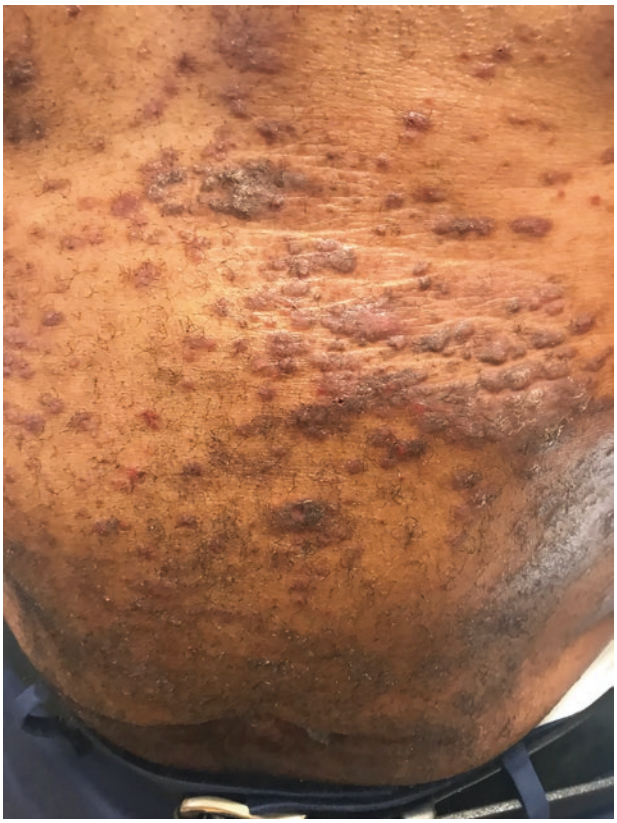


Fig. 2.88 Psoriasis



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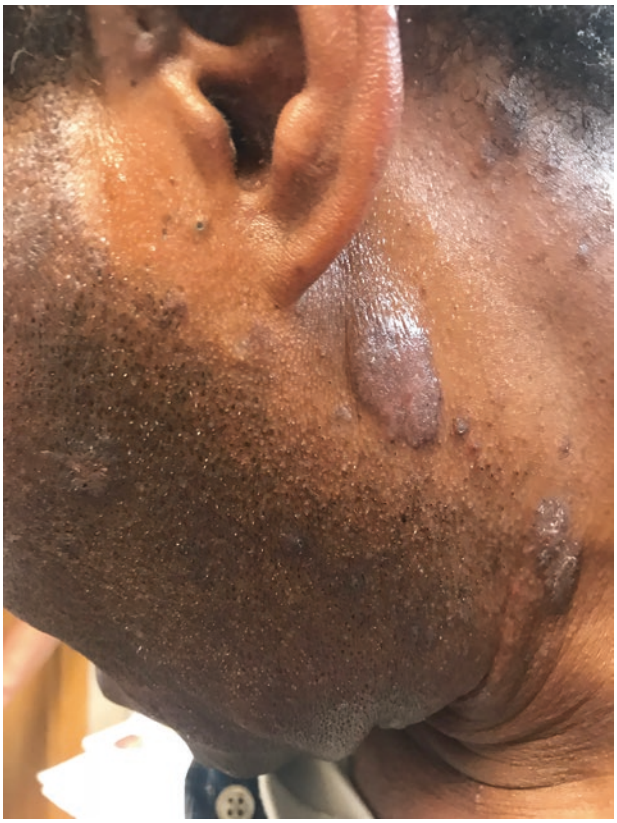

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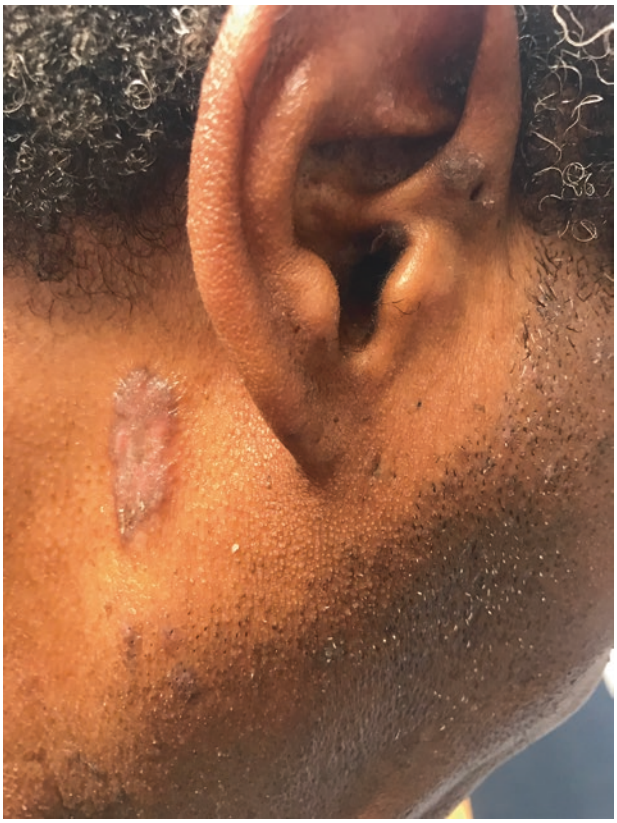

Fig. 2.91 Psoriasis

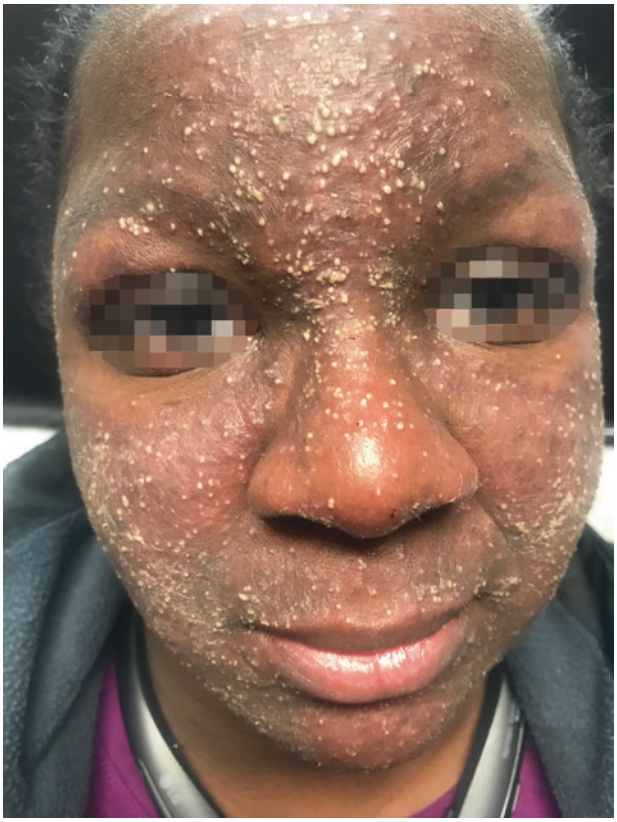

Fig. 2.92 Pustular psoriasis

2.2.2 Management

The approach to treatment of psoriasis is generally consistent across ethnic groups, with first-line therapy consisting of topical steroids, topical vitamin D analogs, combinations of these, topical retinoids, and topical calcineurin inhibitors. Non-topical options include phototherapy, oral immunosuppressant medications, systemic retinoids, and targeted biologic medications. Treatment of associated post-inflammatory dyspigmentation should be postponed until active psoriasis is well controlled, as many skin lightening agents can cause skin irritation and exacerbate the underlying inflammatory disease.

2.3 Lichen Planus

Lichen planus (LP) is a common inflammatory skin disease of the skin, nails, and mucous membranes, with a worldwide incidence of approximately 1%. Onset is typically in middle age (age 30–60), with a slight female predominance. Studies have not demonstrated a racial predilection in LP, but we have observed more in black patients. LP has been noted to develop in association with viral illness (especially hepatitis C), exposure to dental filler materials, autoimmune disease, and medications.

2.3.1 Presentation in Black Skin

The classic presentation of LP—pruritic, polygonal-shaped, purple papules with fine white scale—is most commonly found on the volar wrist, forearms, genitalia, lower extremities, and lower back. These lesions may appear more brown than purple in skin of color, but the associated fine white scale remains present, and the violaceous color of active lesions can often be seen at lesion borders. The oral white streaks called Wickham striae are also present.

Hypertrophic LP, though more common in skin of color, can have a unique appearance in this population. The thick, pruritic, hyperkeratotic papules and plaques of hypertrophic LP can be markedly hyperpigmented or hypopigmented and more diffusely distributed in patients of color (Figs. 2.93–2.115).

The post-inflammatory hyperpigmentation (PIH) seen with lichen planus in skin of color is often darker and more persistent than in other inflammatory skin diseases. Distinguishing PIH from active lesions is important, as it allows for early and appropriate treatment of active disease, decreasing the risk of development of additional PIH.

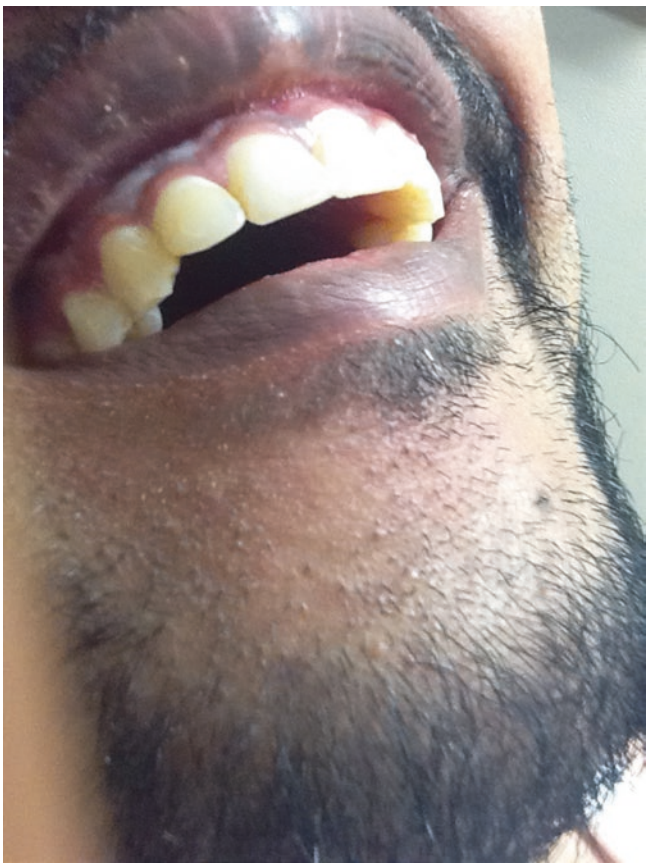

Fig. 2.93 Lichen planus

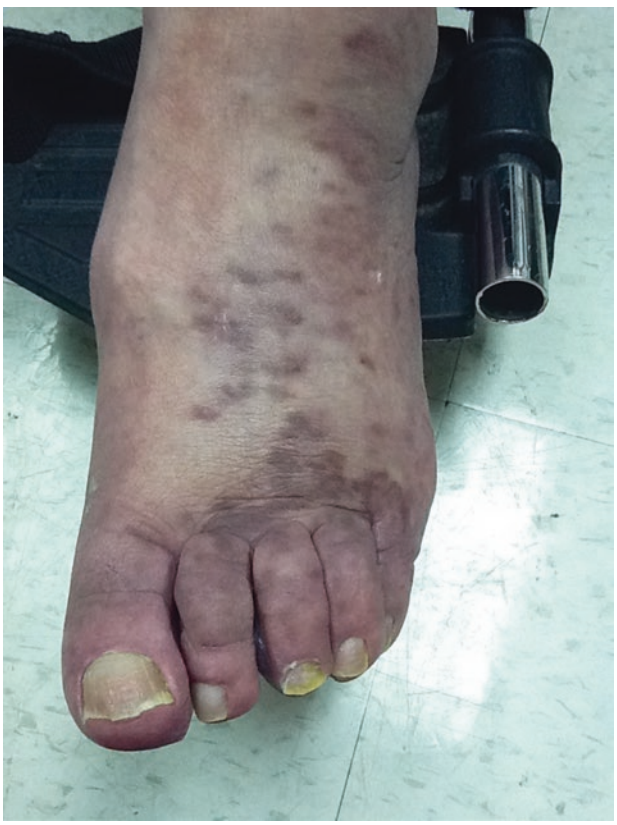

Fig. 2.94 Lichen planus

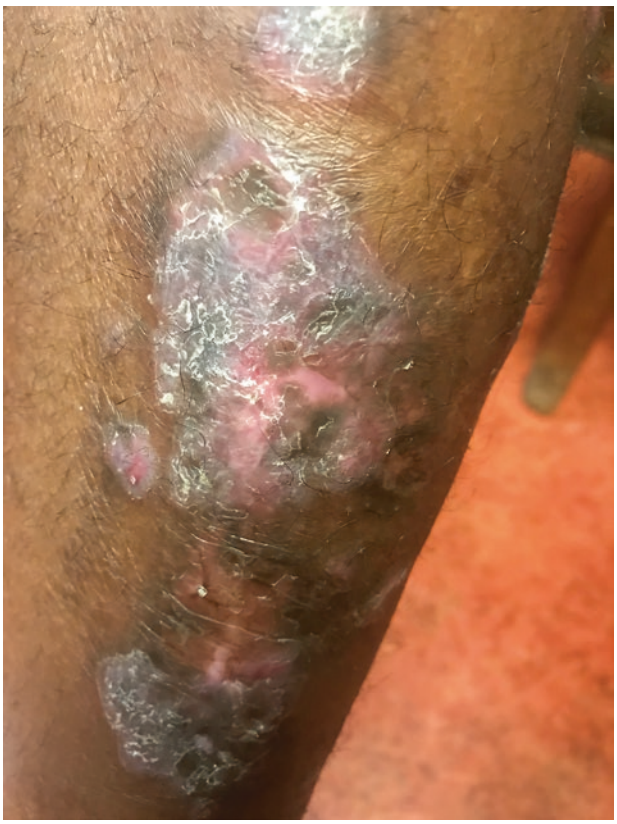

Fig. 2.95 Lichen planus

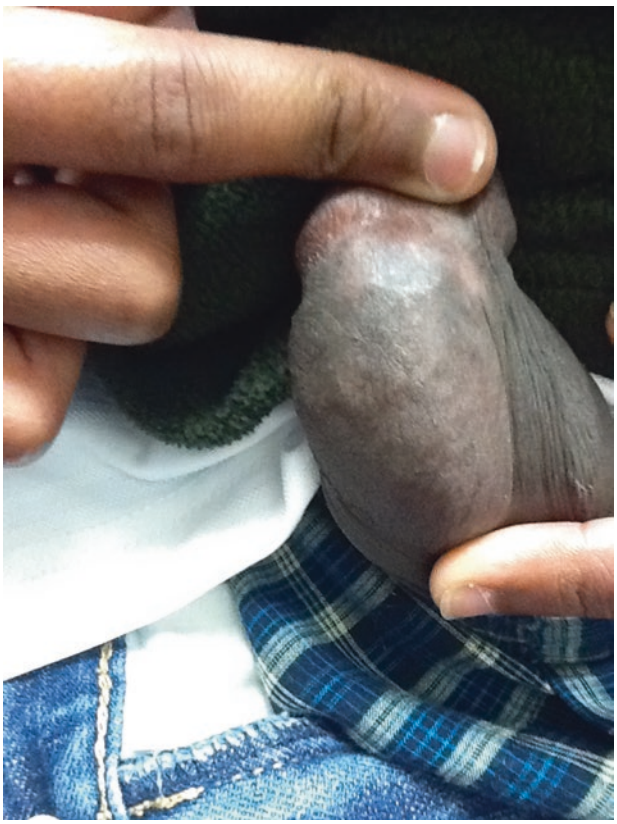

Fig. 2.97 Lichen planus

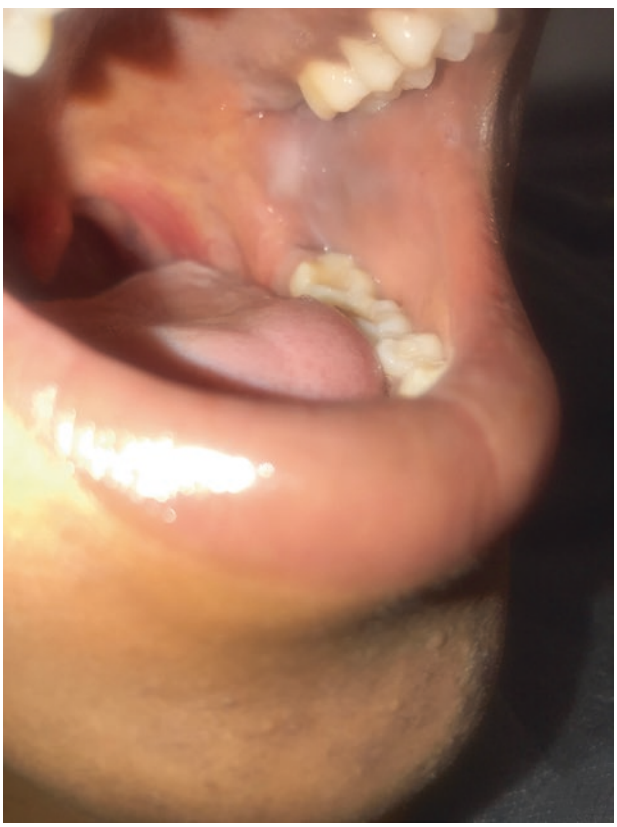

Fig. 2.96 Oral Wickham striae

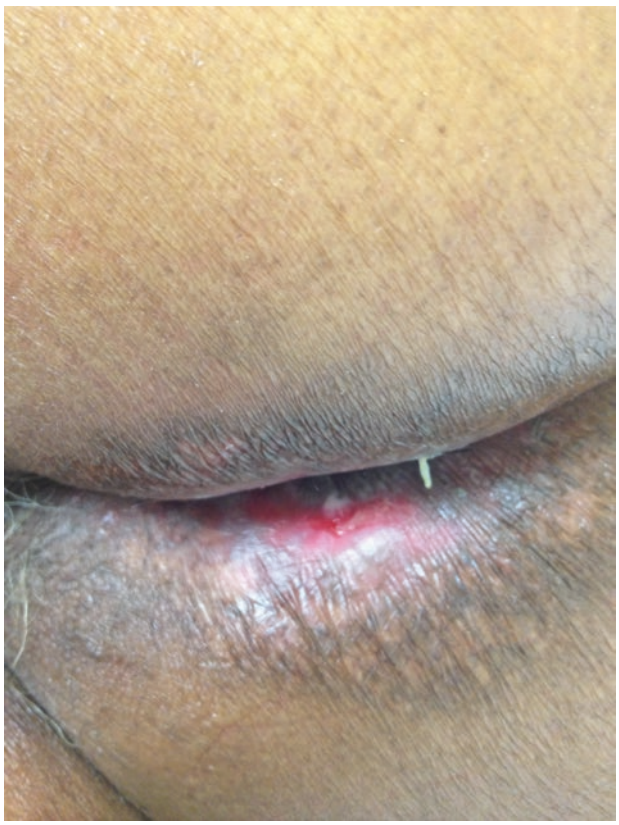

Fig. 2.98 Ulcerative lichen planus

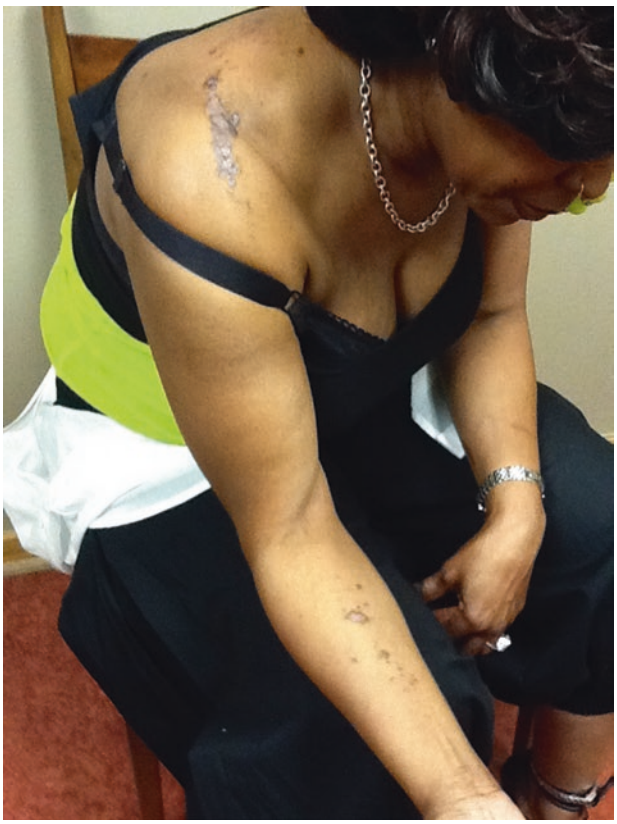

Fig. 2.99 Linear lichen planus

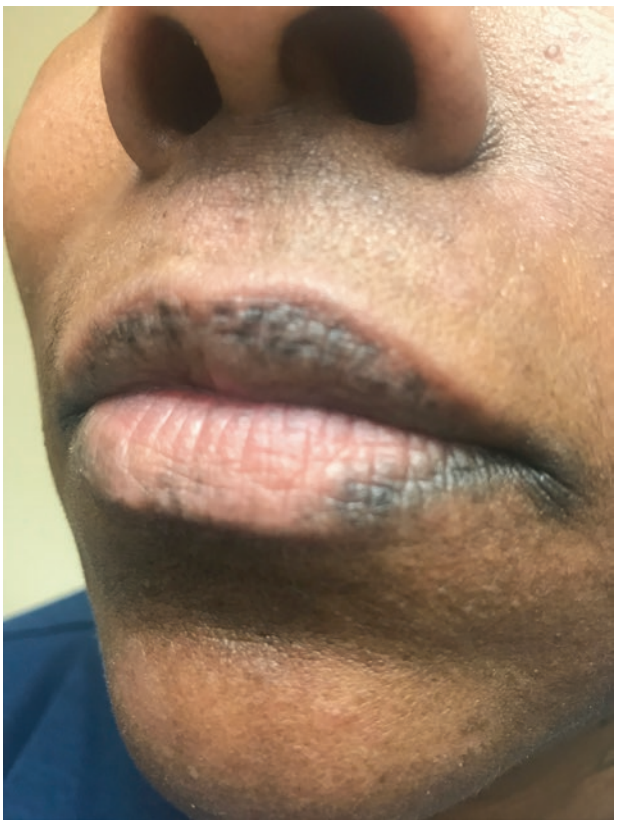

Fig. 2.101 Lichen planus

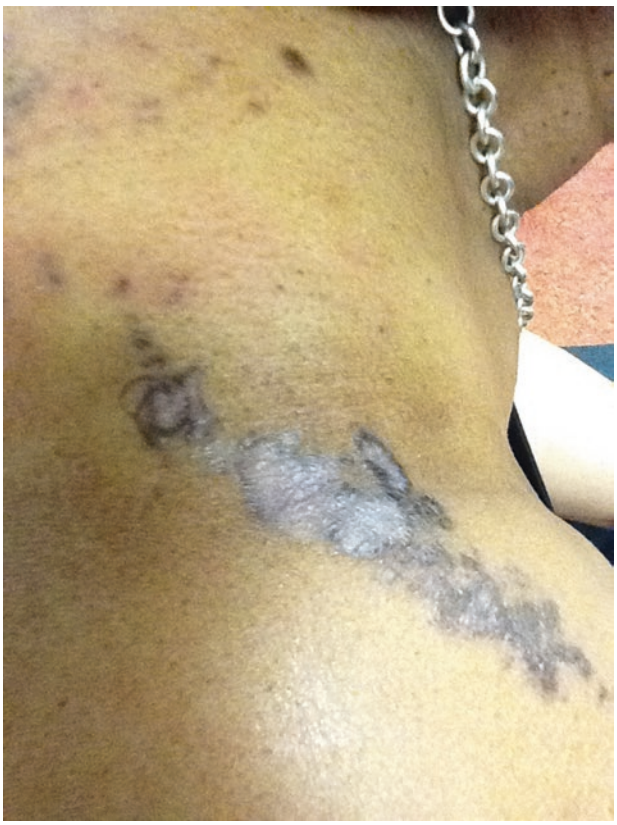

Fig. 2.100 Linear lichen planus

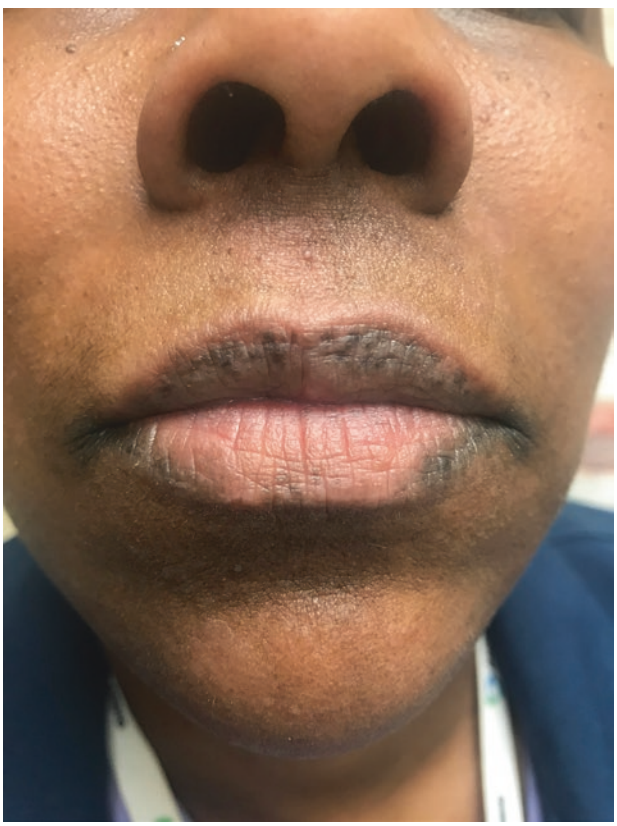

Fig. 2.102 Lichen planus

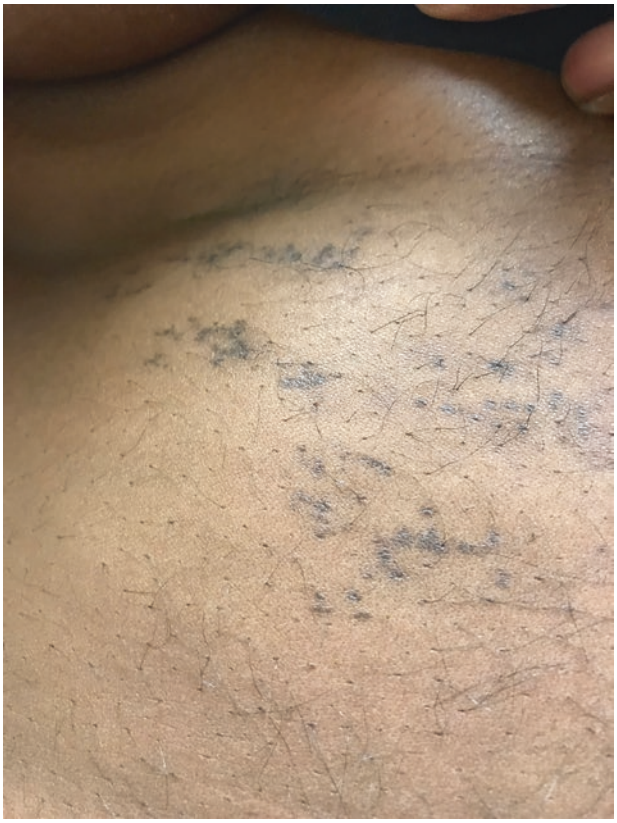

Fig. 2.103 Lichen planus

2.3.2 Management

The treatment approach for LP in skin of color is consistent with the approach in other populations. Topical and oral corticosteroids, and intralesional corticosteroids for hypertrophic variants, are first-line therapy, followed by phototherapy, oral retinoids, and oral immunosuppressants for more severe or treatment-resistant disease.

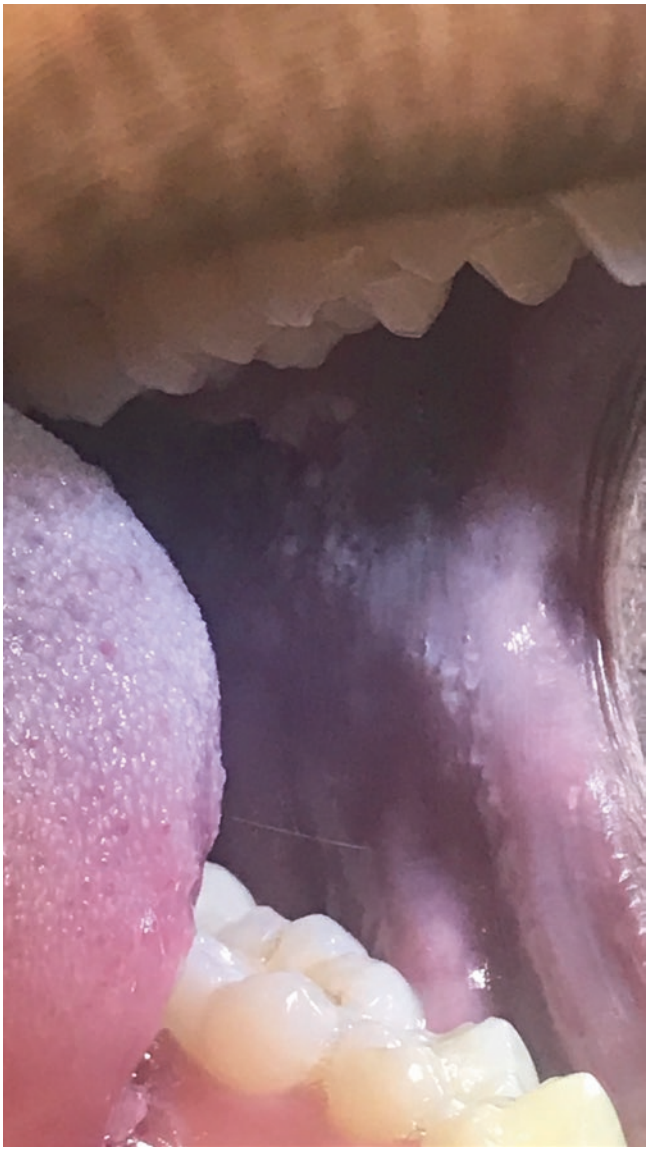

Fig. 2.104 Oral Wickham striae

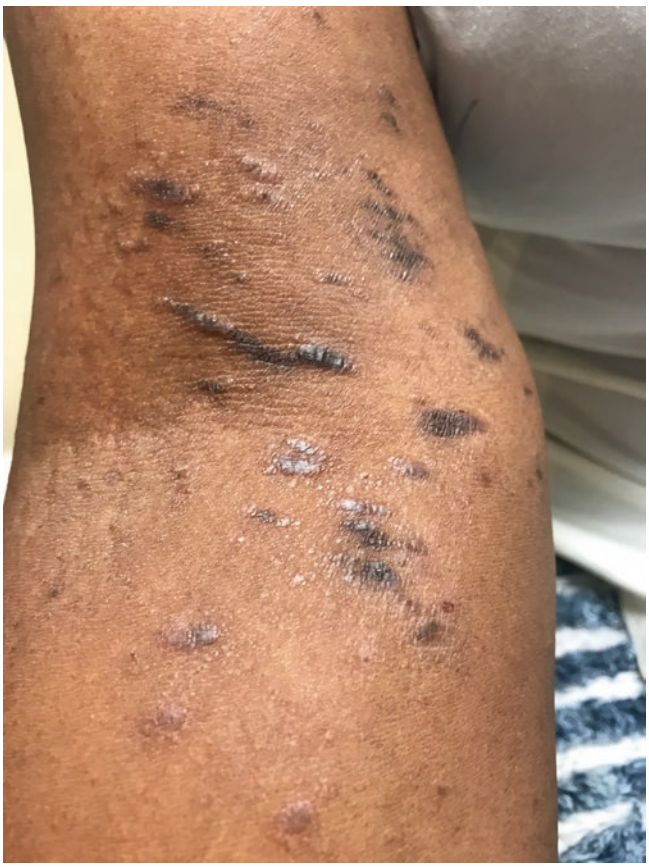

Fig. 2.105 Lichen planus

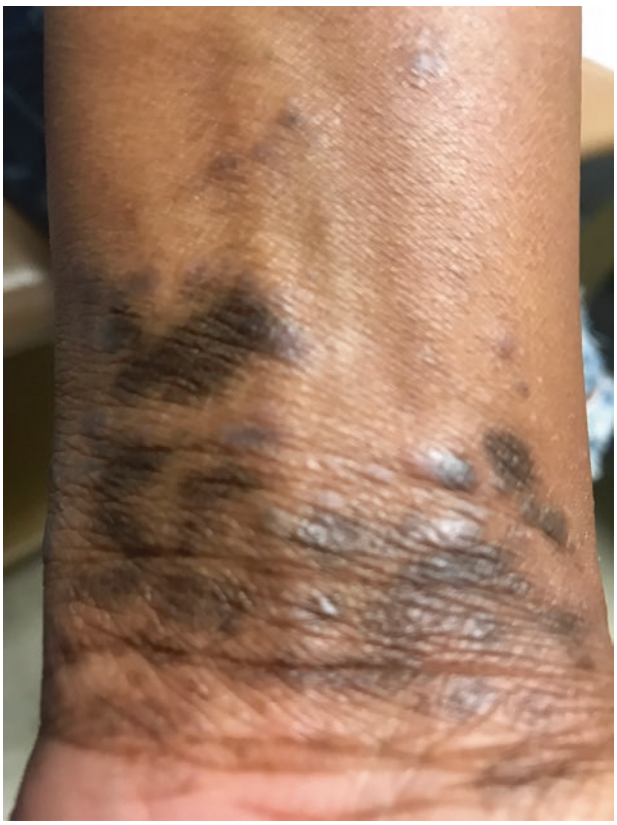

Fig. 2.107 Lichen planus

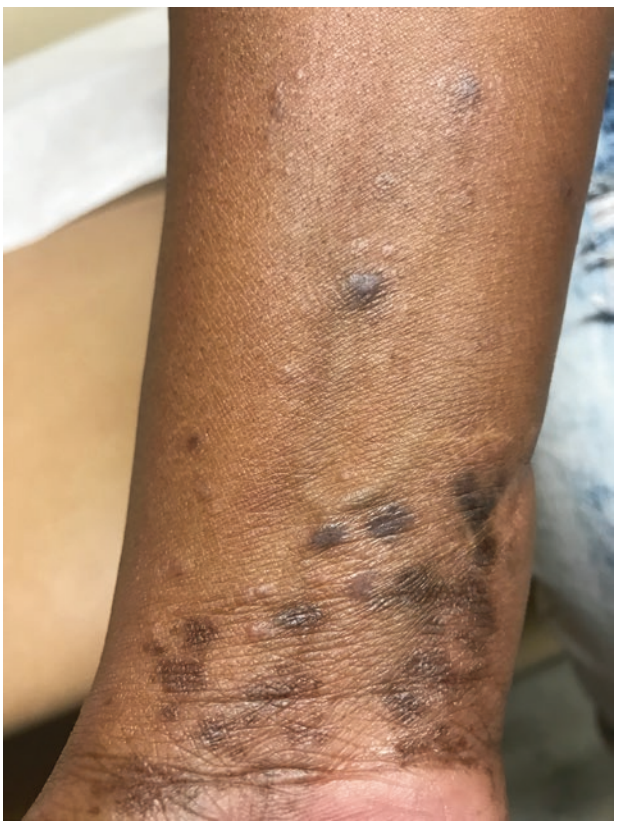

Fig. 2.106 Lichen planus

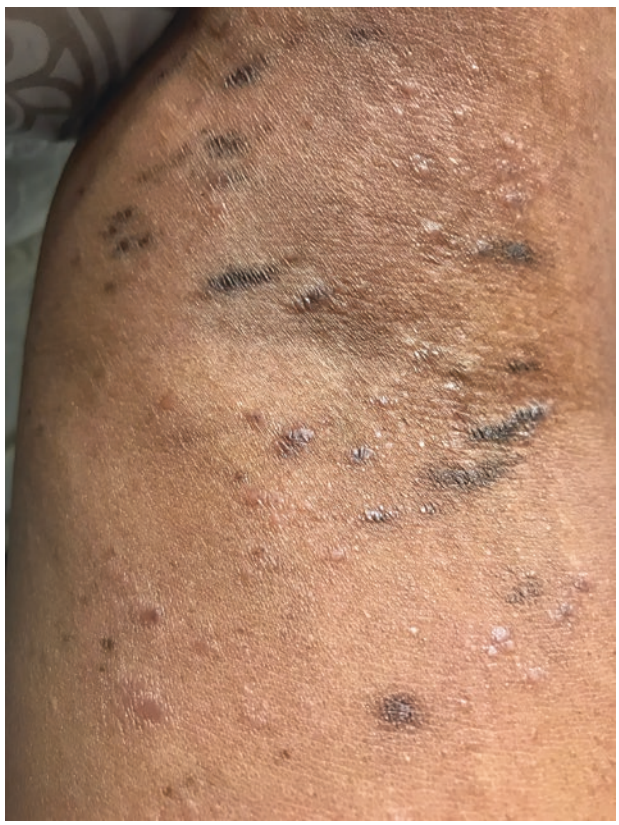

Fig. 2.108 Lichen planus

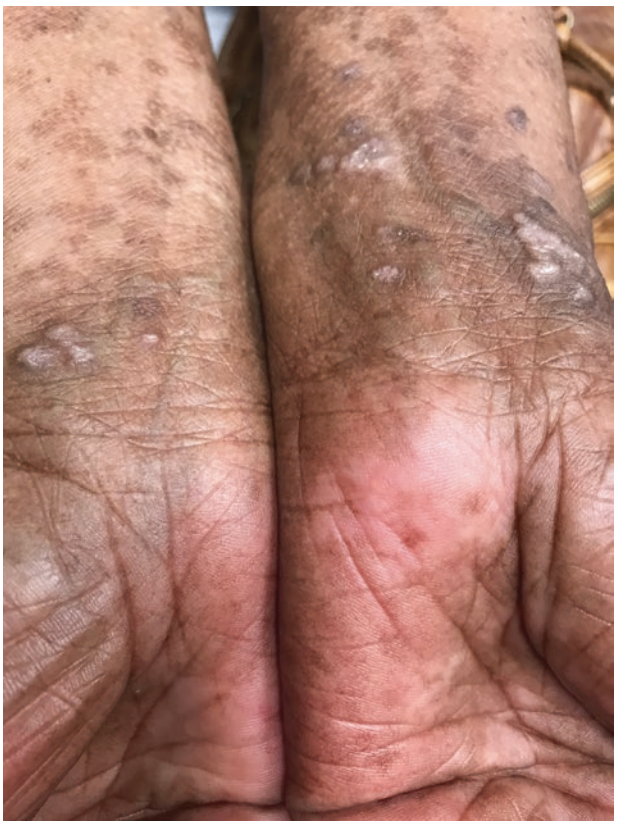

Fig. 2.109 Lichen planus

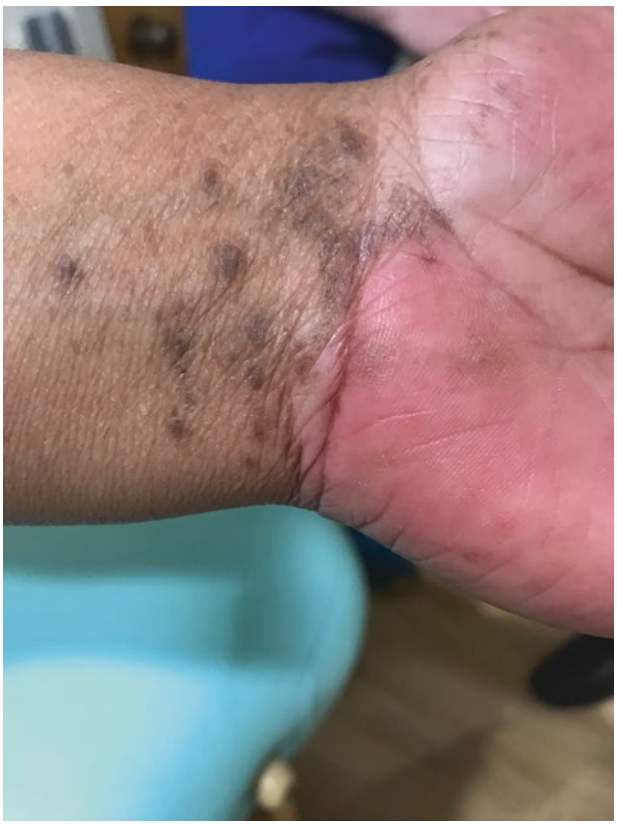

Fig. 2.111 Lichen planus

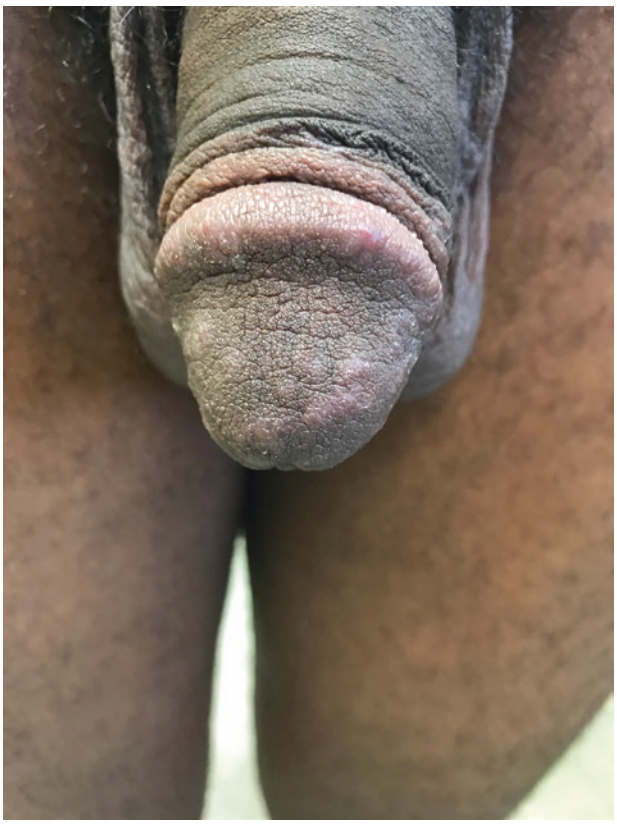

Fig. 2.110 Lichen planus

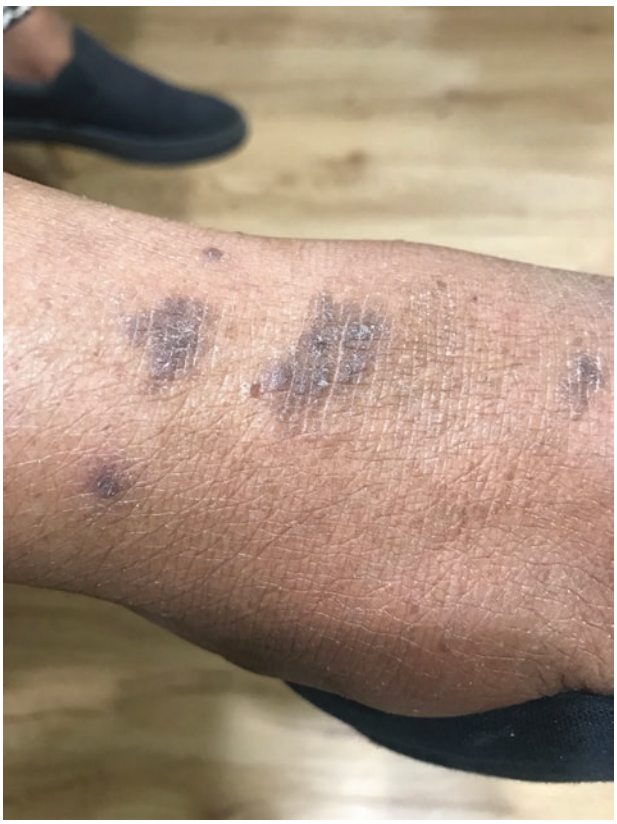

Fig. 2.112 Lichen planus

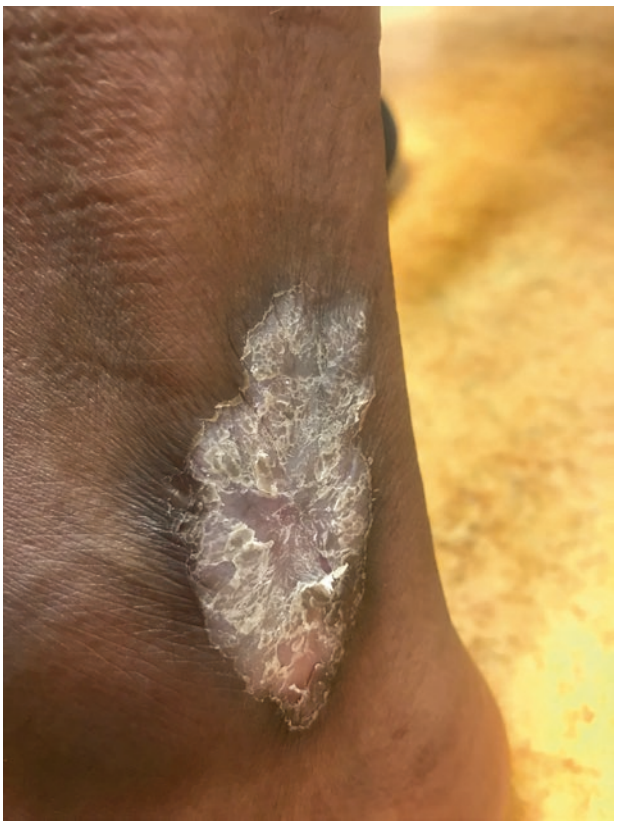

Fig. 2.113 Hypertrophic lichen planus

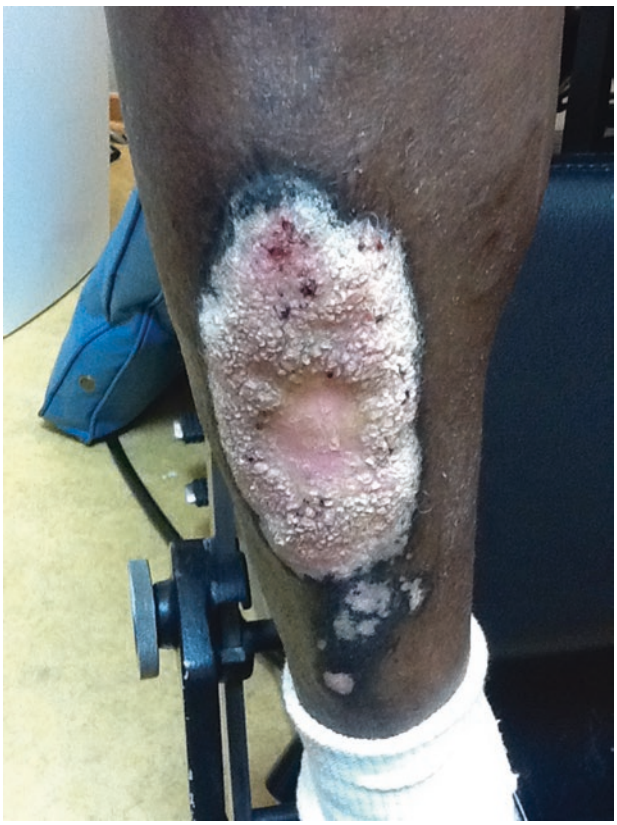

Fig. 2.114 Hypertrophic lichen planus

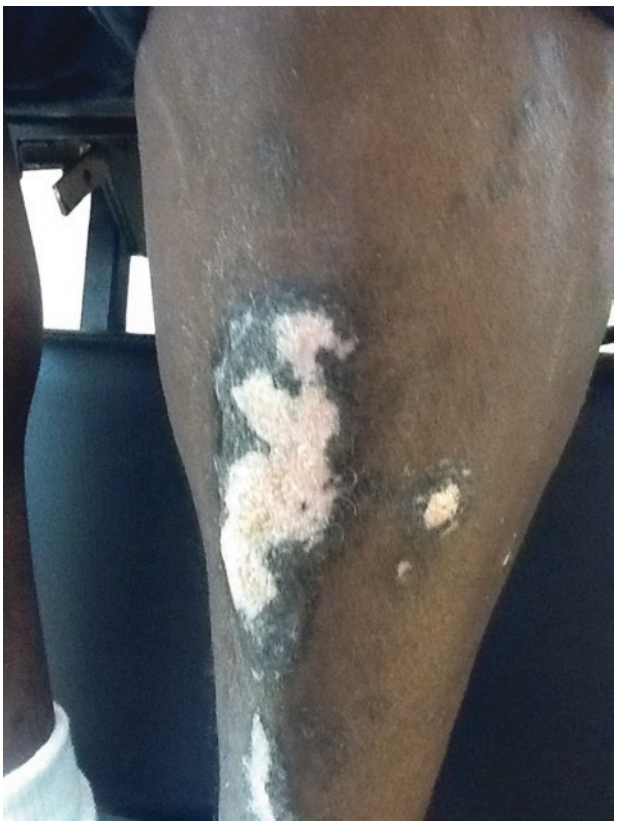

Fig. 2.115 Hypertrophic lichen planus after treatment with oral and intralesional steroids

2.4 Punctate Keratoderma

This rare, autosomal dominantly inherited condition was first described by Rasmussen in 1980. It is characterized by round depressions along the creases of the palms, fingers, and soles, which are filled with conical keratinous plugs. It has been seen in association with atopic dermatitis or manual labor and occurs most frequently in patients of African descent. Friction can worsen the condition, and pain is occasionally reported, but generally the condition is asymptomatic. The etiology of punctate keratoderma of the palmar creases (PKPC) is currently unknown.

2.4.1 Presentation in Black Skin

As noted above, PKPC is seen almost exclusively in patients of African descent, although it has been reported in Caucasian patients. The punctate lesions of PKPC are often small (<3 mm) and hyperpigmented. Focal acral hyperkeratosis (FAH), characterized by similar lesions along the lines of transgredience on the palms and soles, is also inherited in an autosomal dominant fashion, occurs in patients of African descent, and has been reported to occur concurrently with PKPC in at least two patients. It has been suggested that FAH

and PKPC may represent different presentations of the same underlying (as yet unknown) pathophysiologic process (Figs. 2.116–2.141).

2.4.2 Management

Topical retinoids, keratolytic agents, and punch excisions have all been reported as treatment options for PKPC. The efficacy of these treatments is variable, and recurrence is common.

Fig. 2.116 Punctate keratoderma

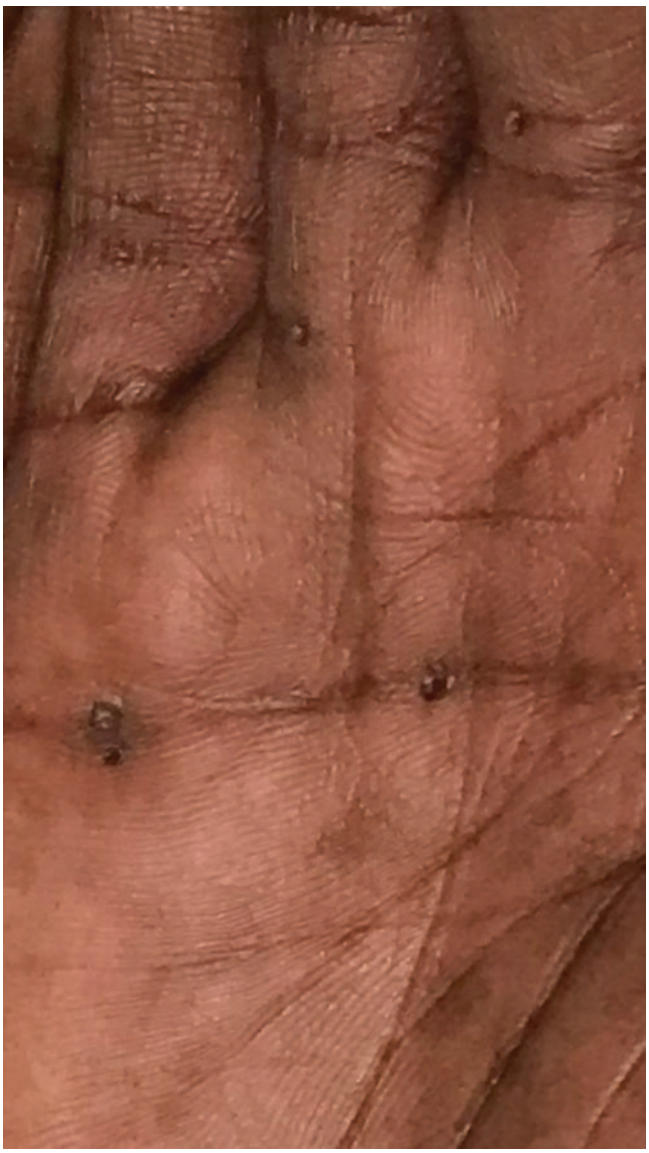

Fig. 2.117 Punctate keratoderma

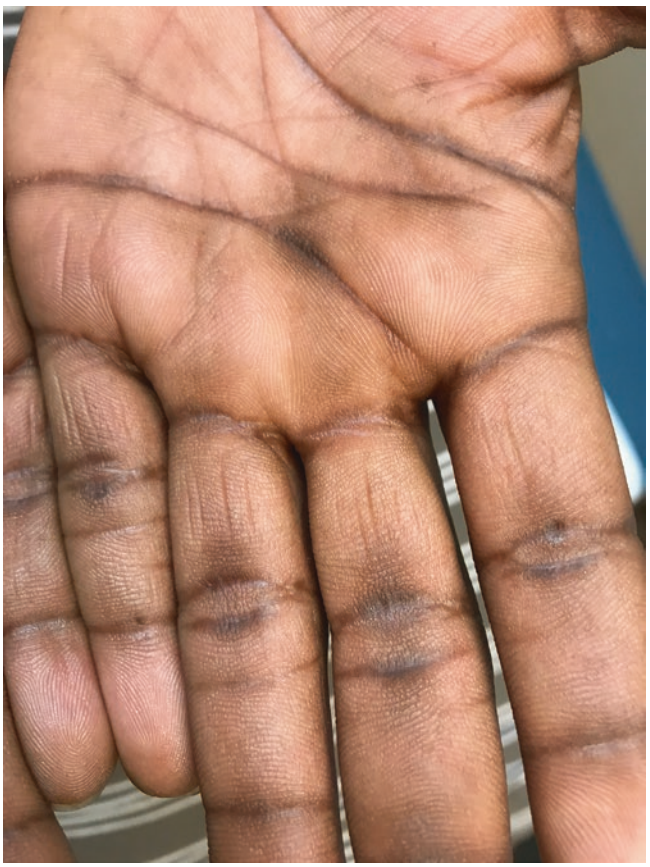

Fig. 2.118 Punctate keratoderma

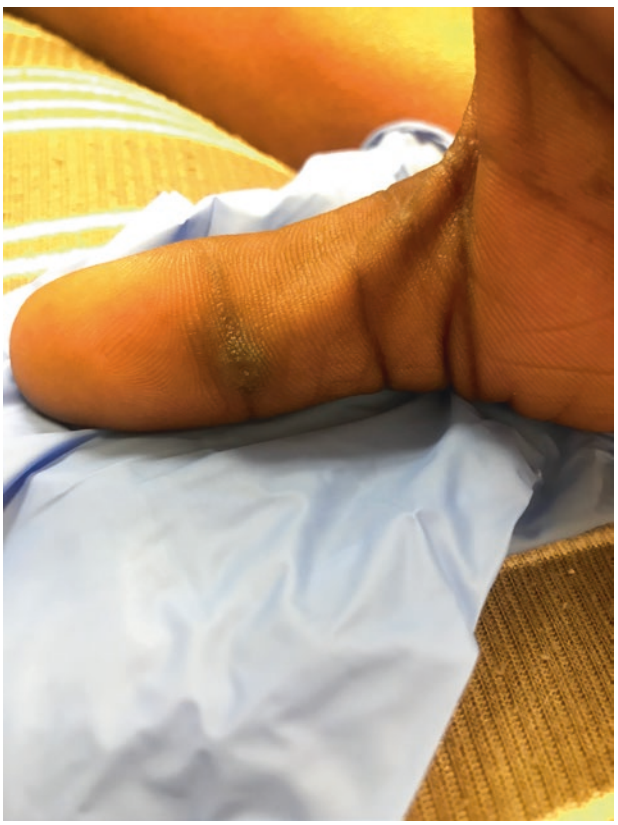

Fig. 2.119 Punctate keratoderma

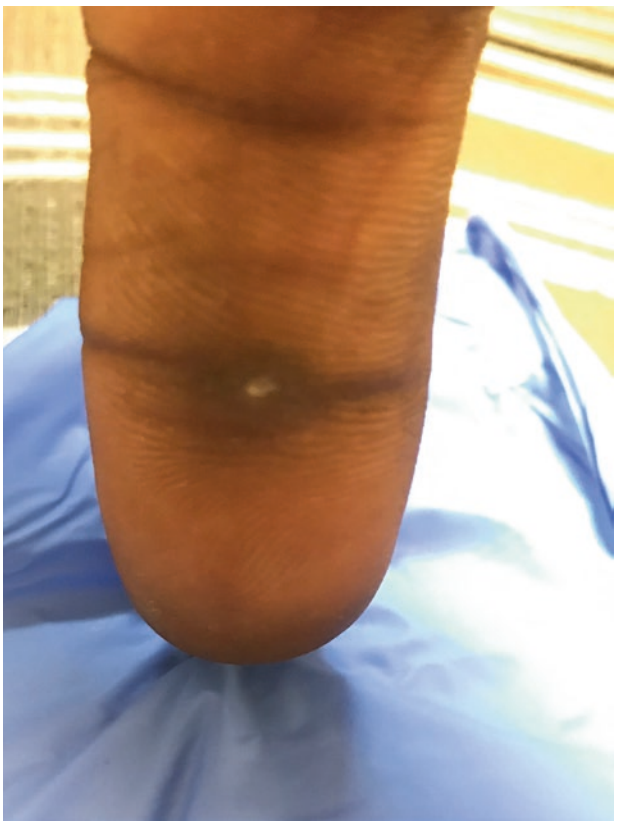

Fig. 2.120 Punctate keratoderma

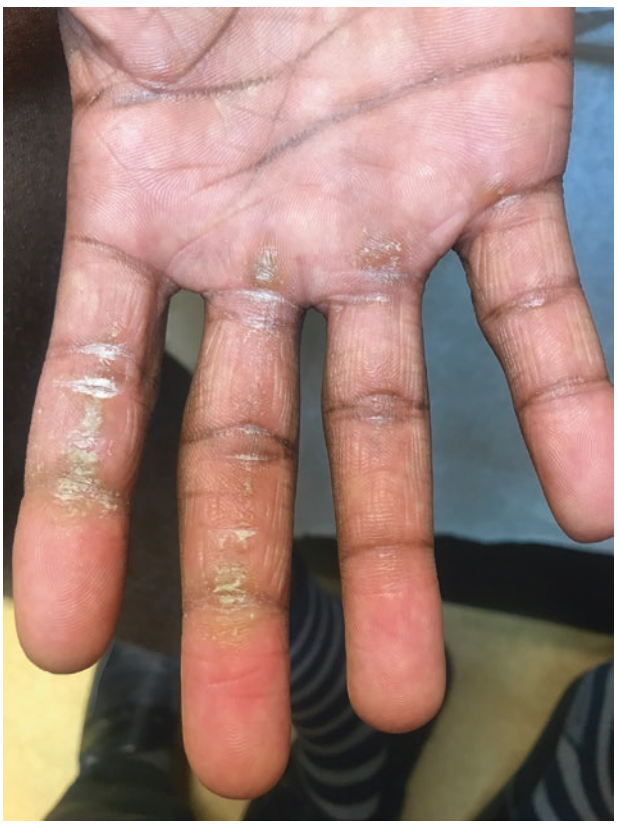

Fig. 2.121 Punctate keratoderma

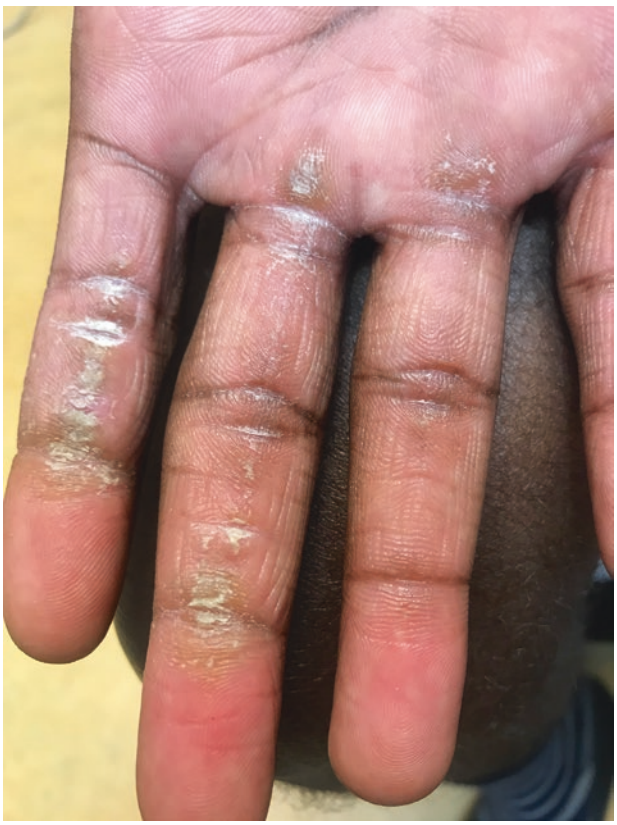

Fig. 2.122 Punctate keratoderma

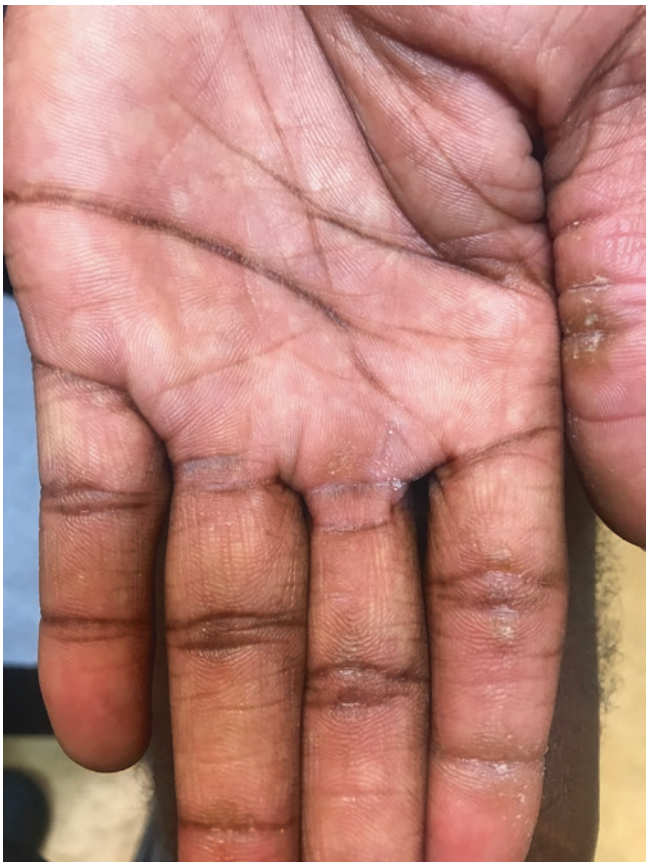

Fig. 2.123 Punctate keratoderma

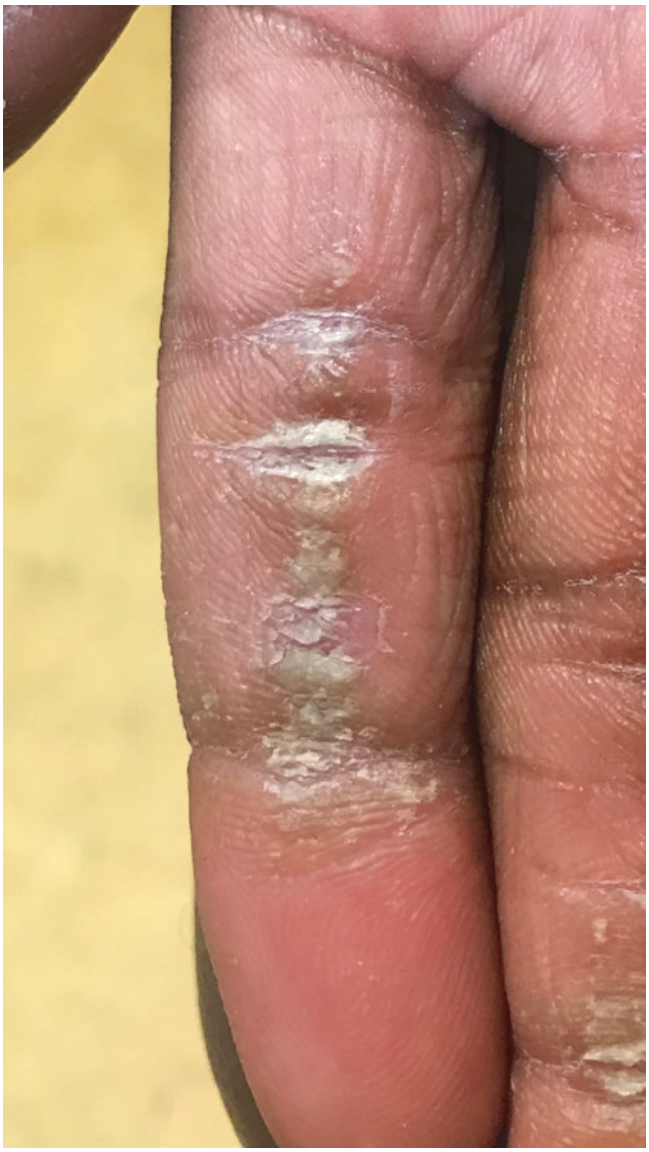

Fig. 2.124 Punctate keratoderma, close-up

Fig. 2.125 Punctate keratoderma

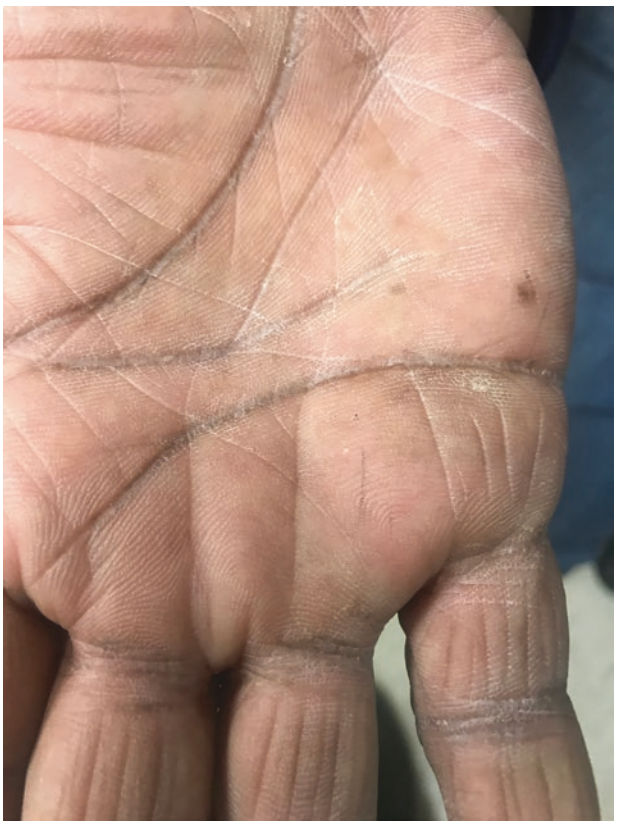

Fig. 2.126 Punctate keratoderma

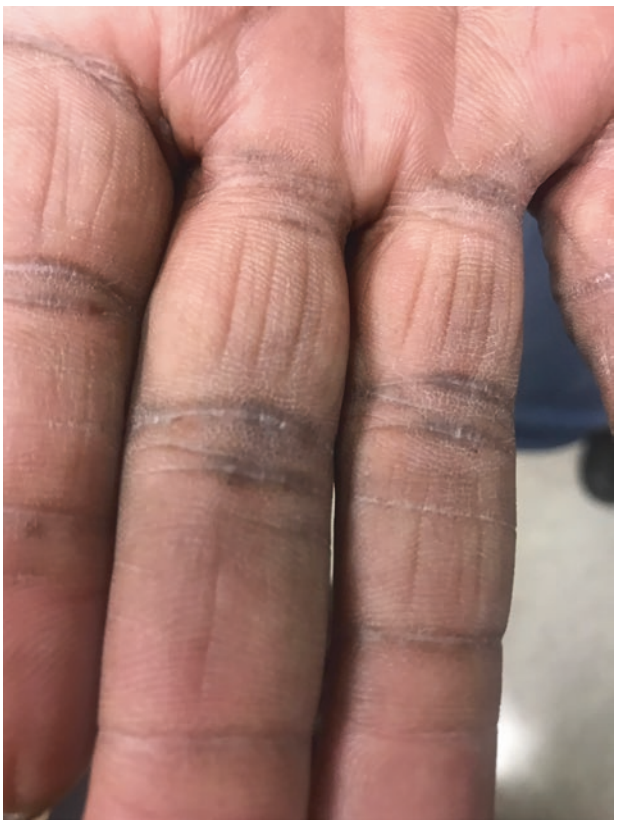

Fig. 2.127 Punctate keratoderma

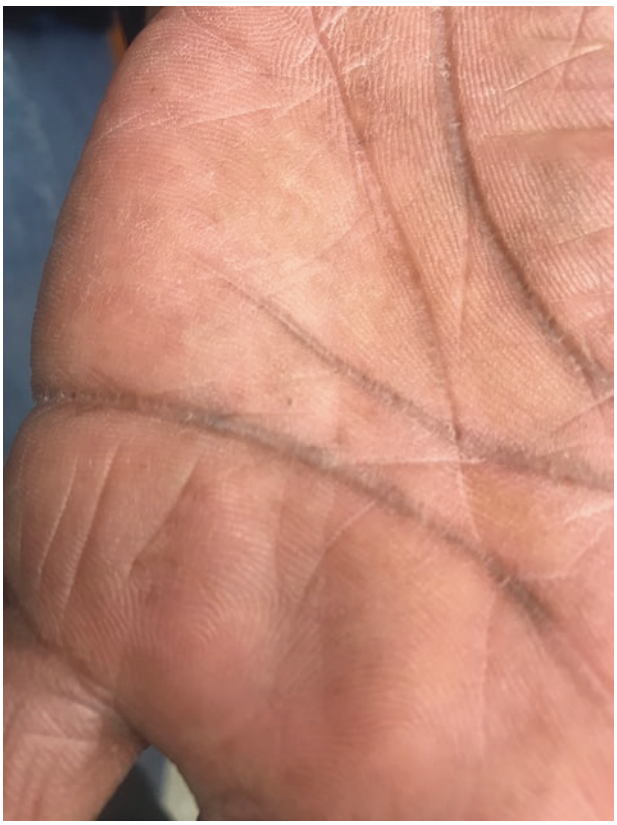

Fig. 2.128 Punctate keratoderma

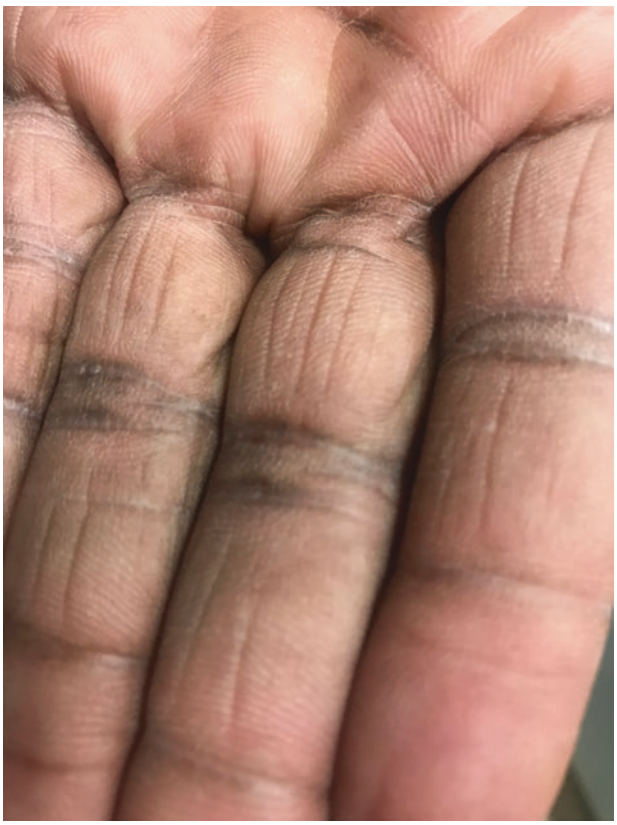

Fig. 2.129 Punctate keratoderma

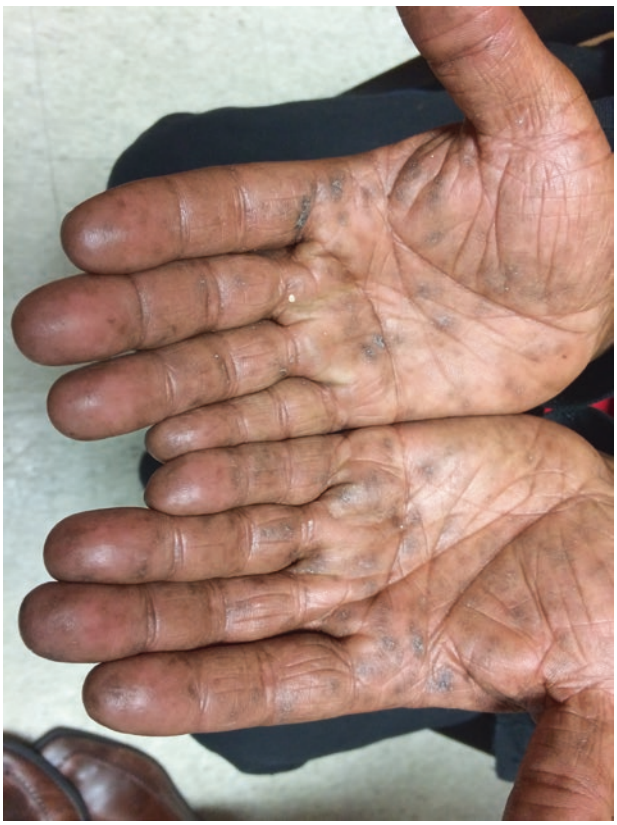

Fig. 2.131 Punctate keratoderma

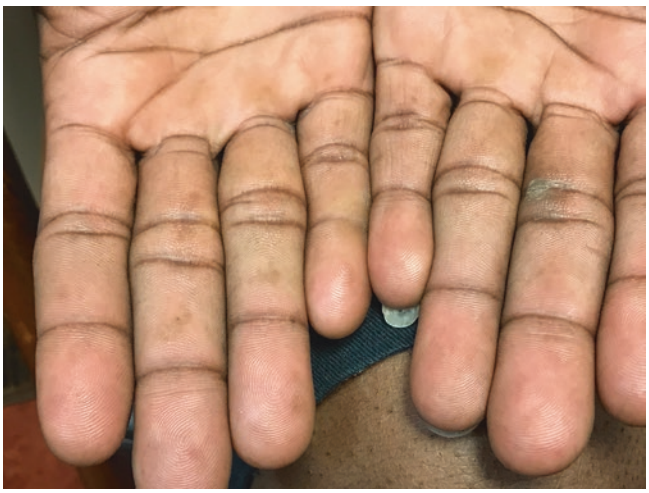

Fig. 2.130 Punctate keratoderma

Fig. 2.132 Punctate keratoderma

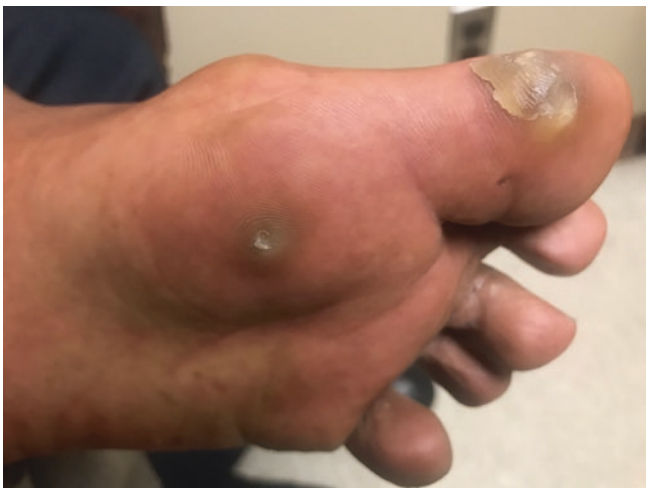

Fig. 2.133 Punctate keratoderma

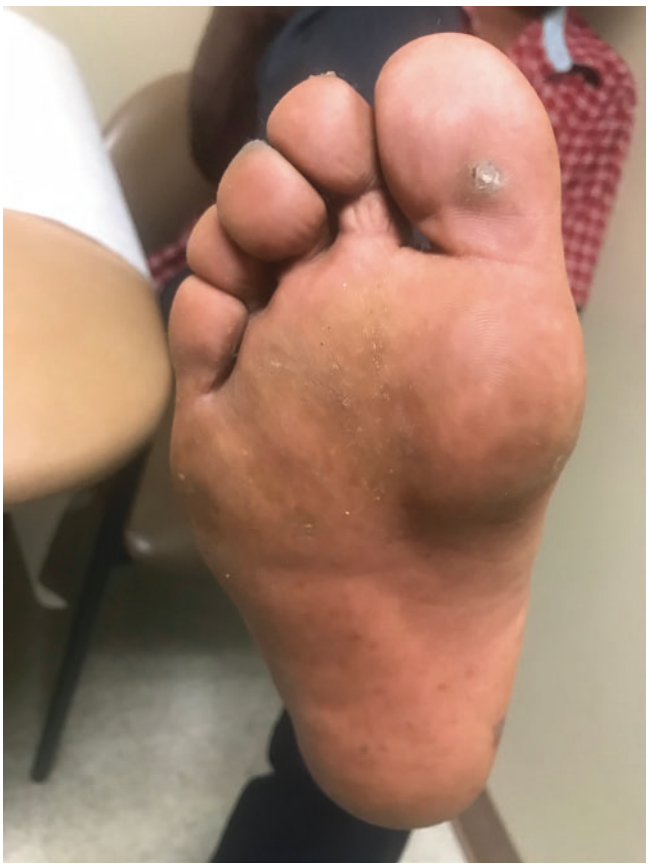

Fig. 2.134 Punctate keratoderma

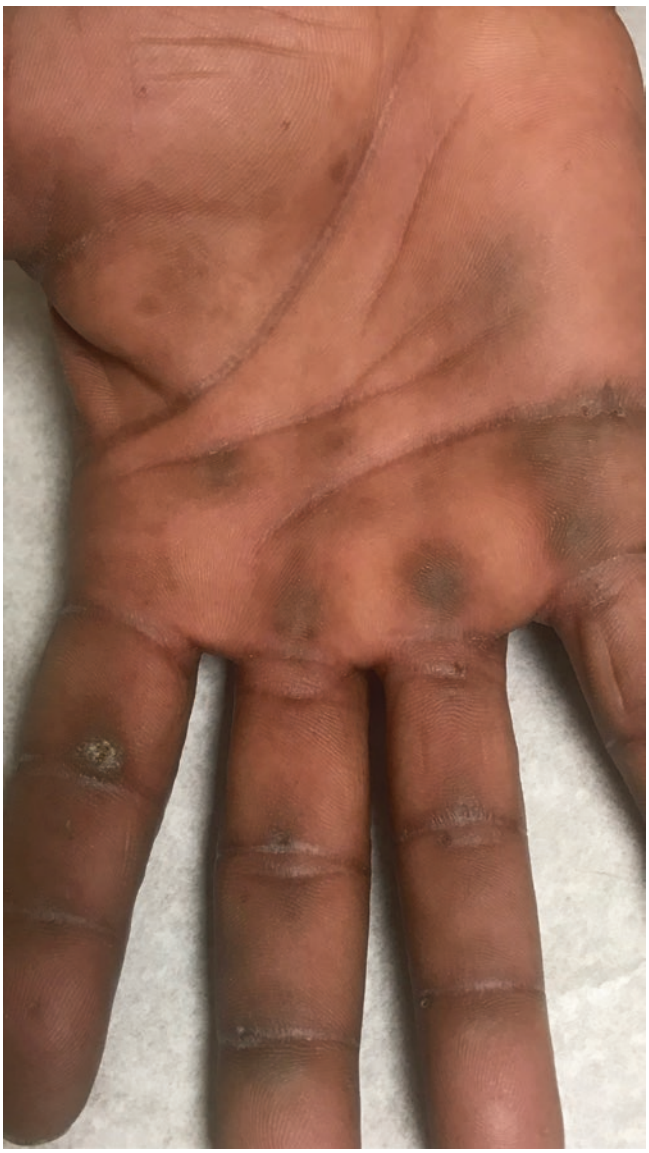

Fig. 2.135 Punctate keratoderma

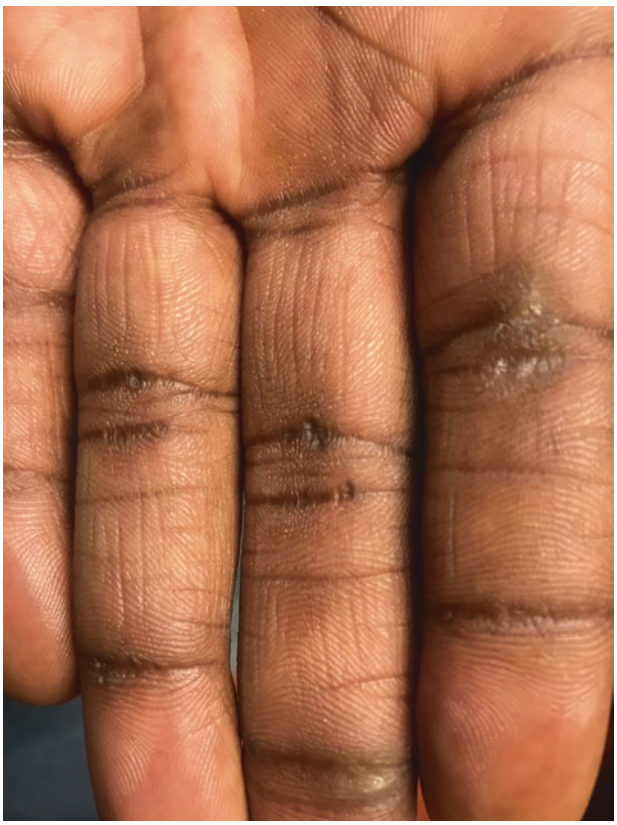

Fig. 2.136 Punctate keratoderma

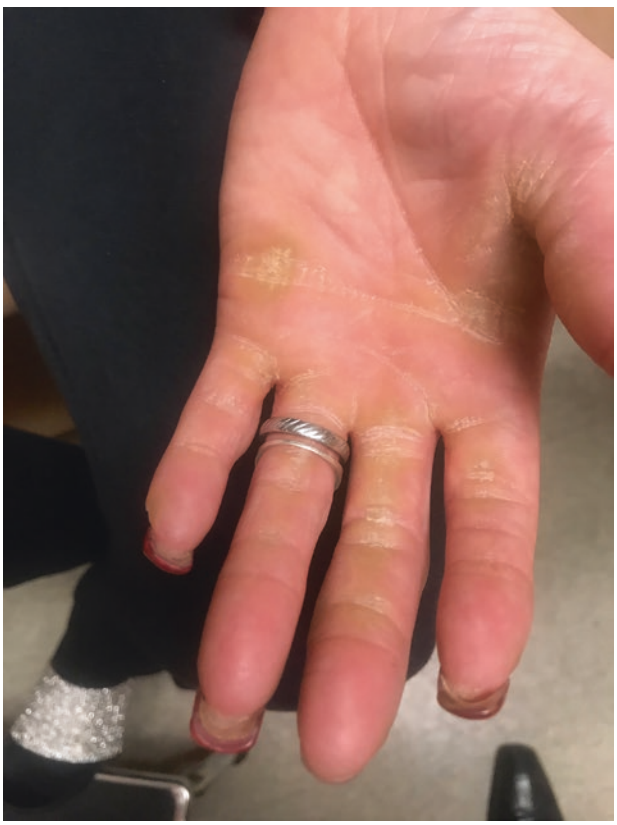

Fig. 2.137 Punctate keratoderma

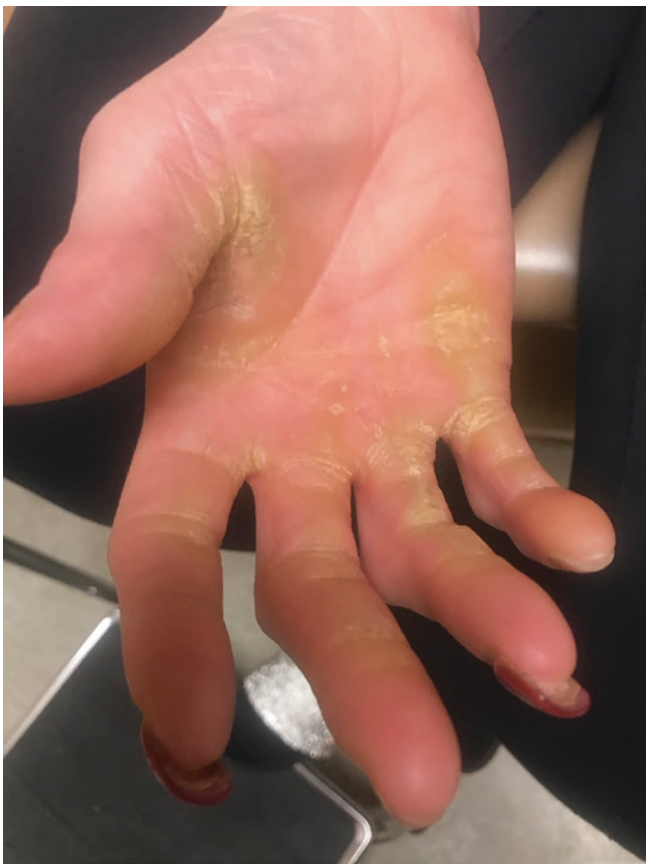

Fig. 2.138 Punctate keratoderma

Fig. 2.139 Punctate keratoderma

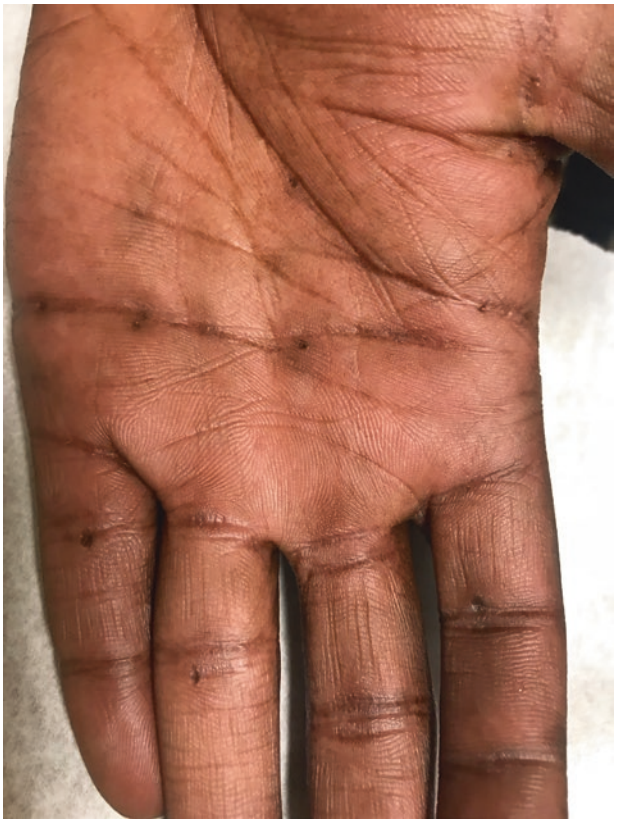

Fig. 2.140 Punctate keratoderma

Fig. 2.141 Punctate keratoderma

2.5 Adnexal Diseases

This section describes adnexal diseases, which arise because of disruptions in the function of eccrine, apocrine, or sebaceous glands. Some of these diseases are more common in skin of color because of unique cultural practices in this population. Other diseases have unique clinical presentations in skin of color, or have a higher prevalence in patients of color for reasons related to the underlying pathophysiology of disease. This section highlights the presentation of adnexal diseases in the context of skin of color, to help the practitioner to recognize the unique features of these conditions in darker skin types.

2.5.1 Acne Vulgaris

The development of acne vulgaris is a complex process that involves underlying genetic factors, hormonal levels, bacterial colonization of the hair follicle, and environmental exposures. The classic lesion of acne develops as the result of abnormal occlusion of the hair follicle, resulting in the formation of a comedo, a cyst-like structure within the hair follicle that becomes filled with shed keratinocytes and sebum. Inflammation can then develop around the comedo, leading to the formation of the erythematous papules and pustules that characterize inflammatory acne. Though the incidence of acne is highest during adolescence, 12% of women and 3% of men will experience acne that persists into adulthood.

2.5.1.1 Presentation in Black Skin

A large study based in the United States showed that among women, acne was more common in those of African descent and Hispanics than in Caucasians or Asians (Figs. 2.142–2.161). Among men, Caucasians are more likely to have severe, nodulocystic variants of acne than black patients. Post-inflammatory hyperpigmentation (PIH) is more common in patients of color, as is the development of keloids in areas of acne scarring.

2.5.1.2 Management

Early and aggressive treatment of acne vulgaris in patients of color is key to preventing and minimizing the development of PIH and scarring. The general treatment approach for acne is similar for all skin types, but for darker skin types, preference may be given to treatment options that can treat PIH and active acne concurrently. These include topical retinoids and azaleic acid. Other first-line therapies include topical antibiotics, topical dapsone, and benzoyl peroxide washes and lotions. Oral antibiotics and oral retinoids should be reserved for disease resistant to topical therapy, or cases where extensive acne scarring is already present.

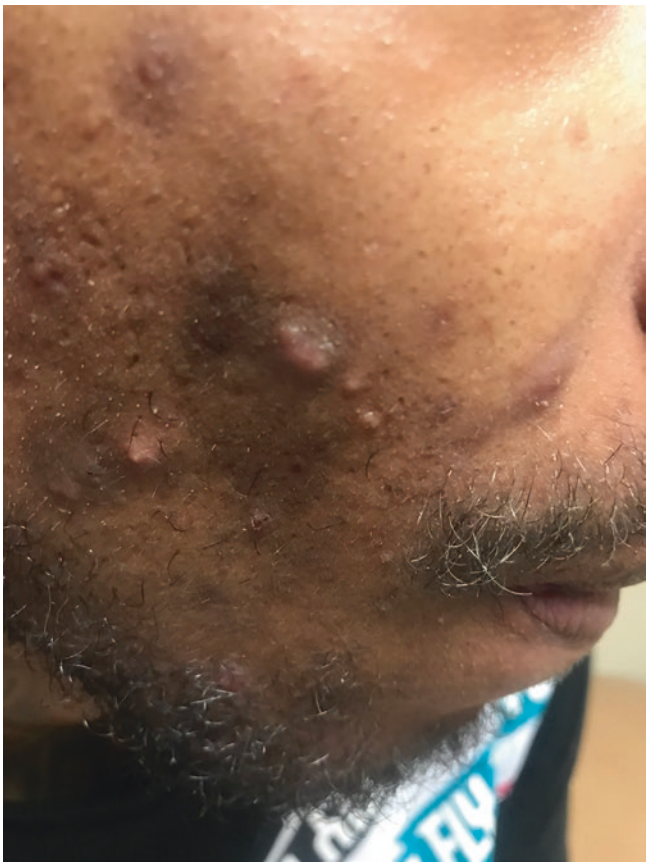

Fig. 2.142 Acne vulgaris

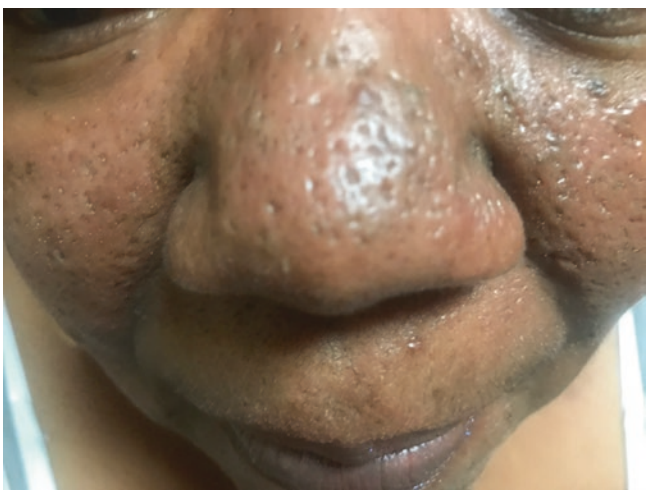

Fig. 2.143 Acne vulgaris

2.5.2 Pomade Acne

Pomade acne represents a special case of acne that develops as a result of application of emollients and oils to the scalp. This condition is seen primarily in patients of African descent (and less commonly in Hispanics), who may apply pomades

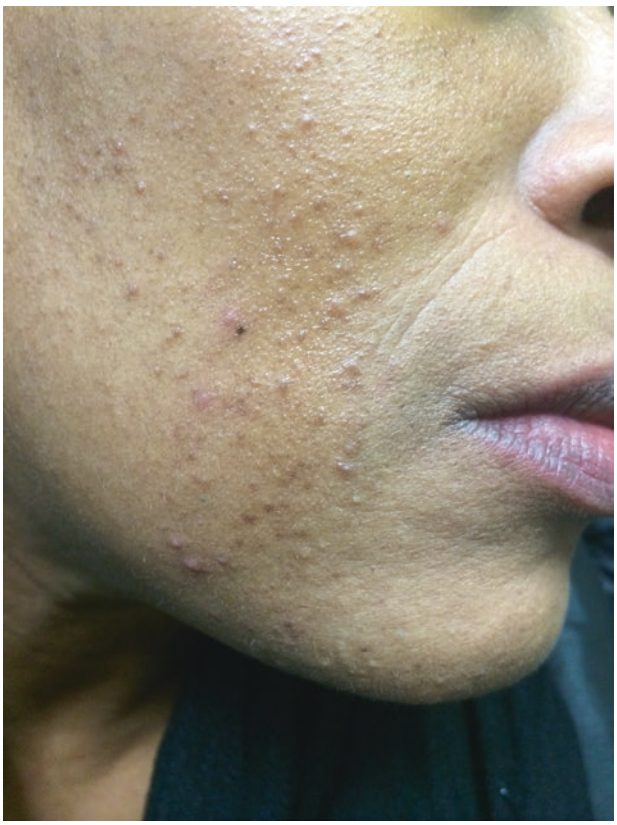

Fig. 2.144 Acne vulgaris

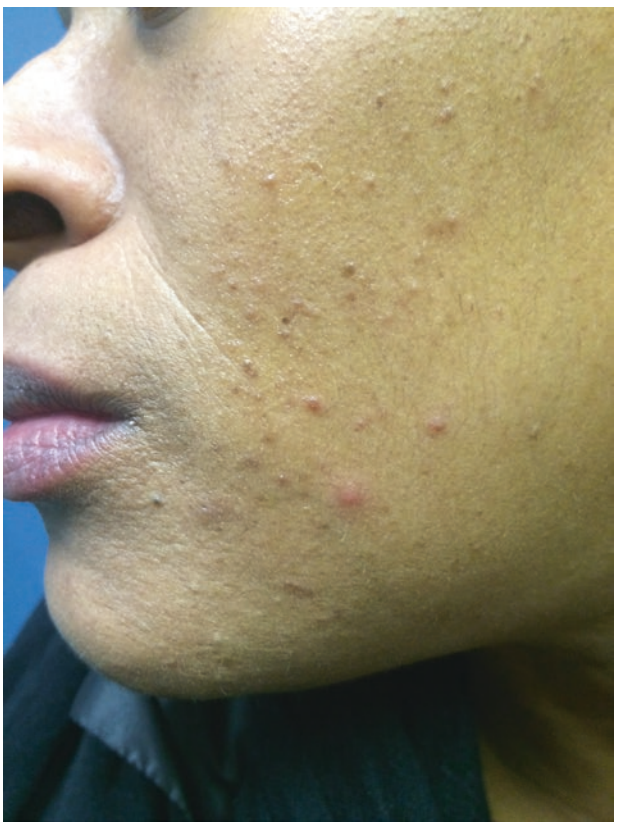

Fig. 2.145 Acne vulgaris

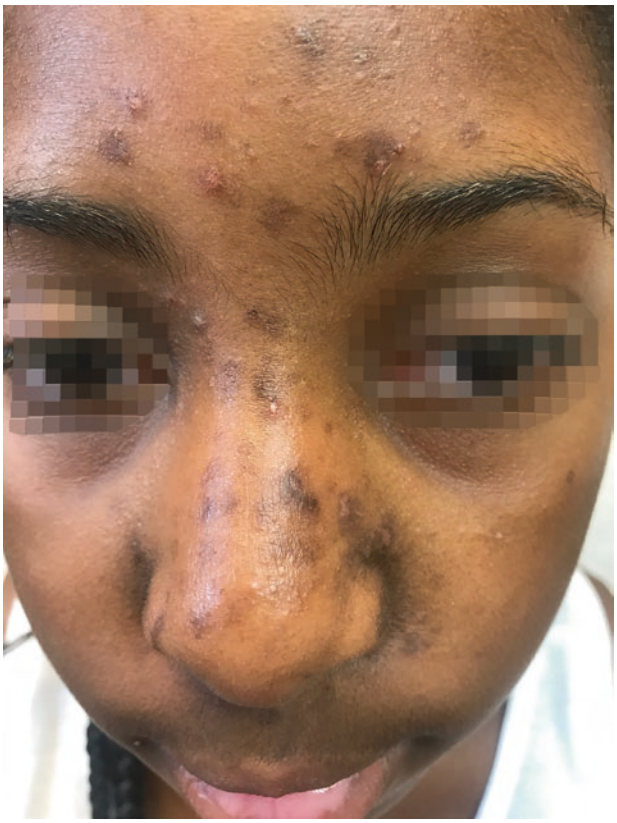

Fig. 2.146 Acne vulgaris

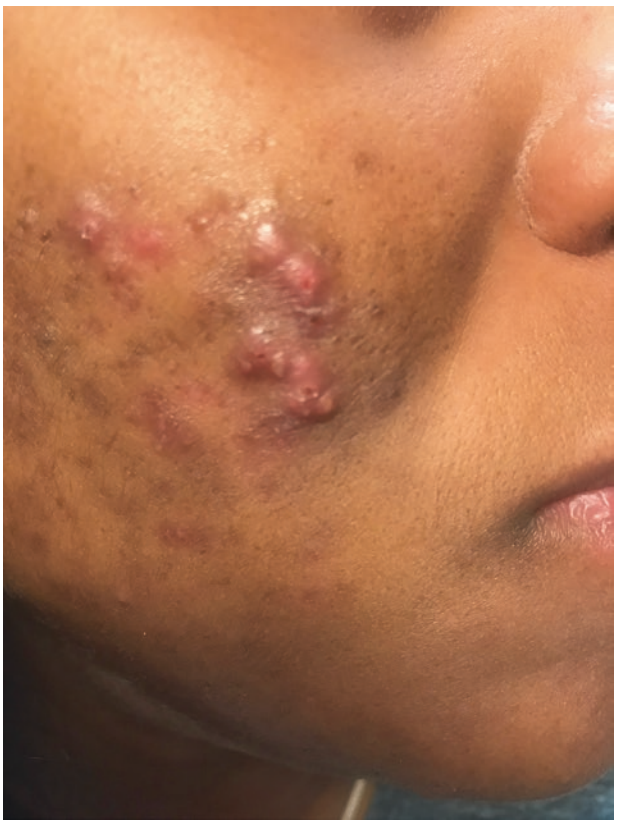

Fig. 2.147 Acne vulgaris

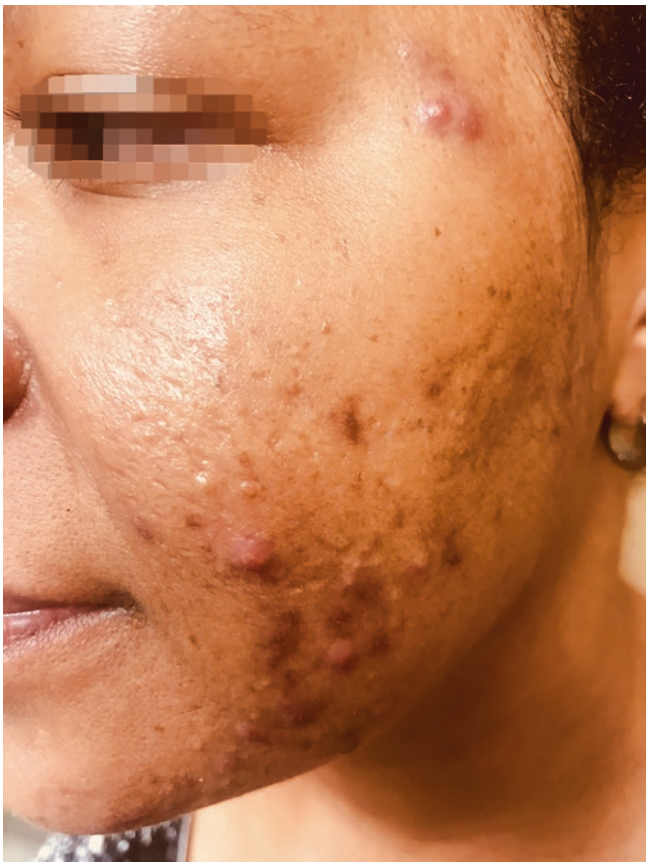

Fig. 2.148 Acne vulgaris

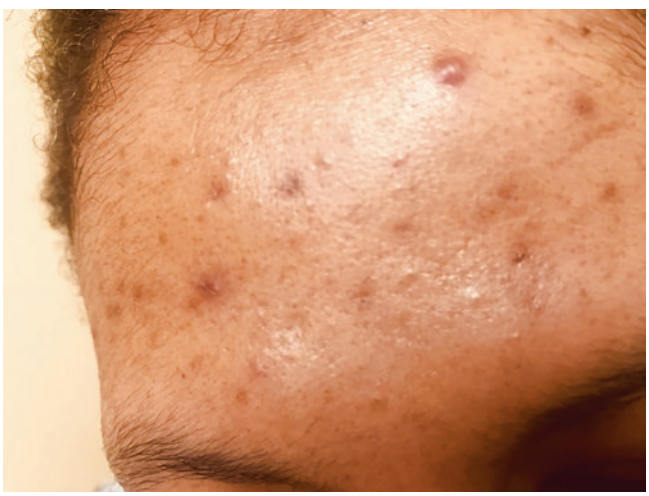

Fig. 2.149 Acne vulgaris

and oils to moisturize the hair and scalp or to achieve specific hairstyles. Disease pathogenesis is thought be due to occlusion of the follicular unit by hair pomades or oils, resulting in the development of comedones and inflammatory papules. It has also been reported in patients who wear "wave caps" or other tightly fitting headwear that leads to occlusion of the skin around the hairline.

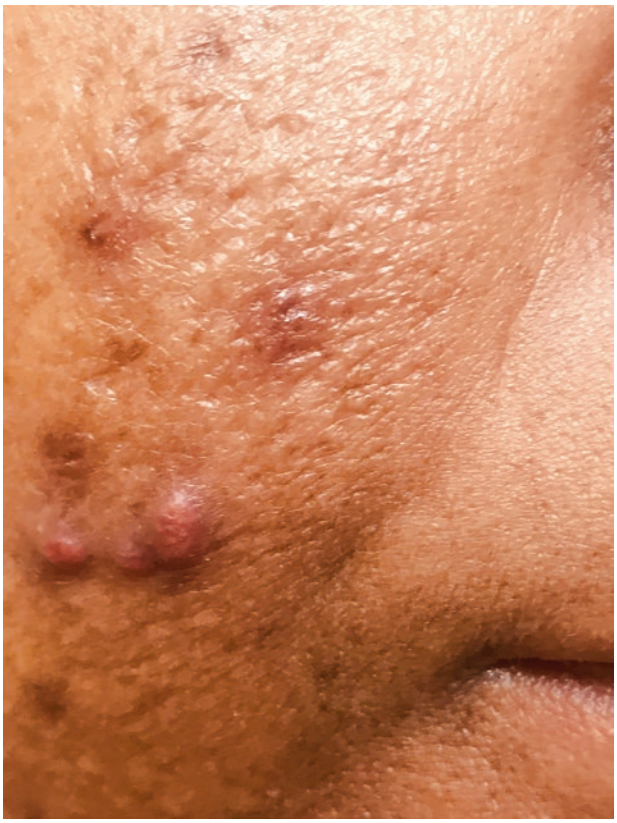

Fig. 2.150 Acne vulgaris

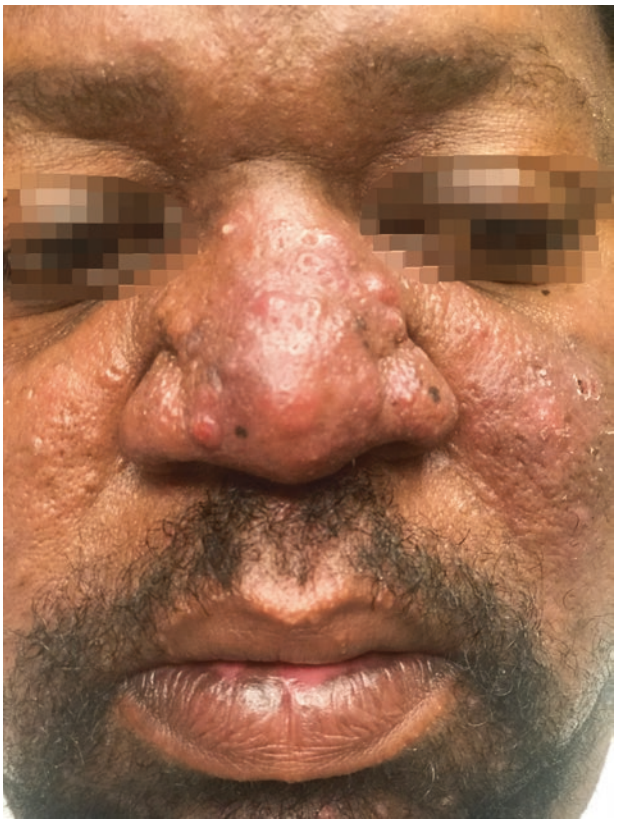

Fig. 2.152 Acne vulgaris with keloid formation

Fig. 2.151 Acne vulgaris

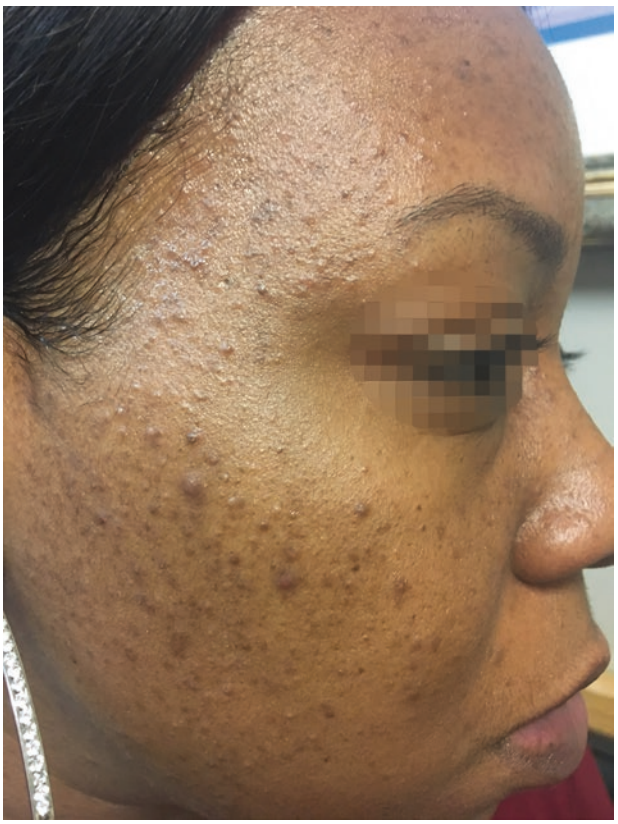

Fig. 2.153 Acne vulgaris

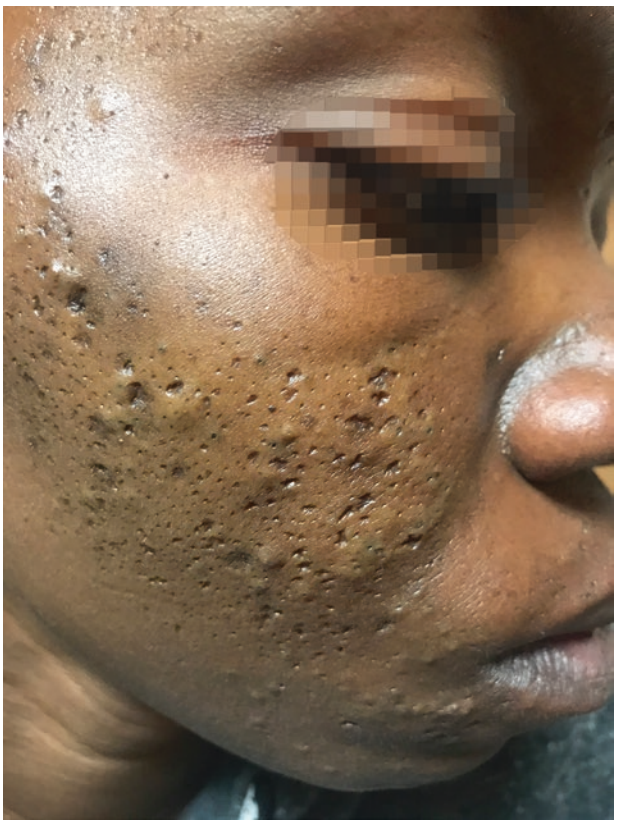

Fig. 2.154 Acne vulgaris

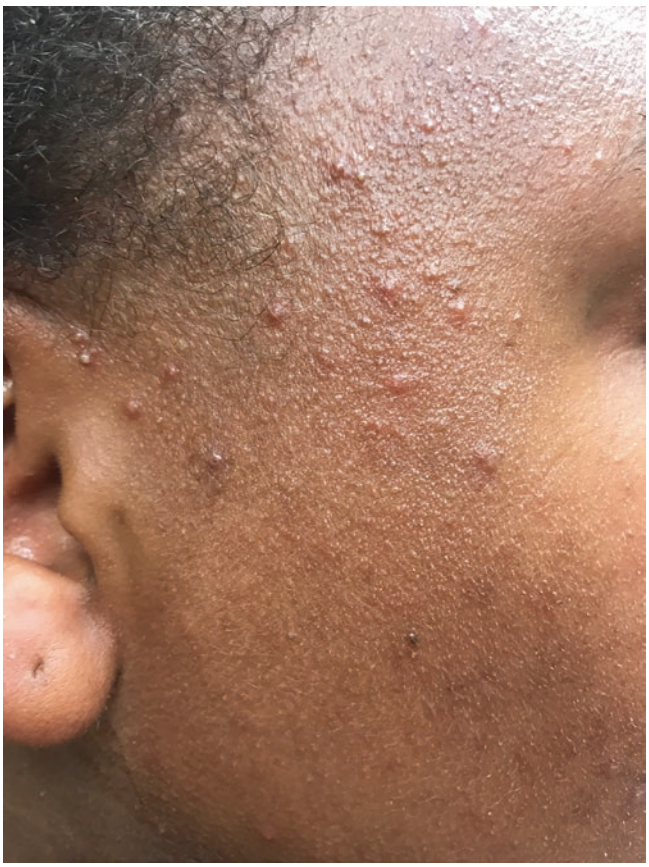

Fig. 2.156 Acne vulgaris

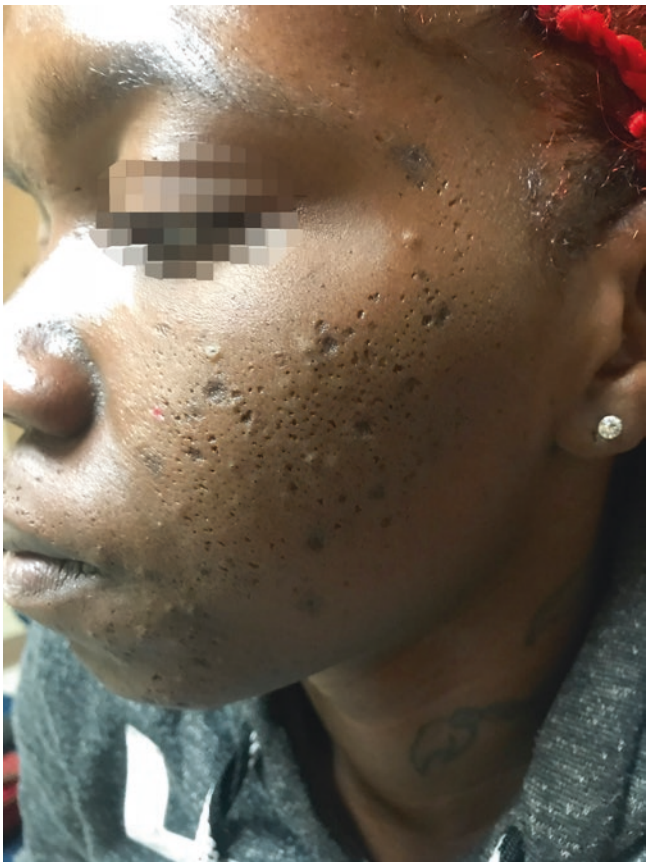

Fig. 2.155 Acne vulgaris

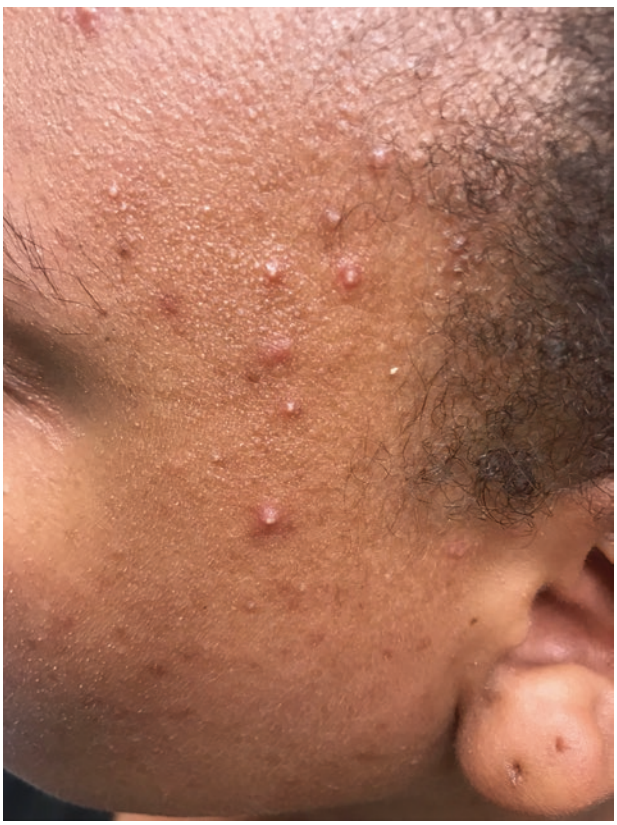

Fig. 2.157 Acne vulgaris

Fig. 2.158 Acne vulgaris

Fig. 2.159 Acne vulgaris

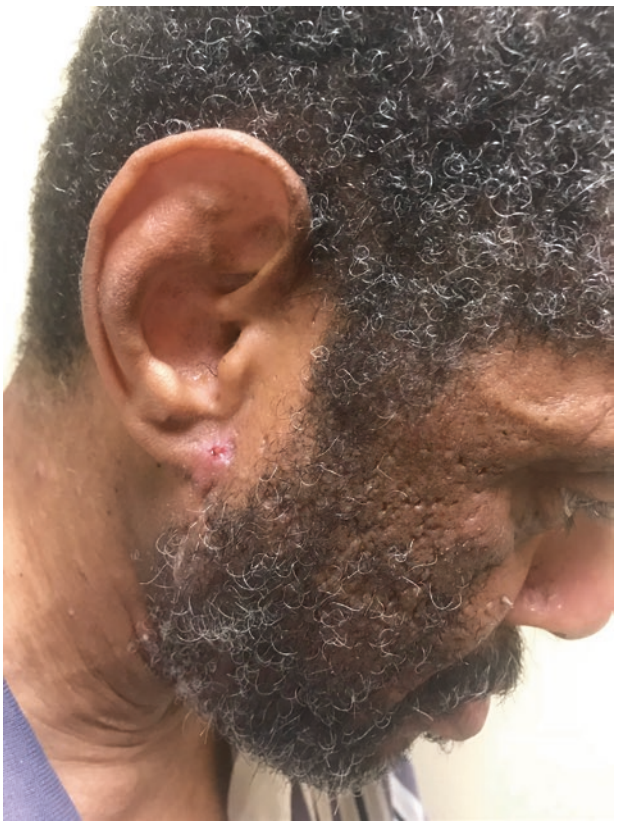

Fig. 2.160 Acne vulgaris

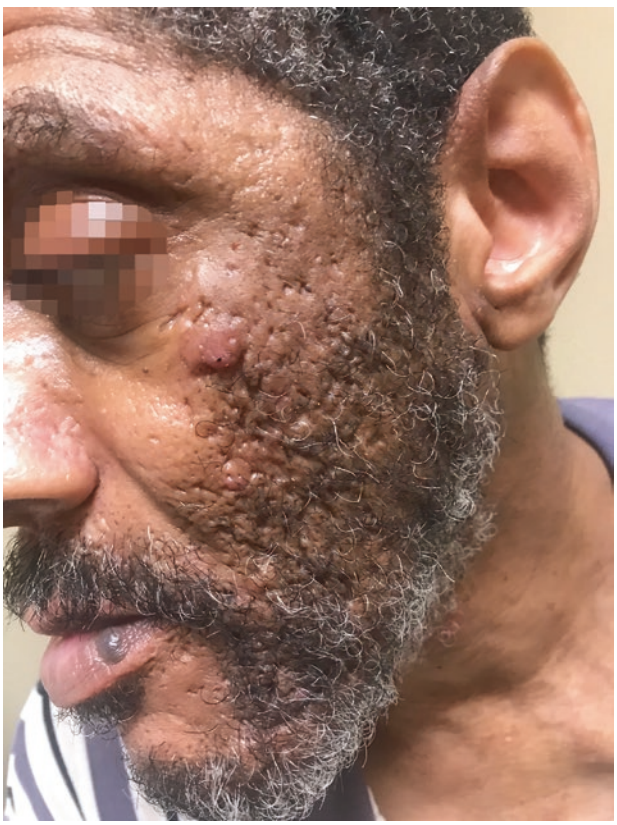

Fig. 2.161 Acne vulgaris

2.5.2.1 Presentation in Black Skin

Presenting as closely grouped, often monomorphic papules or closed comedones on the upper forehead and hairline, pomade acne also can be seen on the cheeks and chin. Inflamed lesions of pomade acne may present with bright red erythema in lighter skin types or may appear brown or purple-brown in darker skin types. Post-inflammatory hyperpigmentation (PIH) is common (Figs. 2.162 and 2.163).

2.5.2.2 Management

Treatment of pomade acne consists of discontinuing the use of occlusive hair pomades, oils, and/or headwear. Topical antibiotics and topical retinoids can also be used if avoidance of hair pomades does not resolve the symptoms.

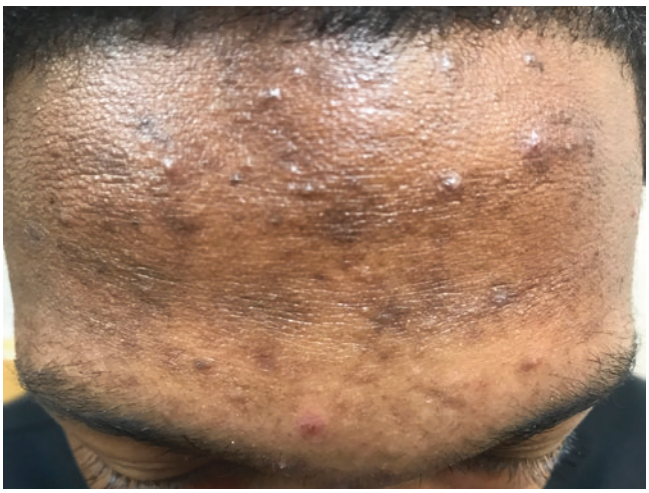

Fig. 2.162 Pomade acne

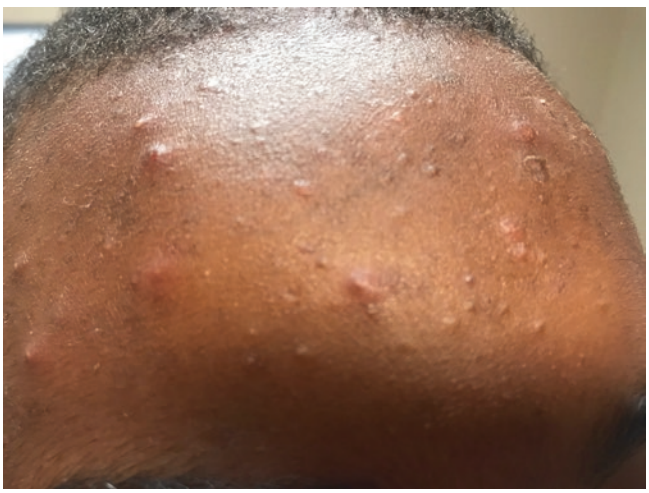

Fig. 2.163 Pomade acne

2.5.3 Rosacea

Rosacea, an inflammatory disease of the pilosebaceous unit (hair follicle), is most prevalent in lighter skin types. It peaks in incidence during the 3rd and 4th decades of life. Vascular hyperreactivity is thought to be the primary mechanism through which rosacea develops, but follicular colonization with the *Demodex folliculorum* mite has also been implicated. The four major subtypes of rosacea—erythematotelangiectatic, ocular, papulopustular, and phymatous—each have a unique clinical presentation.

2.5.3.1 Presentation in Black Skin

In one study of patients in the United States with rosacea, 2% were black, 2.3% were Asian or Pacific Islander, and 3.9% were Hispanic, making this an uncommon condition in patients of color. However, the prevalence of rosacea in patients of color worldwide has been reported to be as high as 10% in some studies. The erythematotelangectatic subtype, which presents with diffuse macular erythema and telangectasia, may be difficult to recognize in darker skin, and therefore may be underdiagnosed in skin of color. Granulomatous rosacea, a minor subtype, has been reported to be more common in patients of color, along with the papulopustular subtype. As with other inflammatory skin conditions, the erythema of papulopustular rosacea and other rosacea subtypes may be masked by post-inflammatory hyperpigmentation and may present as brown or violet in color (Figs. 2.164–2.180).

2.5.3.2 Management

For rosacea variants that feature marked telangiectasia, flushing, and erythema, pulsed dye laser or intense pulsed light can be effective. For the papulopustular variant, treatment with topical metronidazole is the first-line approach, with topical azaleic acid, sodium sulfacetamide, and tretinoin also described in the literature. Severe or treatment-resistant rosacea can be treated with oral antibiotics or isotretinoin. Physical sunblock or sunscreen can be helpful in preventing UV-induced rosacea flares.

2.5.4 Hidradenitis Suppurativa

Hidradenitis suppurativa (HS) develops in areas of skin that bear apocrine glands, most frequently the axillae and anogenital areas, but also infra-mammary and infra-abdominal skin. The pathophysiology of HS is thought to be due to hyperkeratosis of affected hair follicles, leading to follicular occlusion and dilatation. Rupture of dilated follicles induces dermal inflammation, resulting in the clinical presentation of painful, inflammatory nodules and sterile abscesses. Over time, recurrent HS can lead to the development of sinus tracts

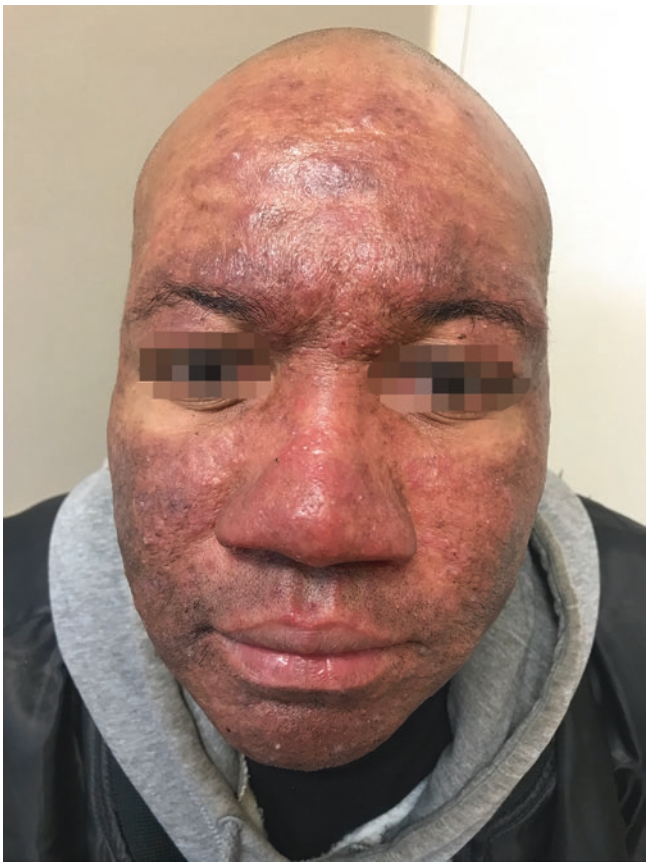

Fig. 2.164 Rosacea

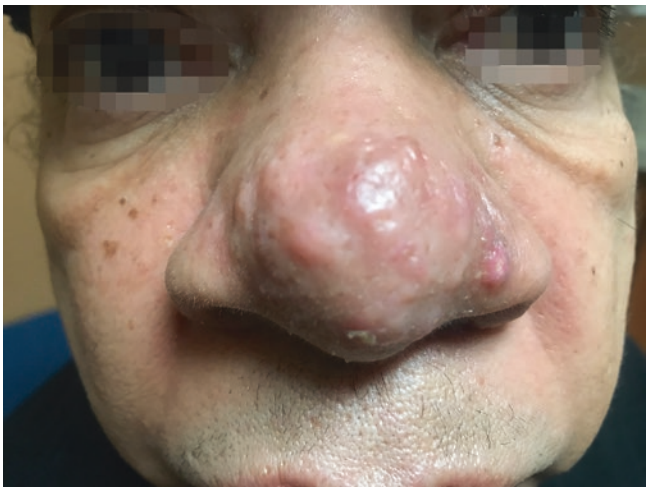

Fig. 2.165 Rosacea

and hypertrophic scarring. Major risk factors for HS include smoking, obesity, and history of metabolic syndrome.

2.5.4.1 Presentation in Black Skin

Hidradenitis suppurativa is more common in women and people of African descent. A recent single-site retrospective study of 375 HS patients in the US showed that

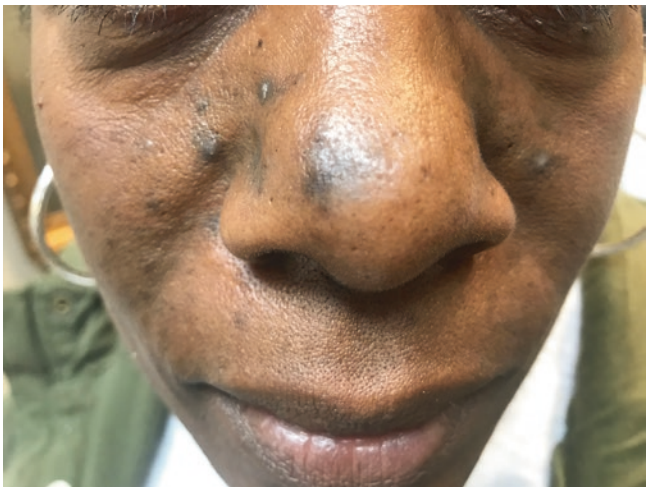

Fig. 2.166 Rosacea

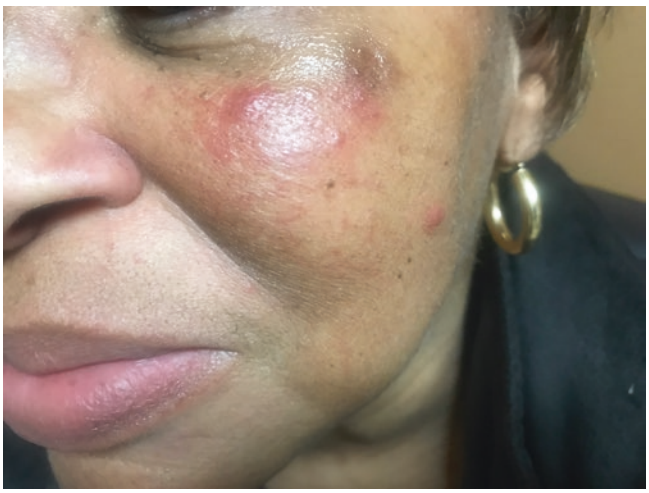

Fig. 2.167 Rosacea

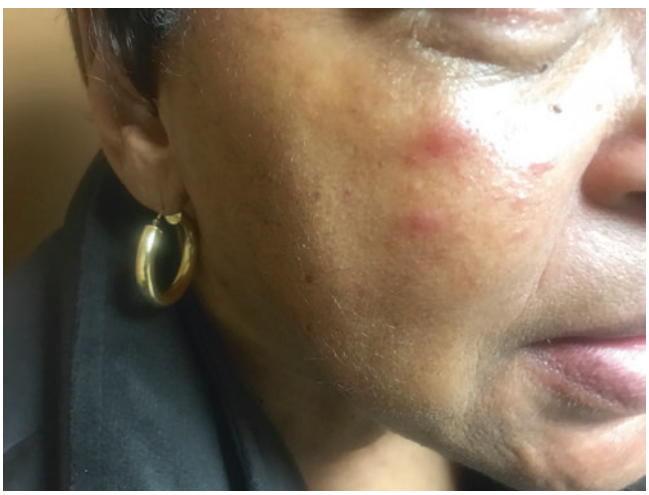

Fig. 2.168 Rosacea

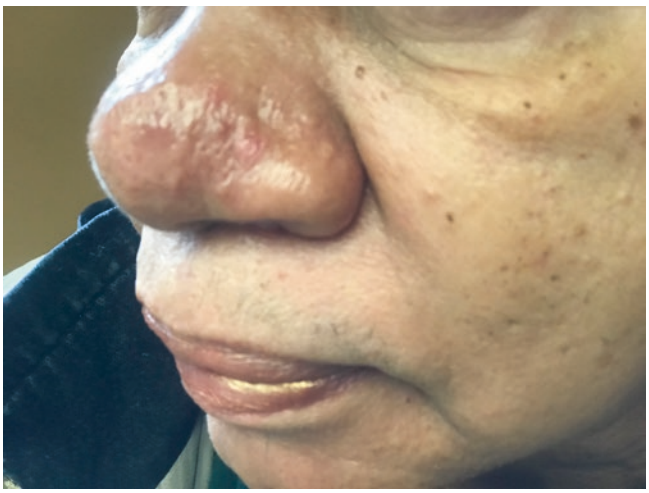

Fig. 2.169 Rosacea

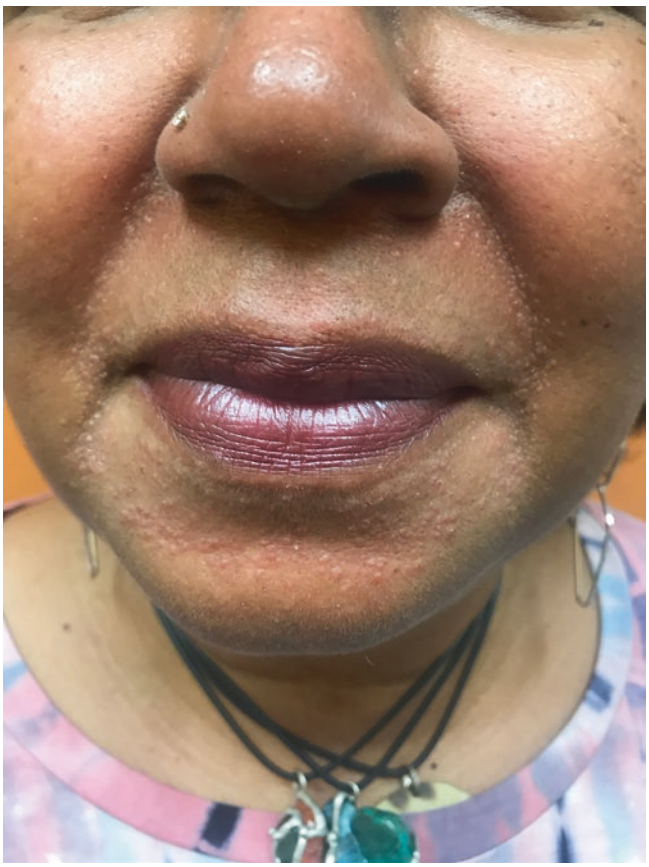

Fig. 2.170 Rosacea

African American patients had twice the odds of having severe HS than non–African American patients, and of being diagnosed at a more advanced stage of disease. Clinically, post-inflammatory hyperpigmentation (PIH) is common, and the erythema of active inflammatory lesions can be masked by this PIH. Close examination for violaceous or dark red erythema at the lesion periphery can

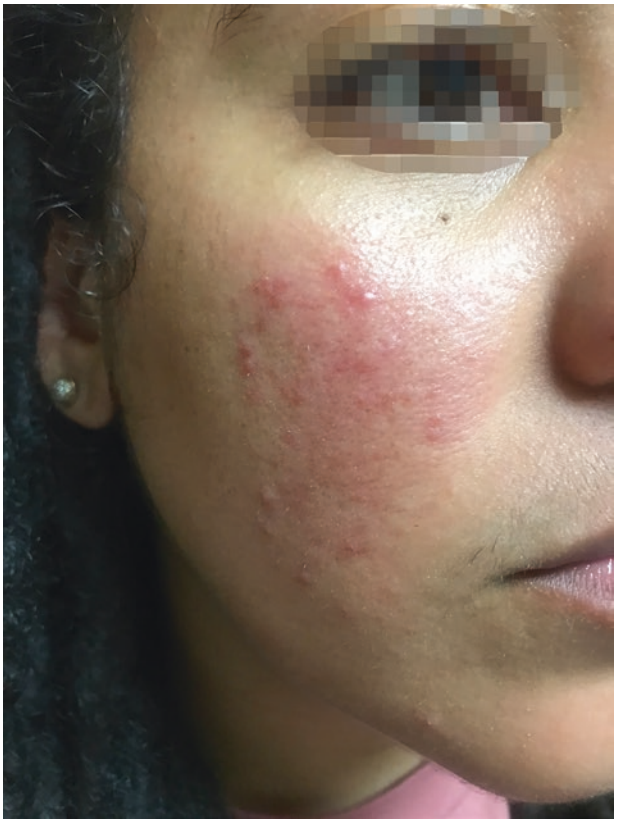

Fig. 2.171 Rosacea

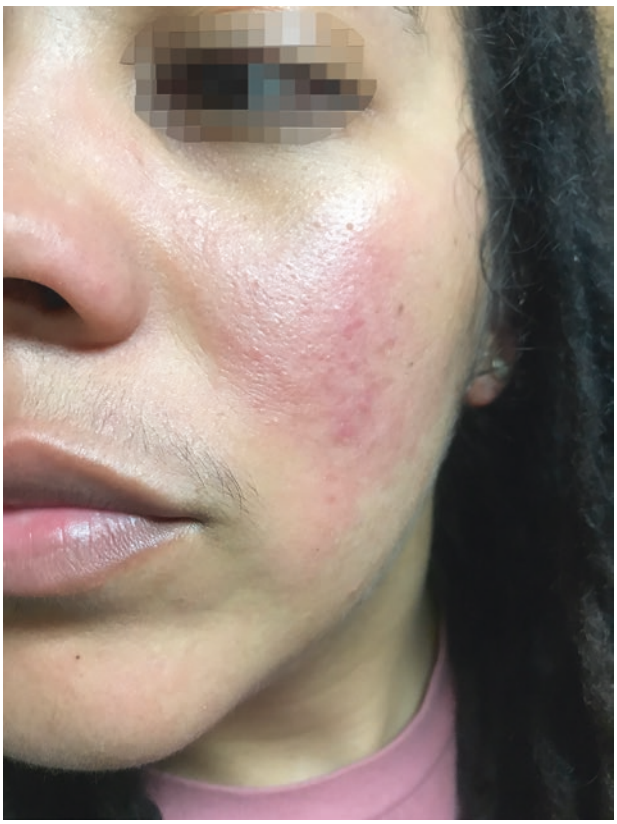

Fig. 2.172 Rosacea

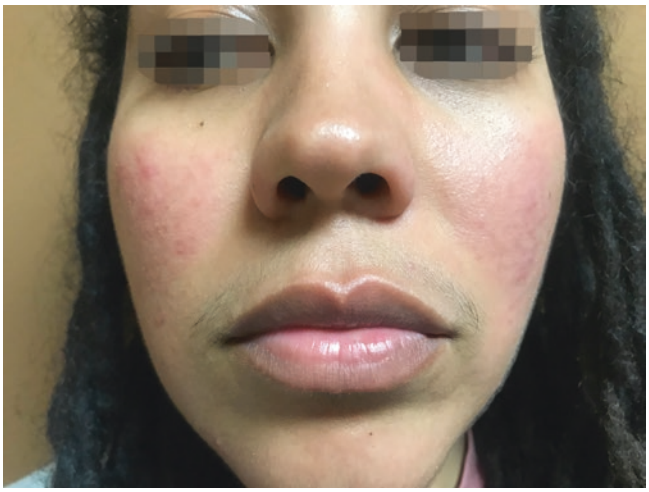

Fig. 2.173 Rosacea

Fig. 2.174 Juvenile rosacea

confirm active disease. According to the Hurley staging system, stage 1 describes HS with one or more abscesses with no scarring or sinus tracts (Figs. 2.181–2.185). Stage 2 has one or more widely spread abscesses that are recurrent and have scarring and/or sinus tract formation (Figs. 2.186–2.192). Stage 3 contains many abscesses that are interconnected by sinus tracts, affecting an entire area of the body (Figs. 2.193–2.202).

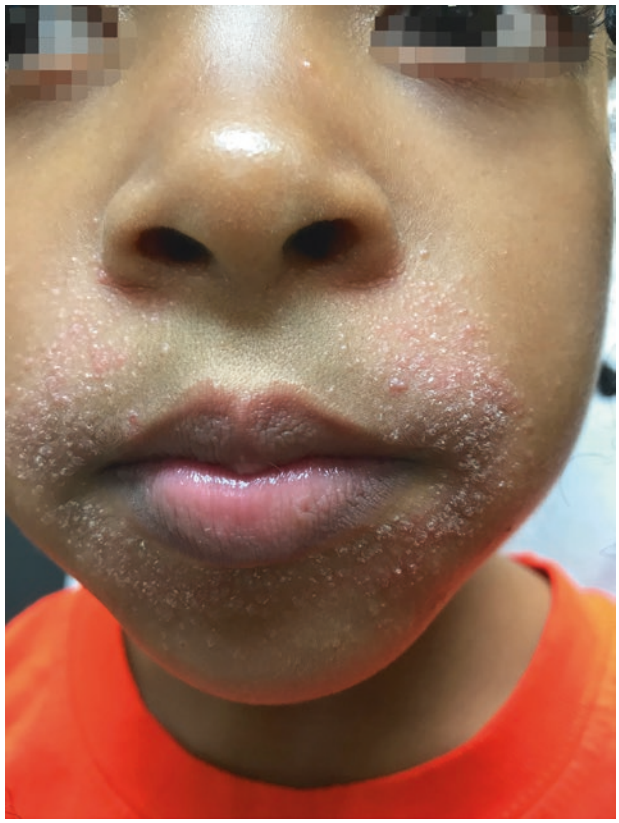

Fig. 2.175 Juvenile rosacea

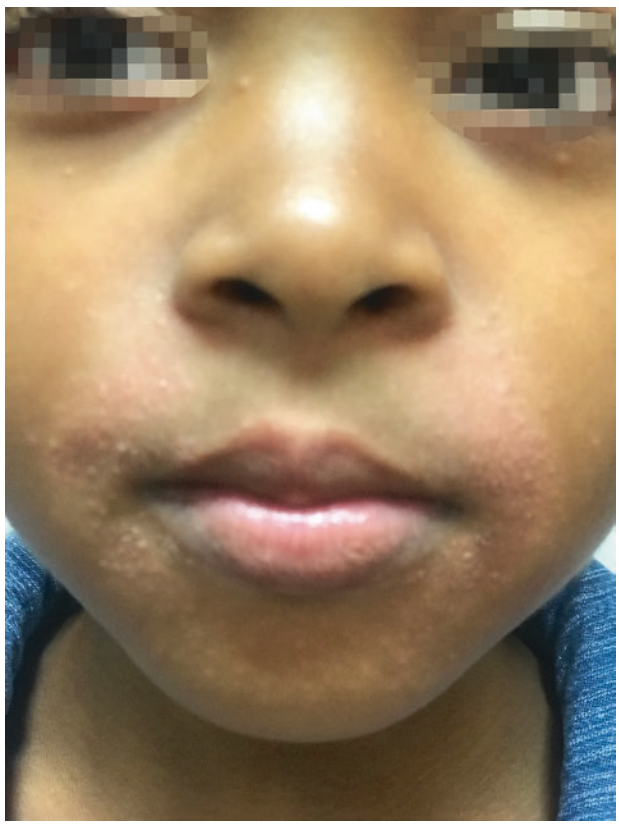

Fig. 2.176 Juvenile rosacea post treatment

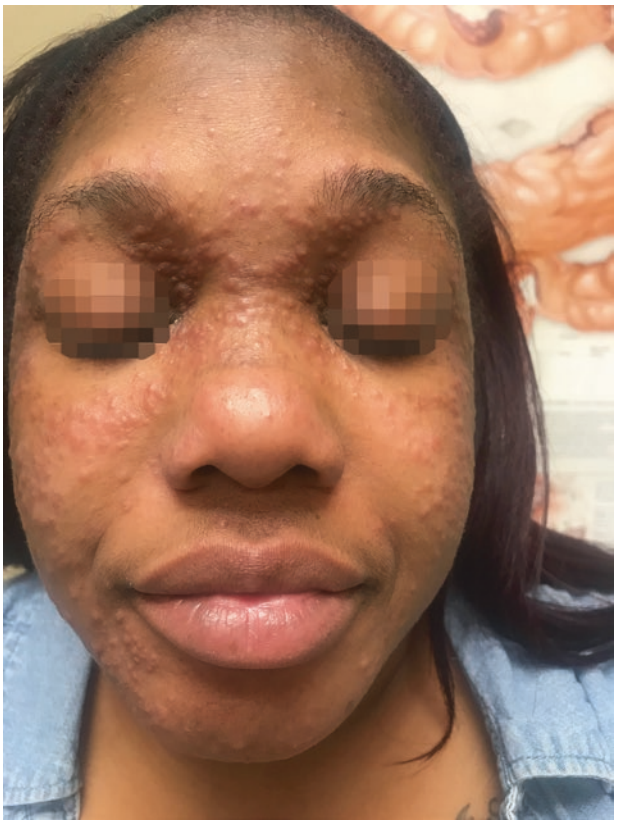

Fig. 2.177 Rosacea

Fig. 2.178 Rosacea

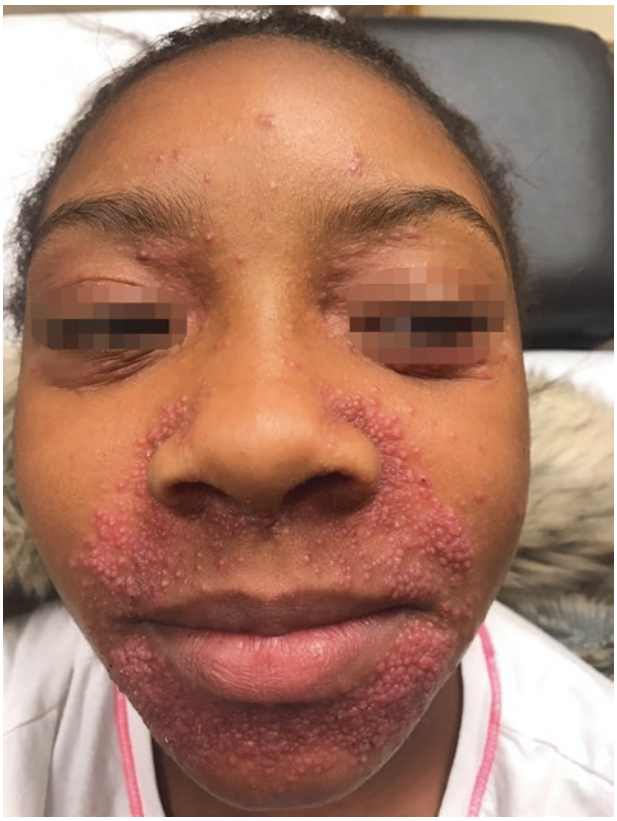

Fig. 2.179 Juvenile rosacea

Fig. 2.180 Juvenile rosacea post treatment

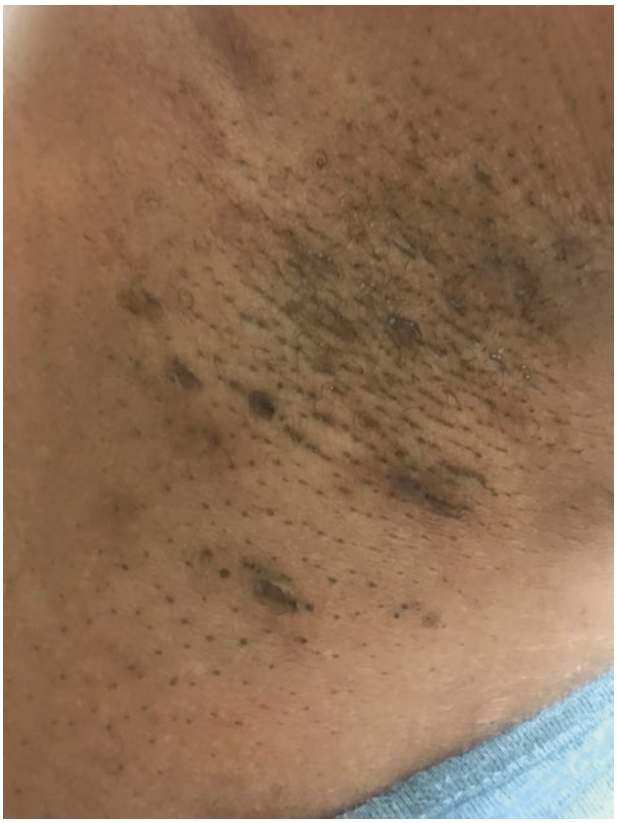

Fig. 2.181 Hidradenitis suppurativa (HS) stage 1

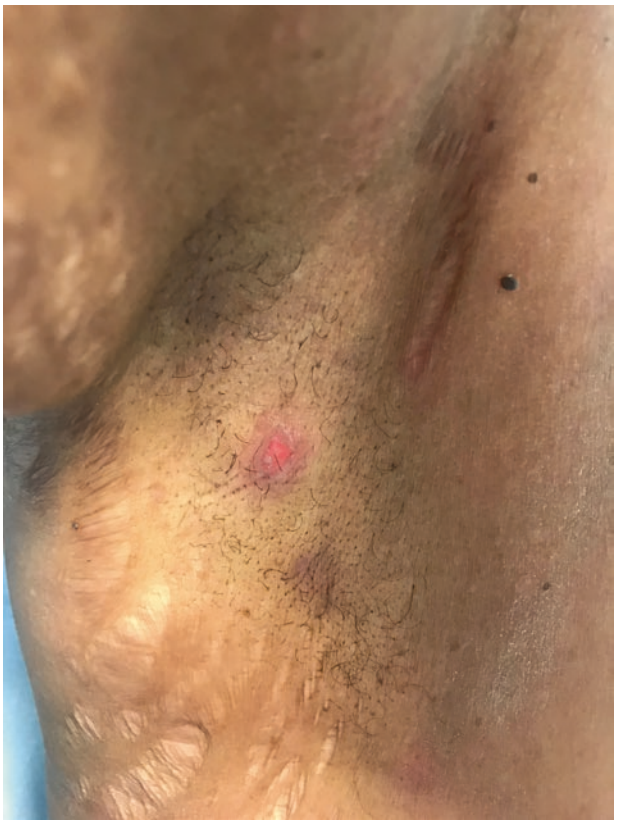

Fig. 2.182 HS stage 1

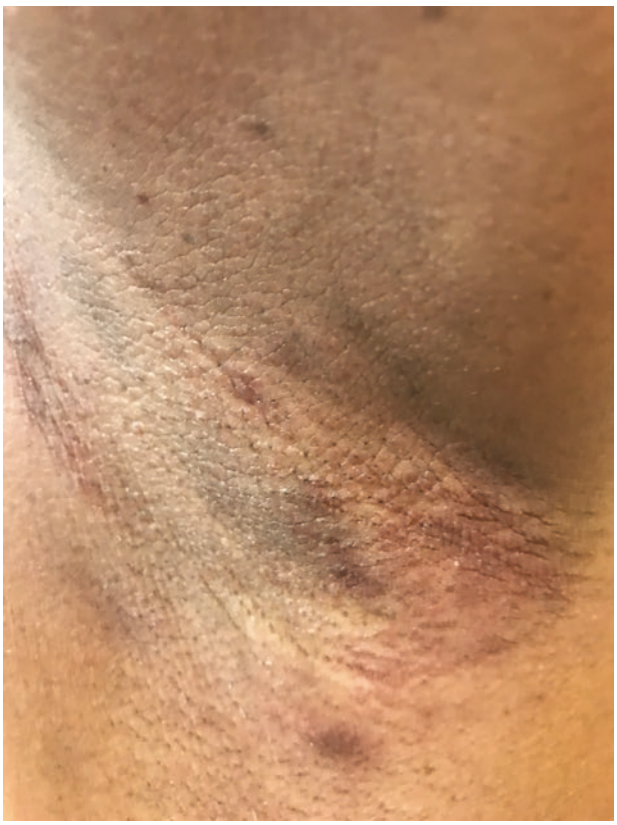

Fig. 2.183 HS stage 1

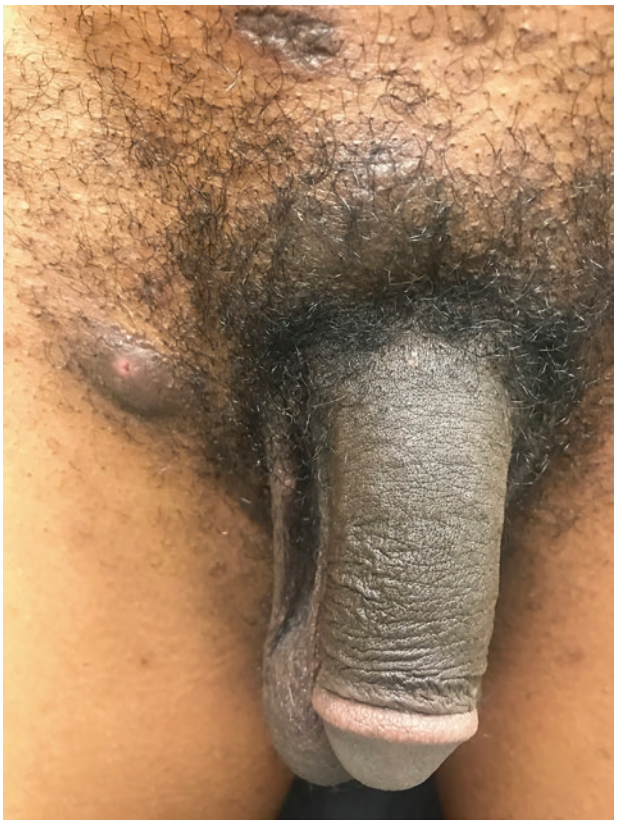

Fig. 2.184 HS stage 1

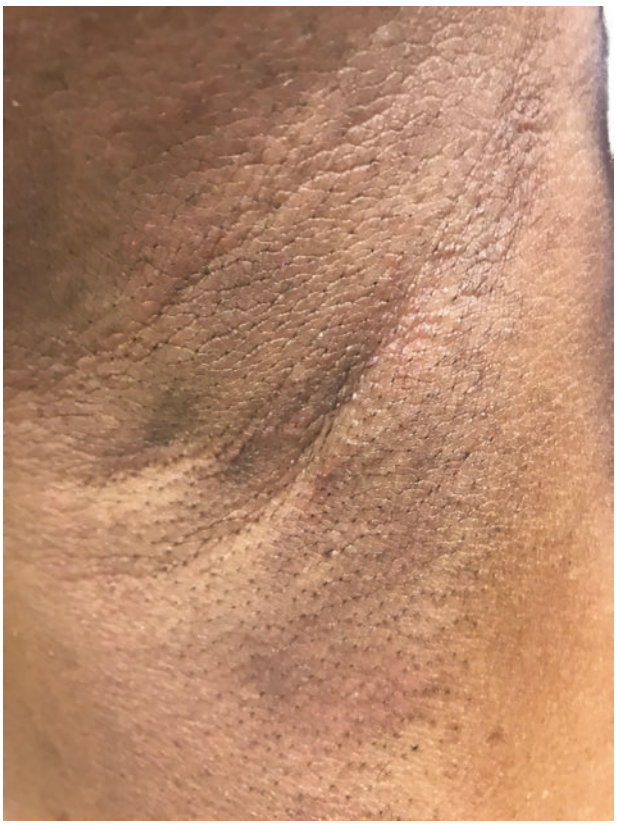

Fig. 2.185 HS stage 1

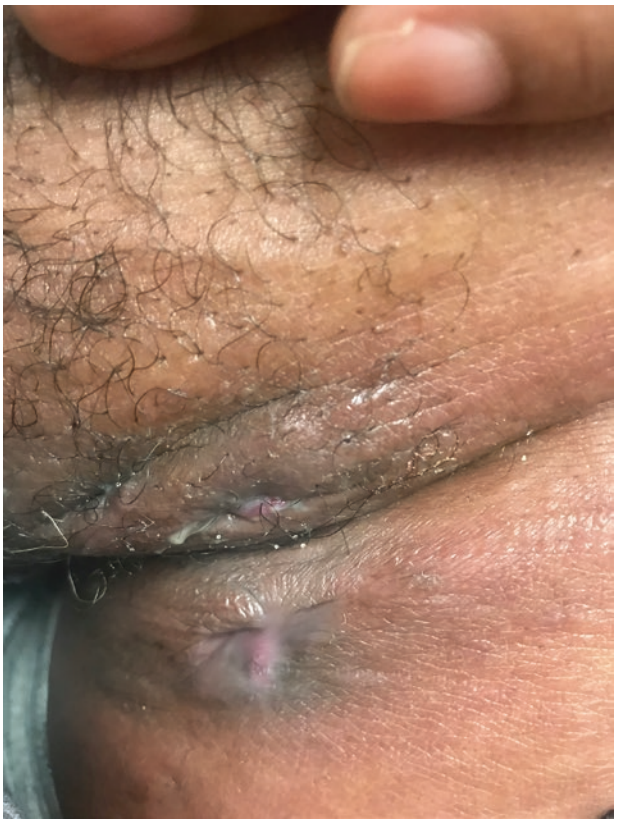

Fig. 2.187 HS stage 2

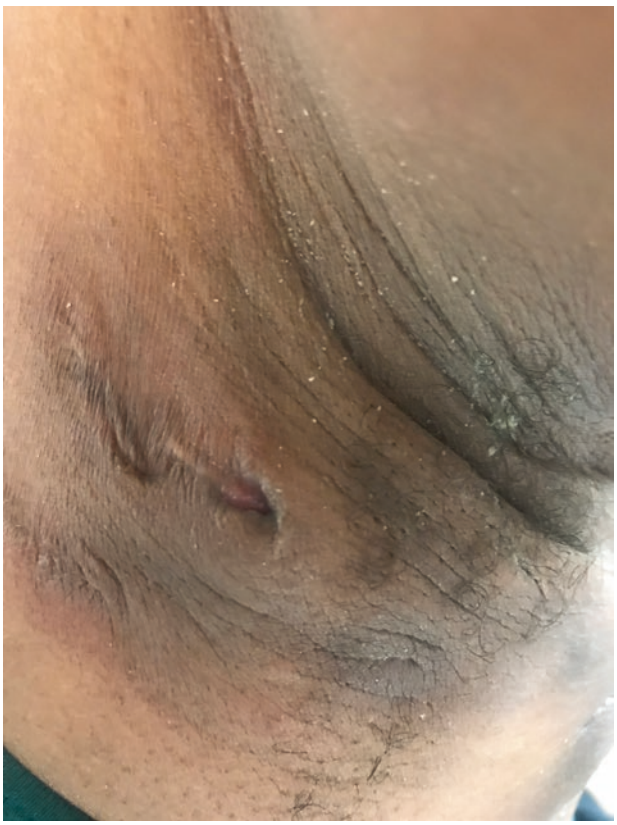

Fig. 2.186 HS stage 2

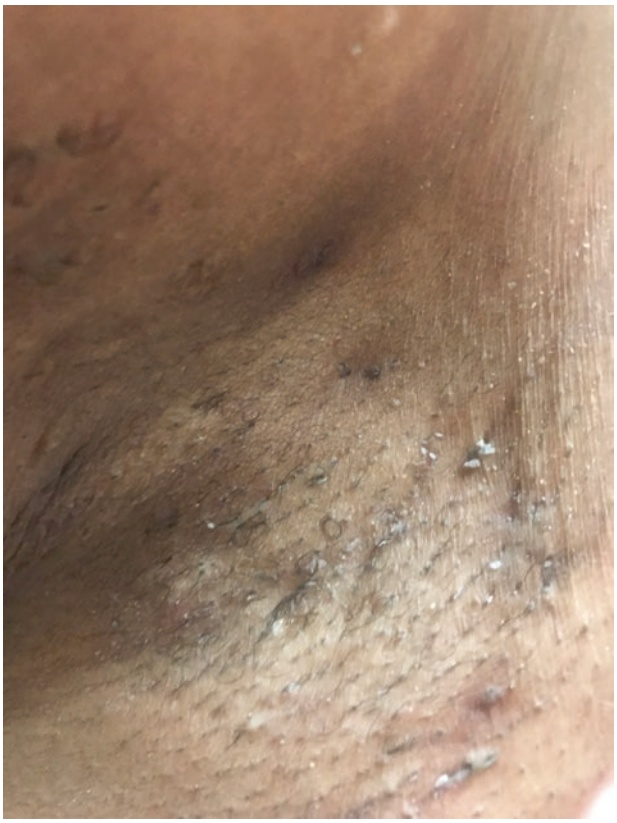

Fig. 2.188 HS stage 2

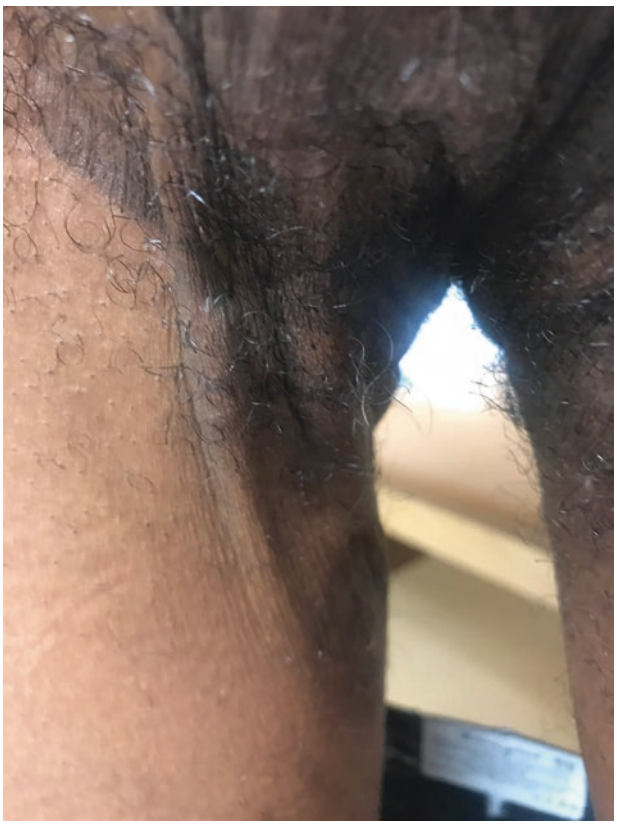

Fig. 2.189 HS stage 2

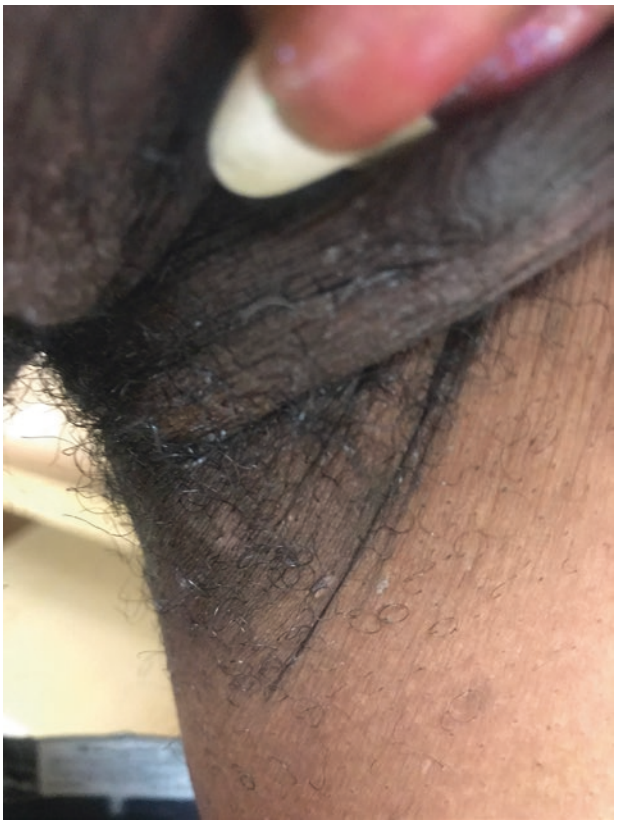

Fig. 2.190 HS stage 2

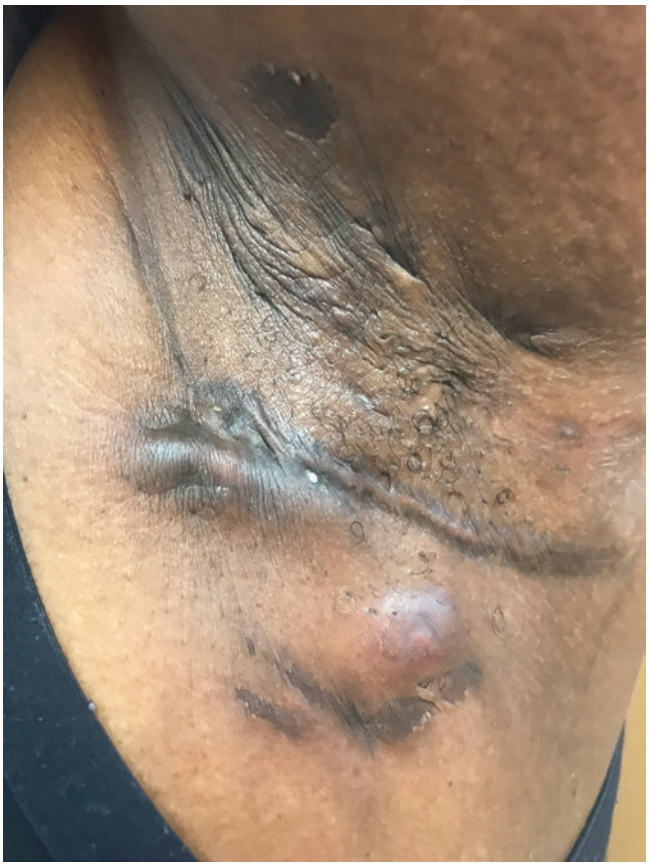

Fig. 2.191 HS stage 2

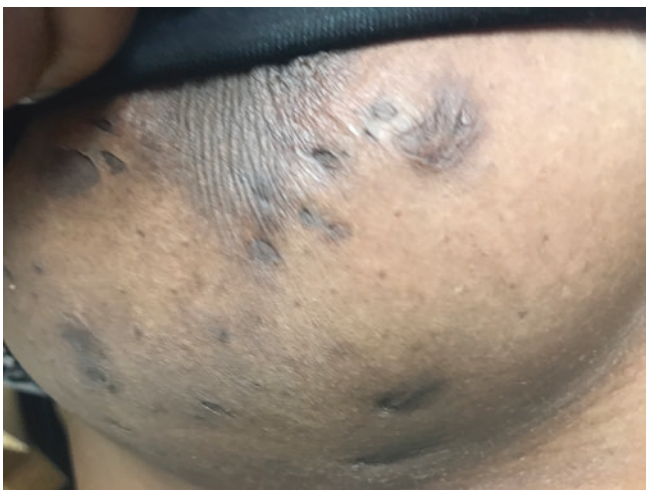

Fig. 2.192 HS stage 2

2.5.4.2 Management

For mild disease, topical antibiotics in combination with antimicrobial cleansers (benzoyl peroxide or chlorhexidine gluconate) are often effective. Oral antibiotics (especially levofloxacin oral retinoids) can be used as second- or third-line therapy for persistent, diffuse, or more severe disease. Biologics also show great promise in treatment of HS; so far only one biological agent has been approved for this purpose.

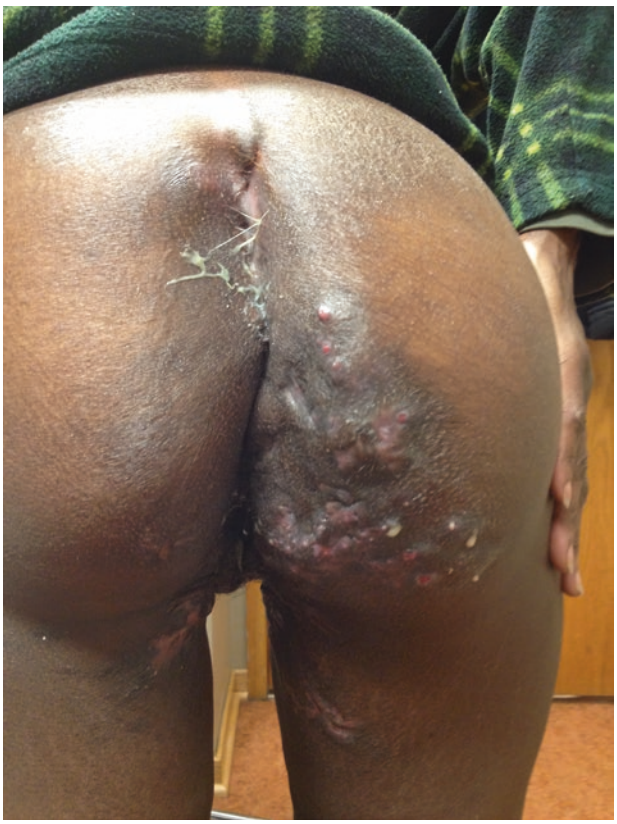

Fig. 2.193 HS stage 3

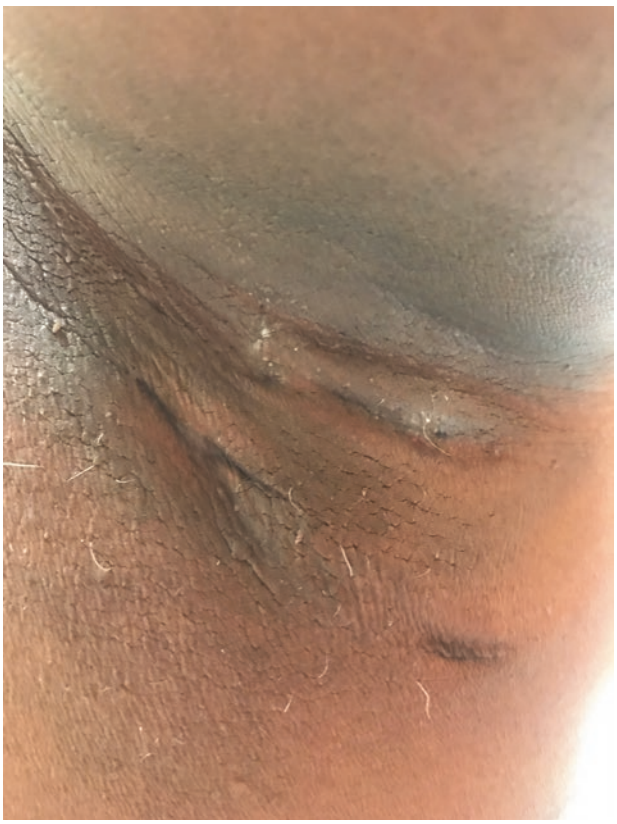

Fig. 2.195 HS stage 3

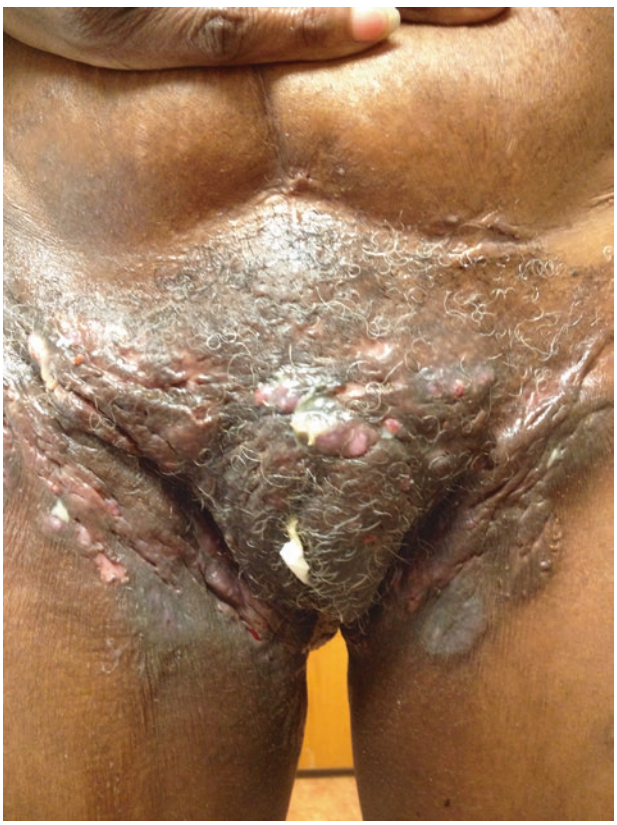

Fig. 2.194 HS stage 3

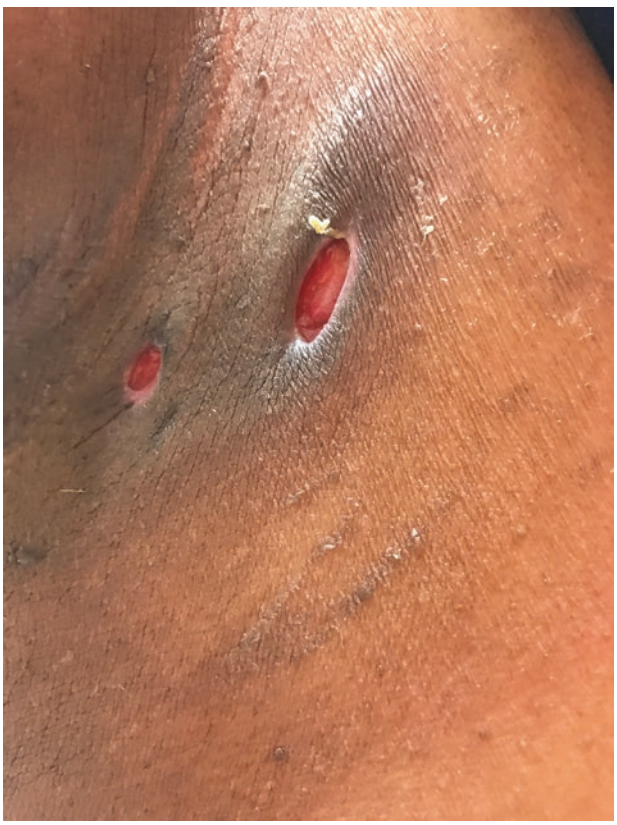

Fig. 2.196 HS stage 3

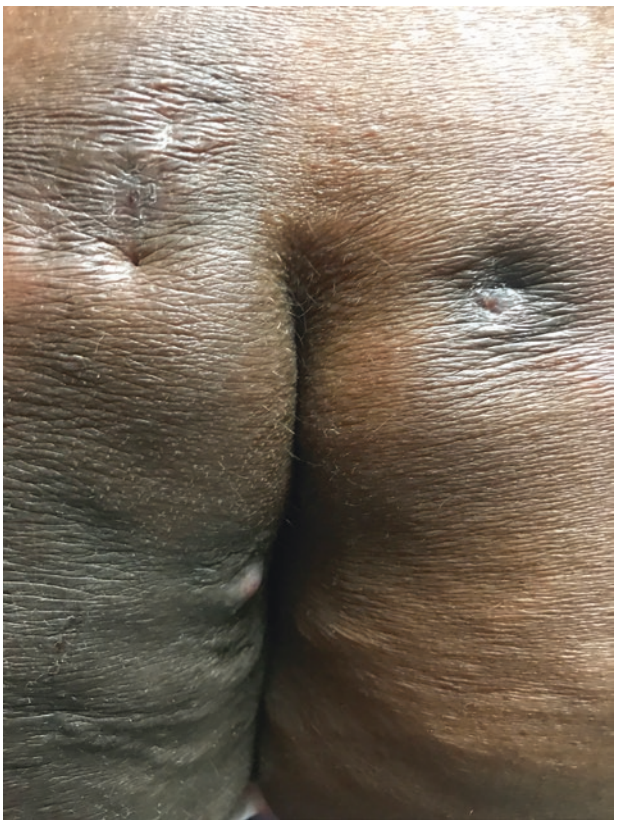

Fig. 2.197 HS stage 3

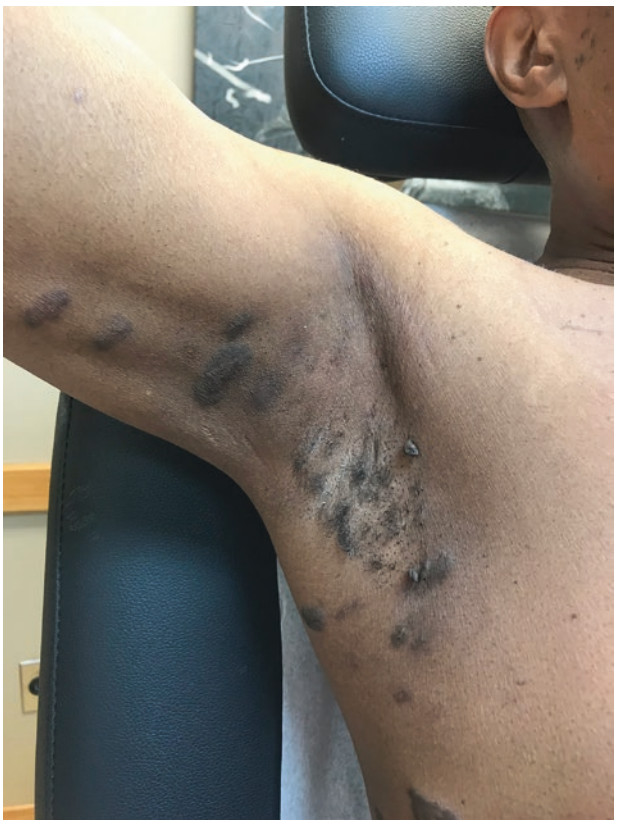

Fig. 2.199 HS stage 3

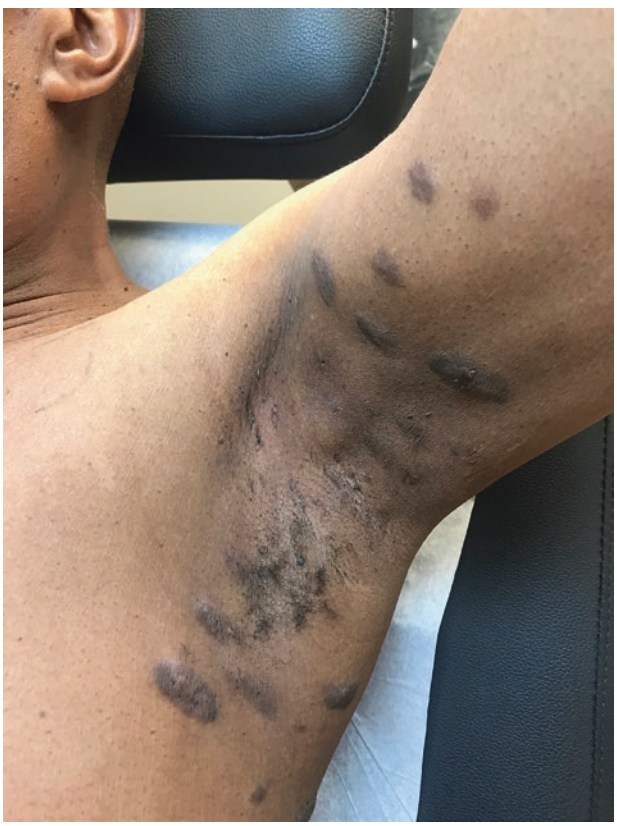

Fig. 2.198 HS stage 3

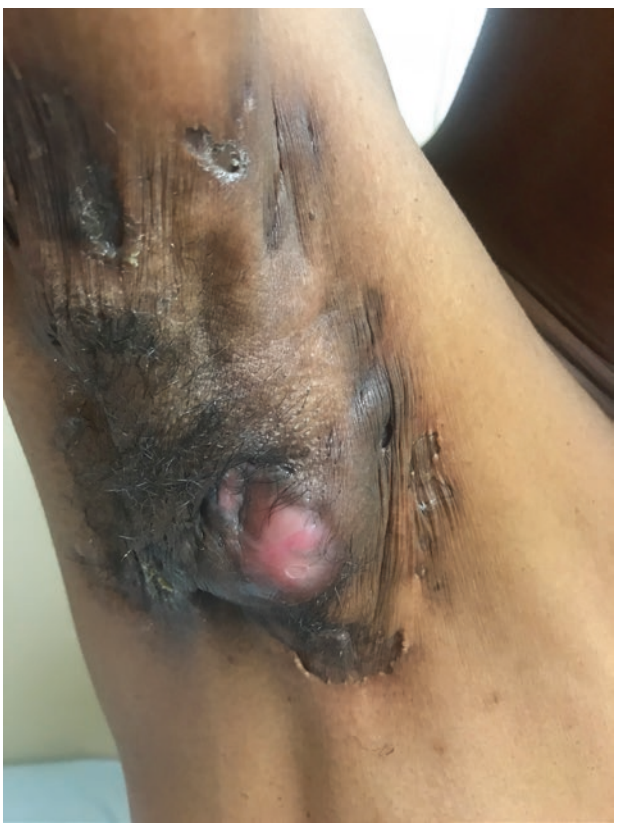

Fig. 2.200 HS stage 3

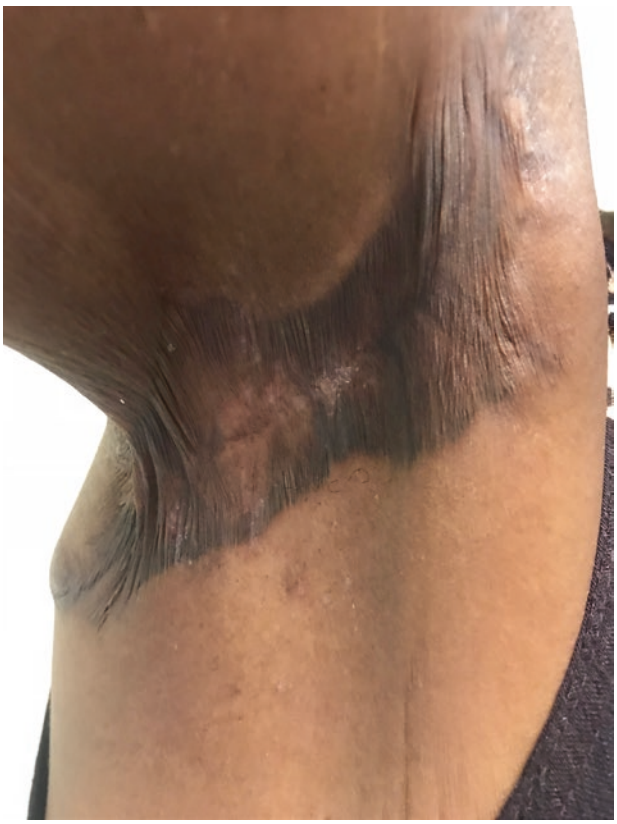

Fig. 2.201 HS stage 3

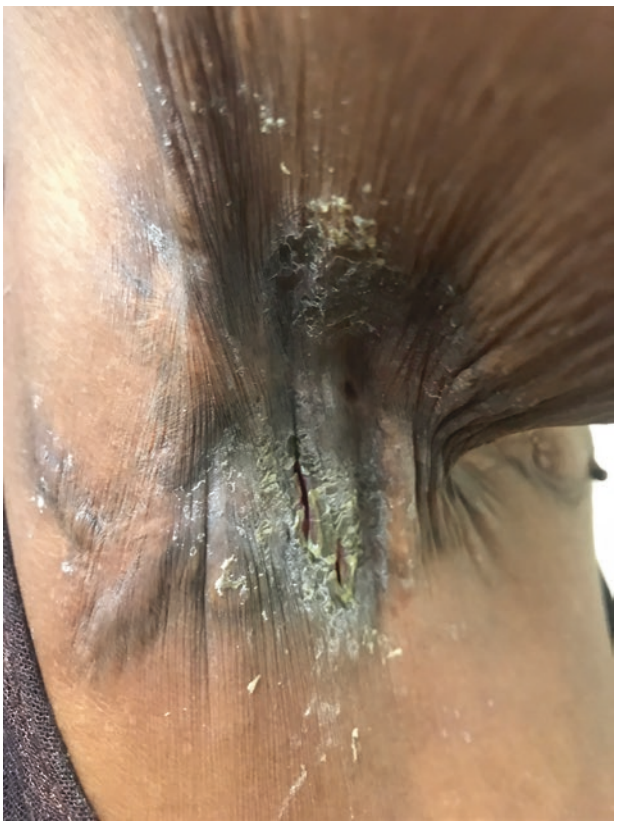

Fig. 2.202 HS stage 3

2.5.5 Pseudofolliculitis Barbae

Pseudofolliculitis barbae (PFB) develops in any area of the neck that has coarse hair and can occur in any race and both sexes. PFB is the result of hair of the neck that is curly and coarse, which can curl back into the skin and cause papules and inflammation that resemble folliculitis, hence the name. If the pseudofolliculitis happens in the neck, it is *barbae* (beard); elsewhere it is just pseudofolliculitis. PFB can be exacerbated with shaving and with close hair removal in any area that one shaves. Genetic factors also contribute to this condition, as do jobs that require shaving for their employees.

2.5.5.1 Presentation in Black Skin

Because most black patients have coarse, curly hair and beards, the condition is seen more often in this group. PFB is a clinical diagnosis; it shows erythematous or skin-colored papules that are firm around the neck area and the shaving areas of the face (Figs. 2.203–2.206). It can be painful, itchy, and burning, and the lesions tend to heal with hyperpigmentation. This condition can happen not only in men but also in women with hypertrichosis or after menopause.

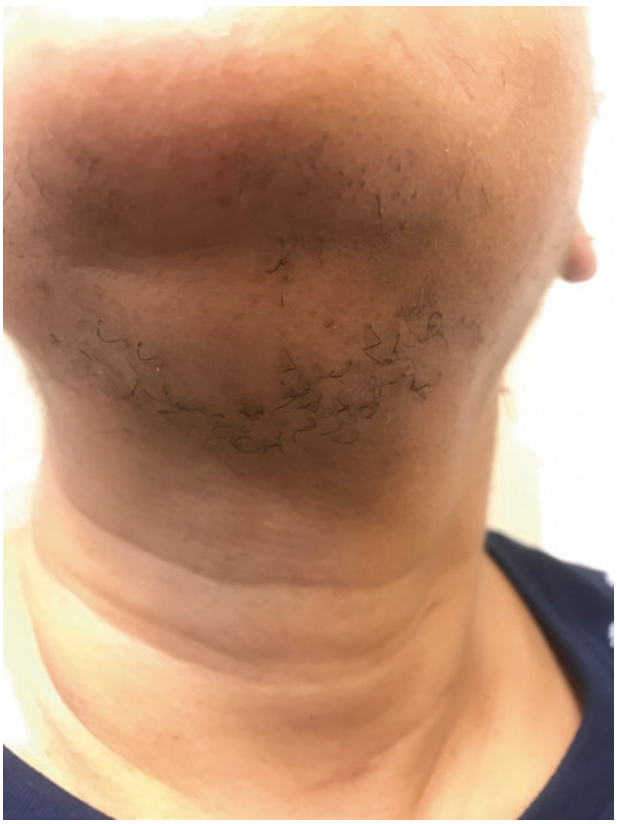

Fig. 2.203 Pseudofolliculitis barbae (PFB)

Fig. 2.204 PFB

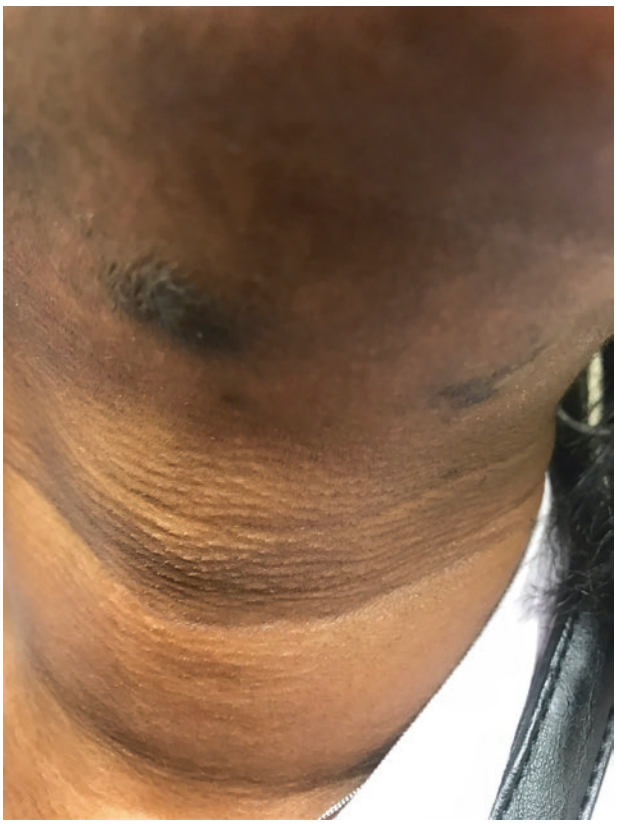

Fig. 2.205 PFB

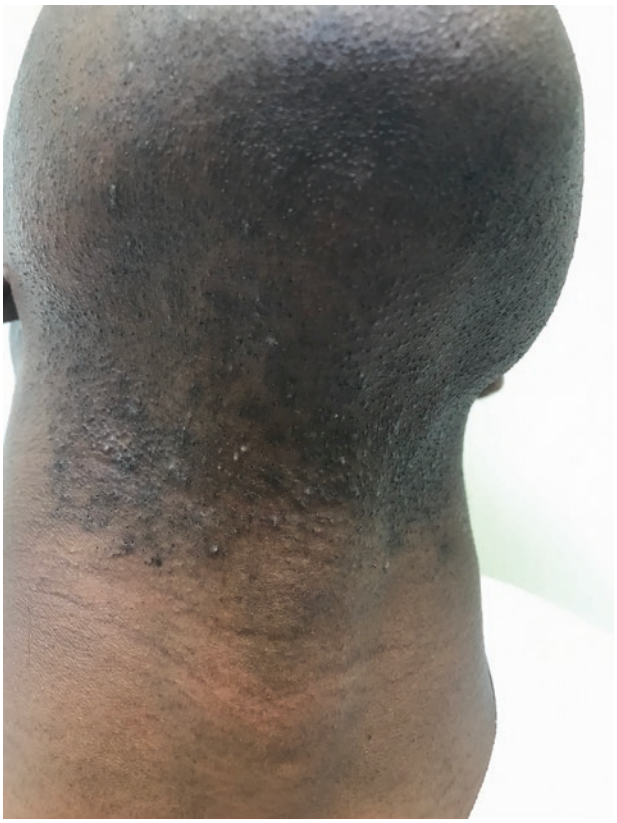

Fig. 2.206 PFB

2.5.5.2 Management

The best way to avoid this condition is to avoid shaving or close trimming of the hair areas, including plucking the beard. IF one must shave, close shaving is not advised. Laser hair removal can be substituted for shaving of the neck areas.

Topical steroids and antibiotics and benzoyl peroxide can be helpful with PFB. Interlesional steroids and antibiotics are helpful for more severe cases.

2.5.6 Disseminated and Recurrent Infundibulofolliculitis

Disseminated and recurrent infundibulofolliculitis (DRIF) is rare condition that involves the hair follicles and presents as widespread, mildly pruritic papules that resemble goose bumps on the trunk, arms, or neck.

2.5.6.1 Presentation in Black Skin

This condition so far has been exclusively reported in black patients and in hotter climates. As noted above, its papules are follicle-based, with mild keratosis and pruritus (Figs. 2.207–2.211). It mostly affects younger men.

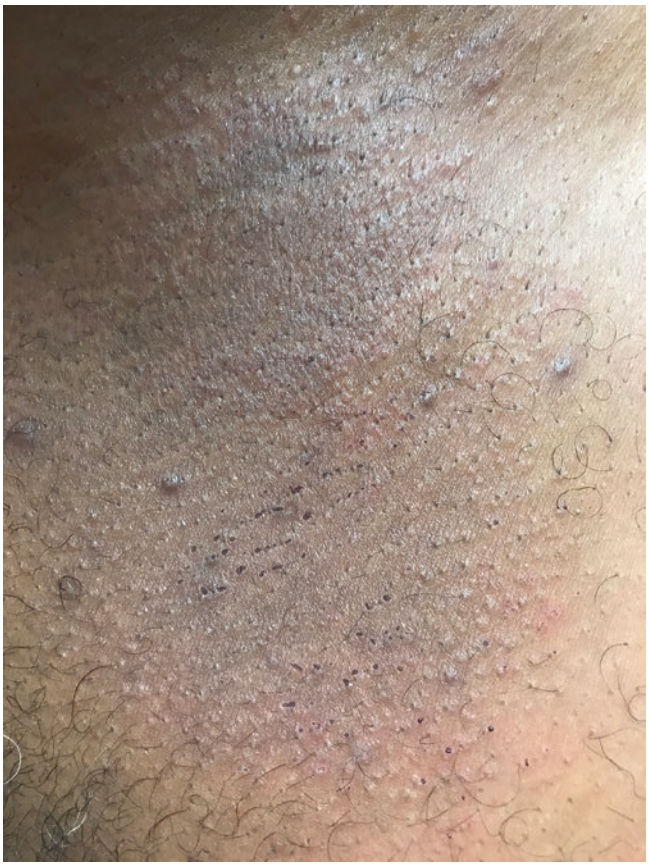

Fig. 2.207 Disseminated and recurrent infundibulofolliculitis (DRIF)

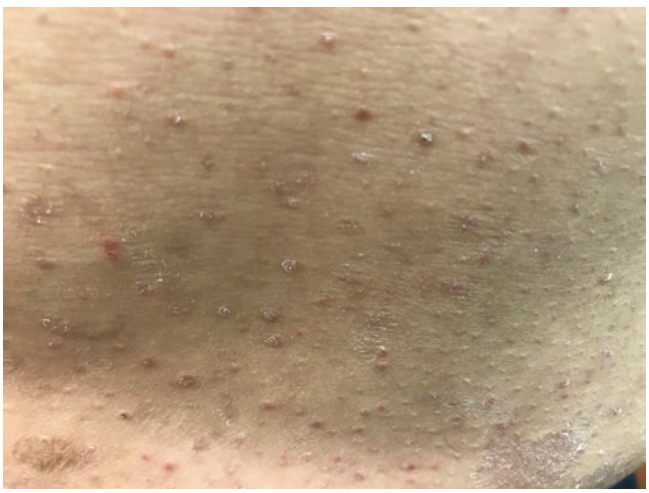

Fig. 2.208 DRIF

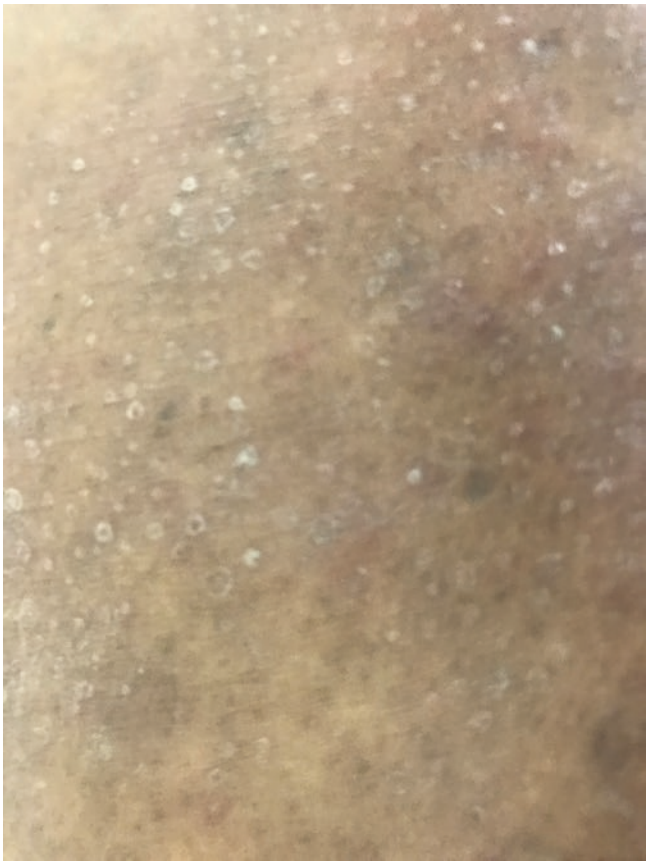

Fig. 2.209 DRIF

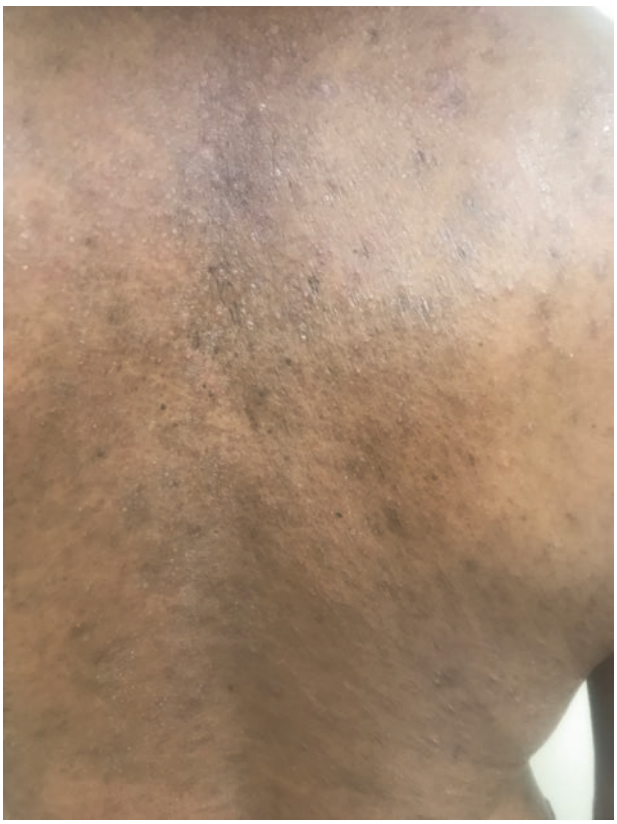

Fig. 2.210 DRIF

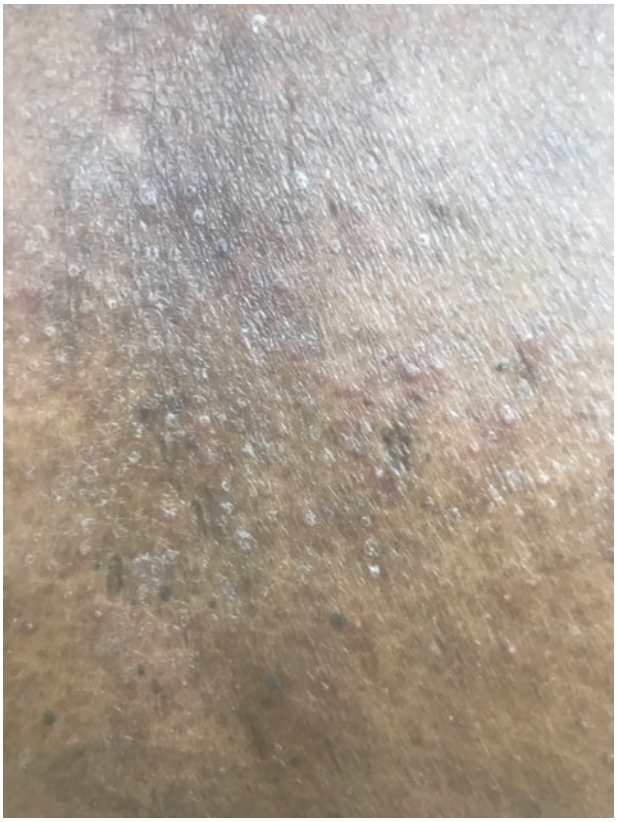

Fig. 2.211 DRIF

2.5.6.2 Management

Mild topical steroids and keratolytic agents are useful in this condition.

2.6 Hidrocystoma

Hidrocystomas are benign cysts of sweat gland origin that present primarily on the face, though they can occur on other sites. The subtypes, eccrine and apocrine, are defined by unique histologic features. Hidrocystomas can occur as solitary lesions or in multiple arrangements. The pathogenesis of hidrocystomas varies by type. Eccrine hidrocystomas develop as the result of excessive cystic dilatation of eccrine ducts, whereas apocrine hidrocystomas are thought to be benign neoplasms of the apocrine sweat gland coils. Data on the epidemiology of hidrocystoma is limited, but a recent US-based study found that the incidence of hidrocystoma was higher in women than in men, and African Americans were more likely to have hidrocystomas than either Caucasians or Hispanics.

2.6.1 Presentation in Black Skin

In addition to the classic translucent, flesh-colored to blue-gray cystic presentation of hidrocystoma, lesions may be more darkly pigmented in skin of color (Figs. 2.212–2.217).

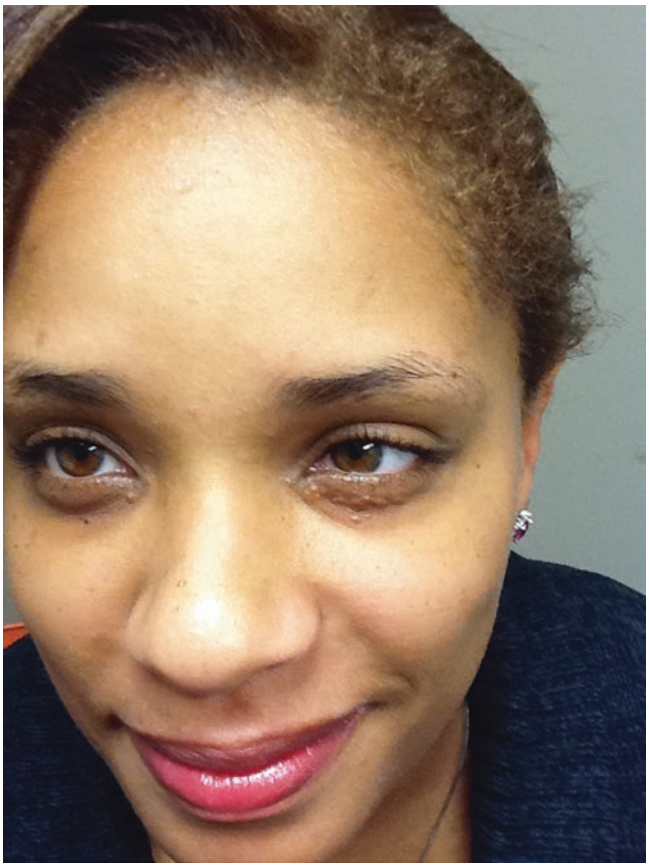

Fig. 2.212 Hidrocystoma

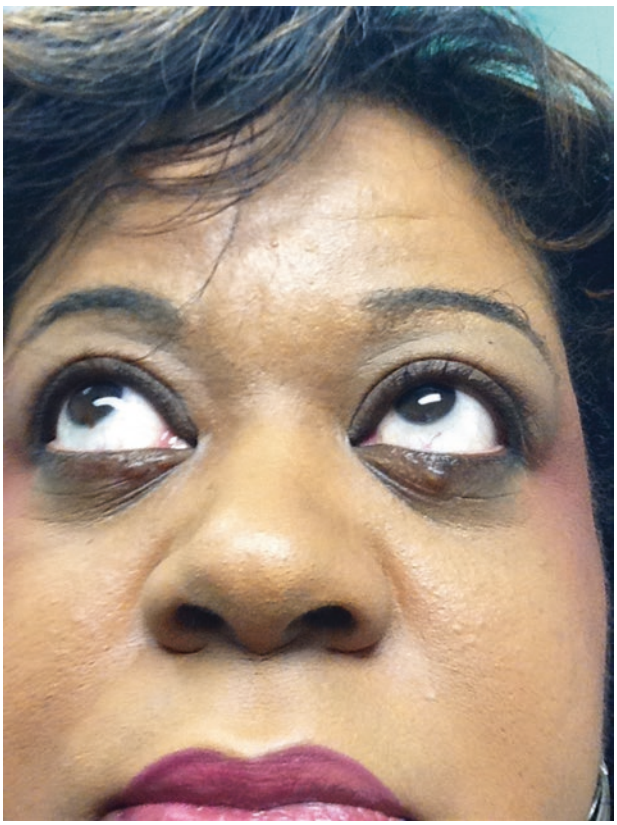

Fig. 2.213 Hidrocystoma

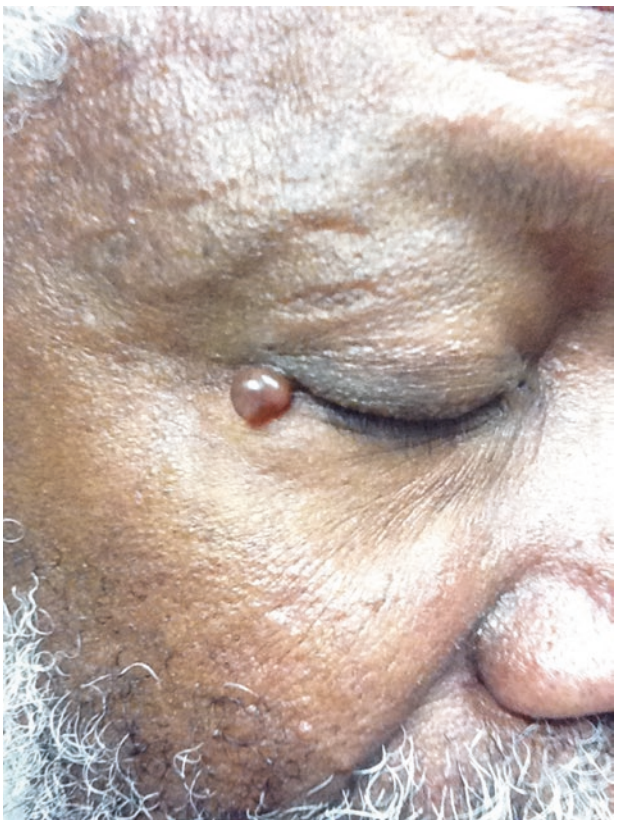

Fig. 2.214 Hidrocystoma

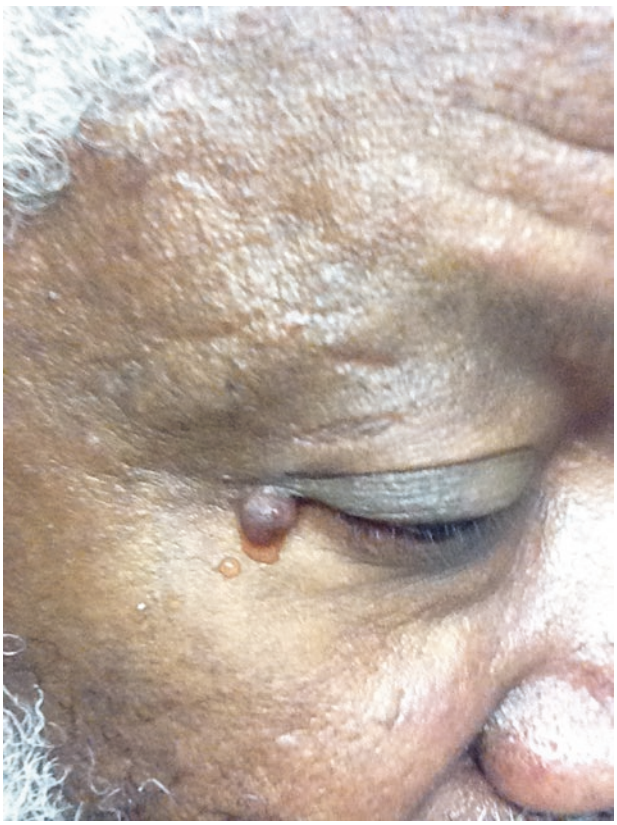

Fig. 2.215 Hidrocystoma, showing clear fluid after puncture

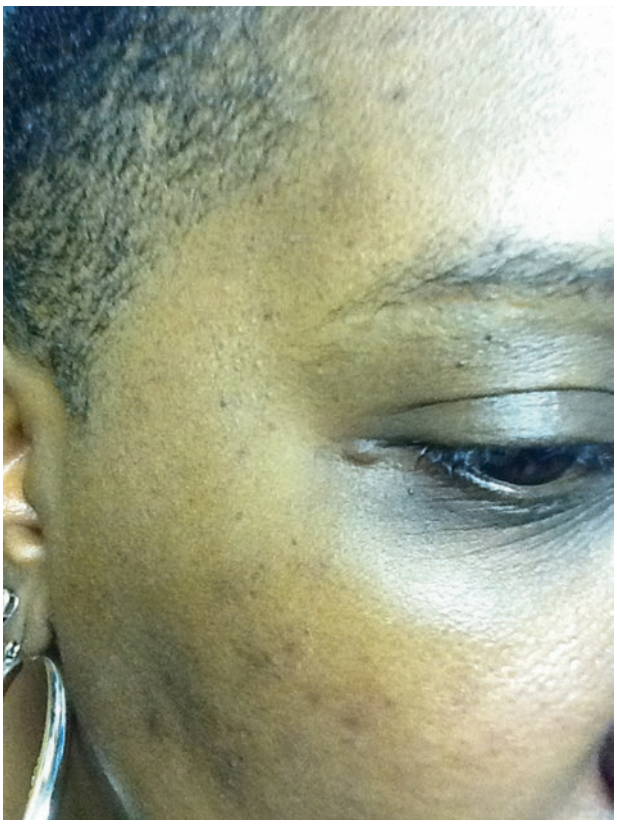

Fig. 2.216 Hidrocystoma

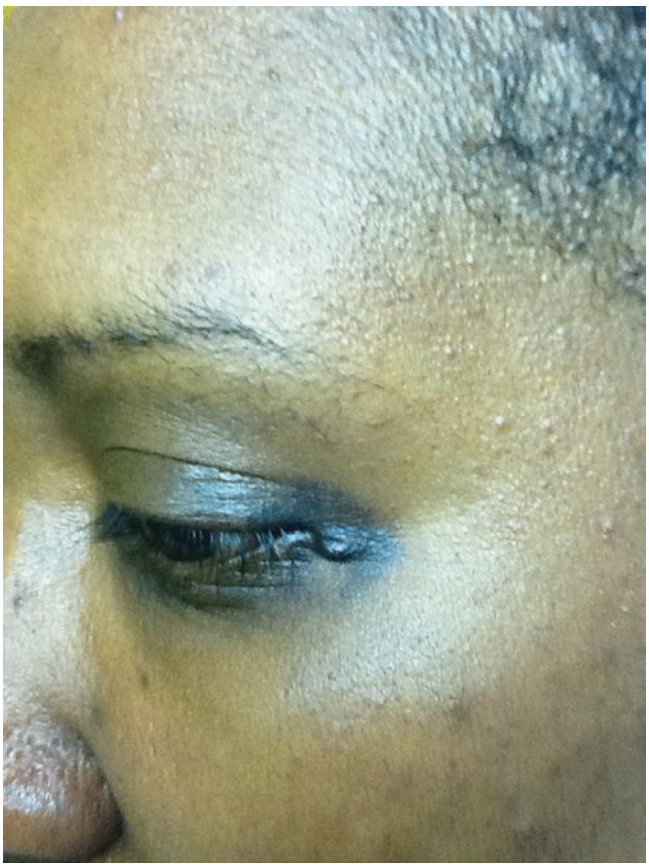

Fig. 2.217 Hidrocystoma

2.6.2 Management

Treatment options include simple excision, electrodesiccation, or CO_2 laser. Because of the high risk of scarring and permanent hyperpigmentation with use of CO_2 laser in deeply pigmented skin, this approach may be less appropriate for darker skin types.

2.7 *Pityrosporum* Folliculitis

Pityrosporum folliculitis (PF) is an inflammatory disease of hair follicles caused by hair follicle colonization by species of the superficial skin yeast *Malassezia*. Occurring on the trunk, upper arms, and less commonly the face, PF classically presents with pruritic or asymptomatic 2- to 3-mm dome-shaped, flesh-colored or erythematous papules with a central dell-like punctum. More severe cases may present as pustules, nodules, or cysts. More common in tropical climates, PF also has been associated with long-term antibiotic use, diabetes mellitus, and immunosuppression.

2.7.1 Presentation in Black Skin

The erythema of inflamed PF lesions may appear purple-brown or dark brown in skin of color (Figs. 2.218–2.232). In patients

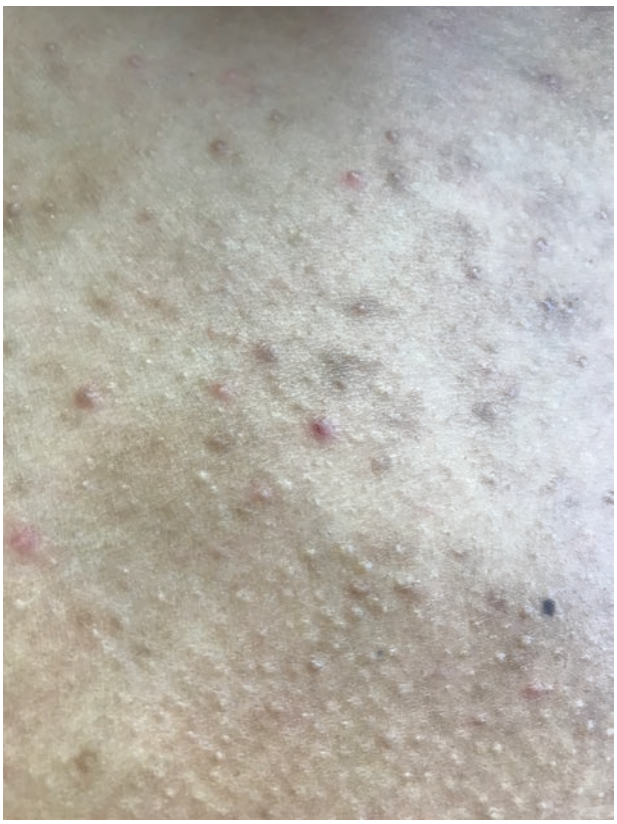

Fig. 2.219 PF

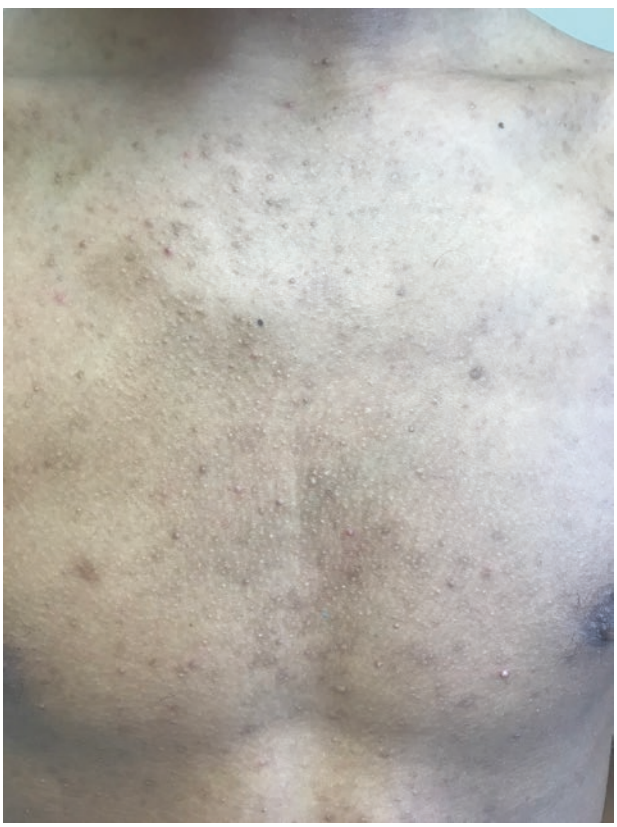

Fig. 2.218 *Pityrosporum* folliculitis (PF)

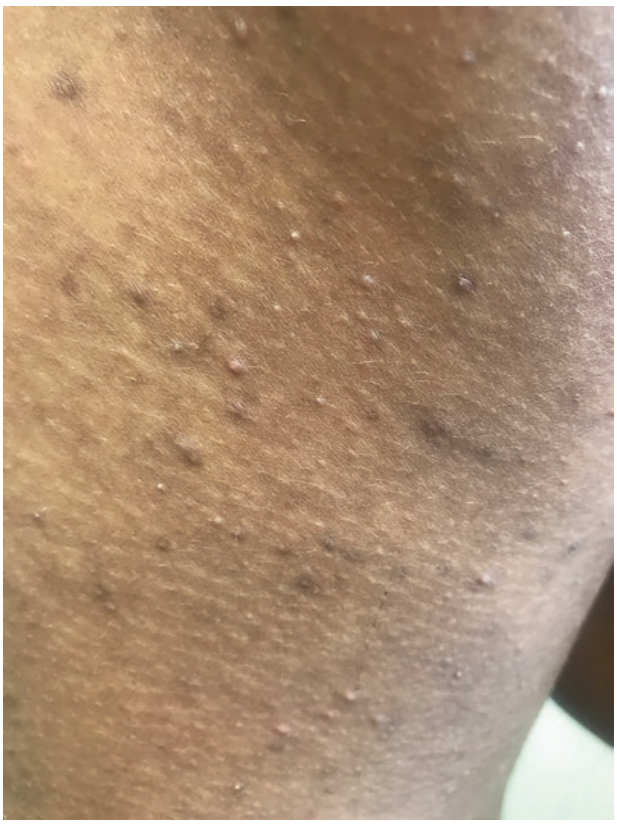

Fig. 2.220 PF

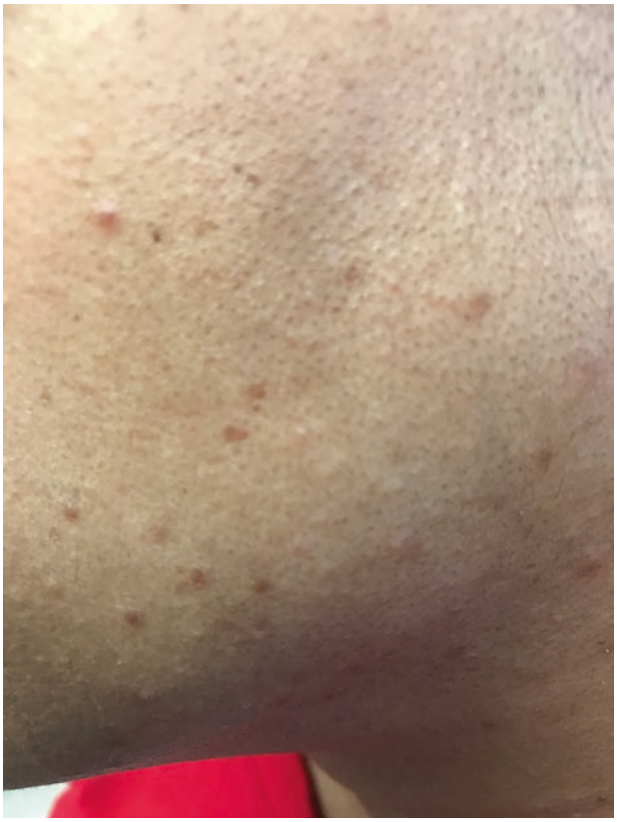

Fig. 2.221 PF

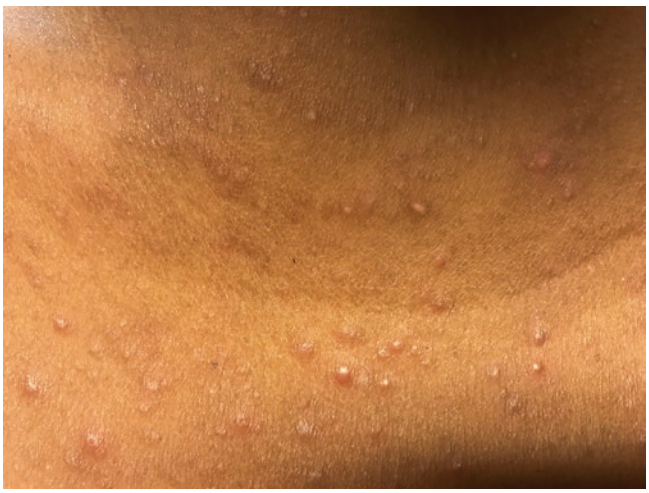

Fig. 2.222 PF

of color in tropical climates, the disease may be more likely to present with pustules or cysts, and with facial involvement.

2.7.2 Management

Topical treatment with antifungal agents is first-line therapy. Oral antifungal medications can be used for disease that is severe, diffuse, or resistant to topical treatment.

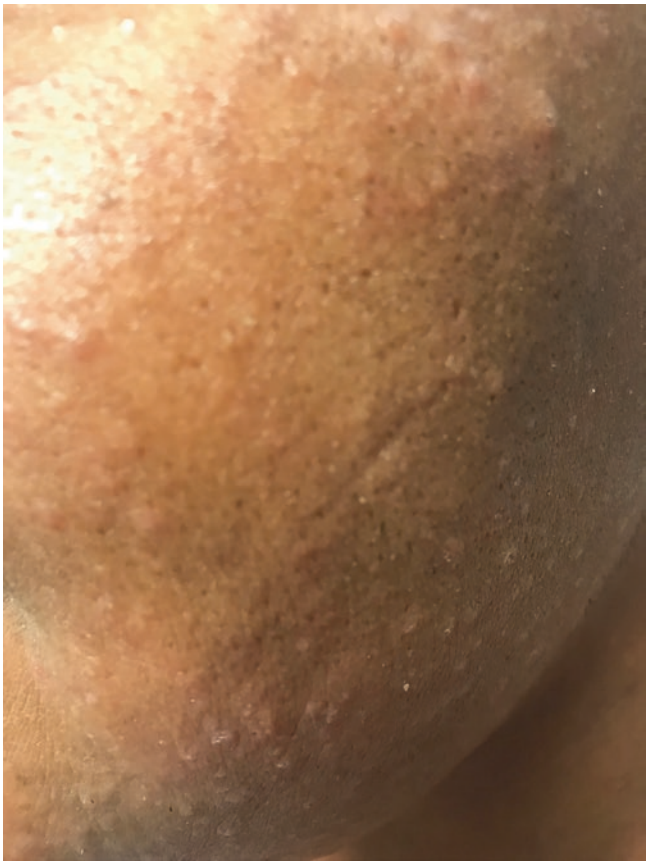

Fig. 2.223 PF

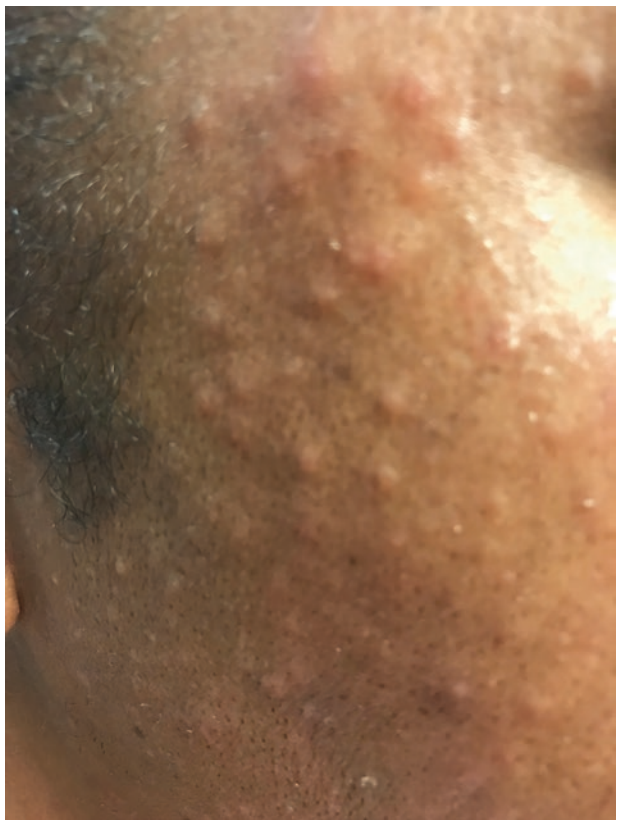

Fig. 2.224 PF

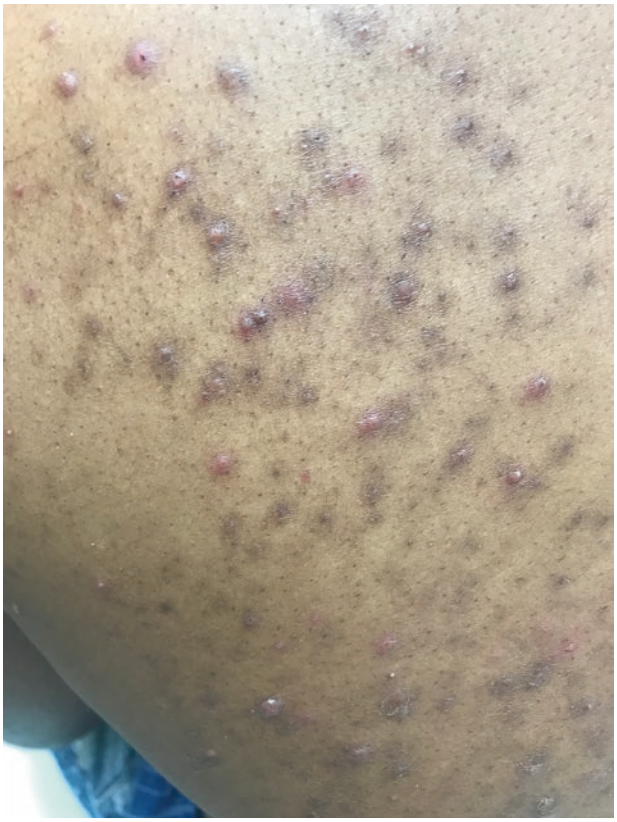

Fig. 2.225 PF

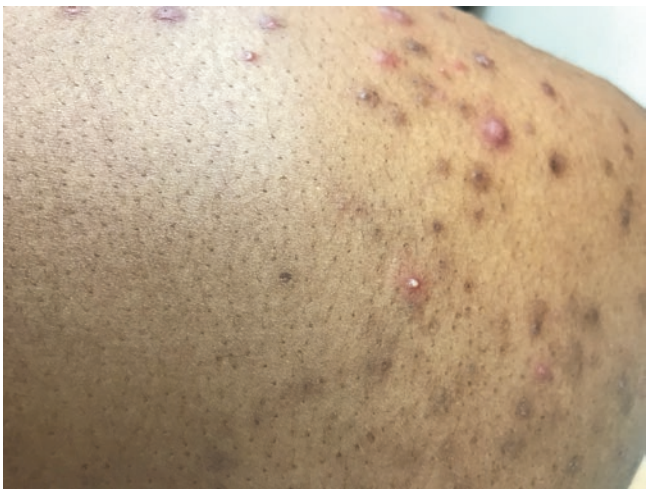

Fig. 2.226 PF

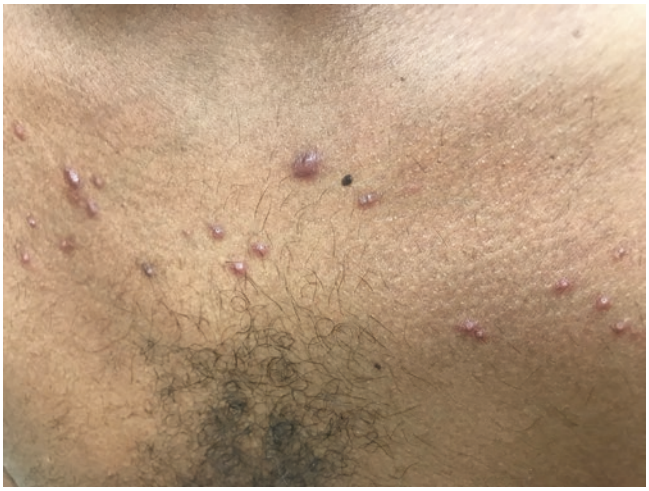

Fig. 2.227 PF

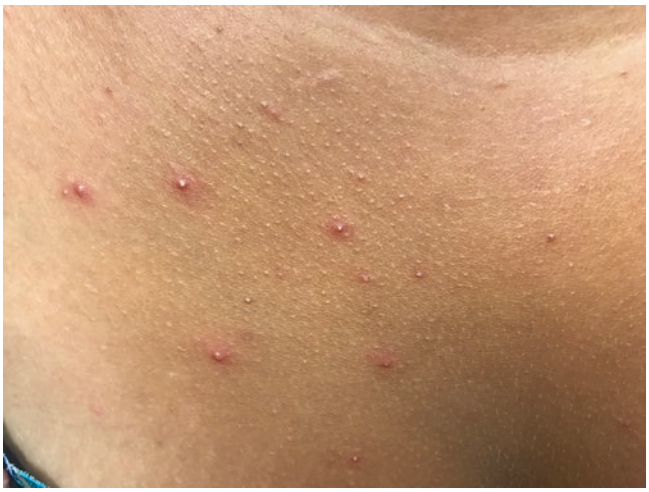

Fig. 2.228 PF

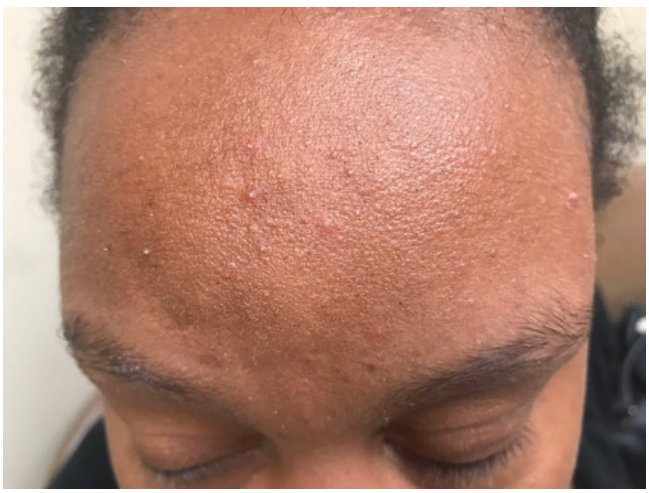

Fig. 2.229 PF

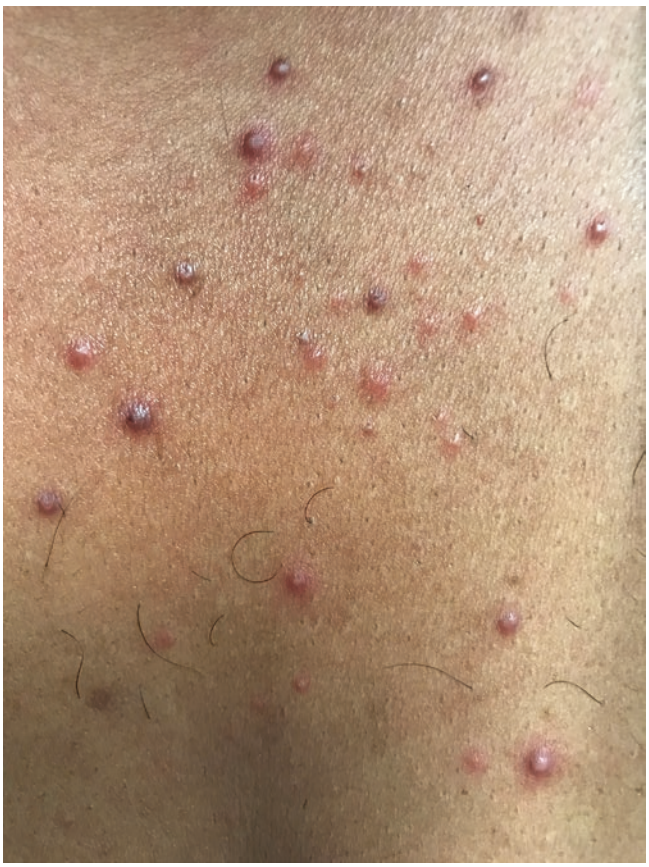

Fig. 2.230 PF

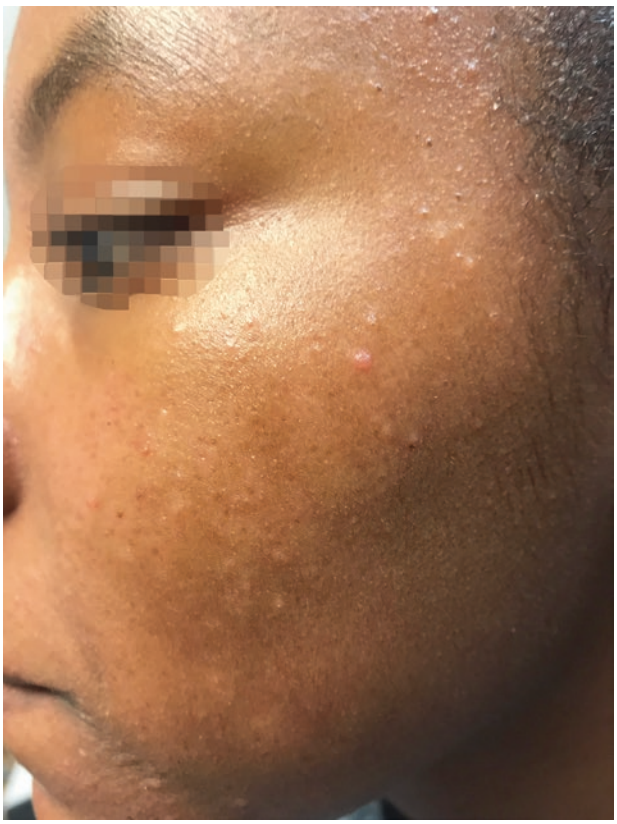

Fig. 2.231 PF

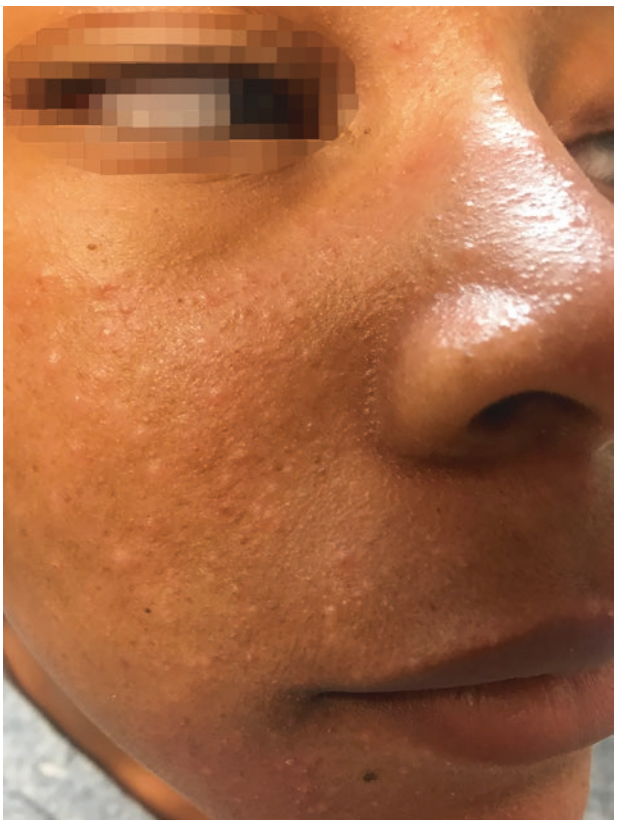

Fig. 2.232 PF

2.8 Dermatosis Papulosis Nigra

Dermatosis papulosis nigra (DPN) appears as small, brown to black papules on the face, neck, and trunk of many patients of color, especially those of African and Asian descent. These lesions tend to increase with age and sometime are itchy and cause irritation when they come in contact with jewelry and clothing. Pathologically, they are like seborrheic keratoses (SK), and clinically, they can coalesce to resemble them. (Otherwise, SKs are rare in black patients.) Over half of patients with DPN report a family history of DPN.

2.8.1 Presentation in Black Skin

Small brown to black papules on the face and neck and around the cheek are characteristics of DPN (Figs. 2.233–2.246).

Many black patients are affected by this condition. Women are affected more than men, and the more florid cases are only in women. DPN can happen at any age, but is usually seen more often after the fourth decade of life. DPN favors the cheeks and side of the face and neck, followed by the chest, arms, back, and axillae.

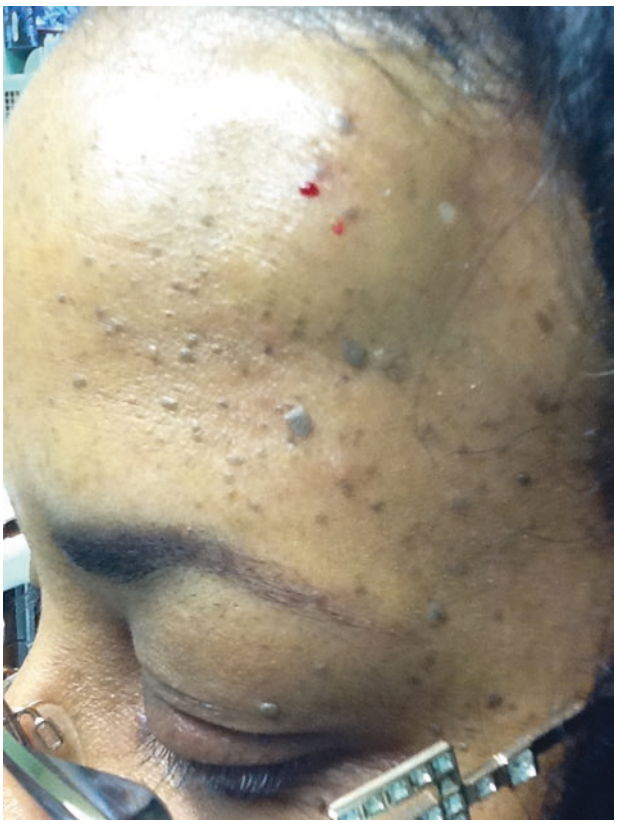

Fig. 2.233 Dermatosis papulosis nigra (DPN)

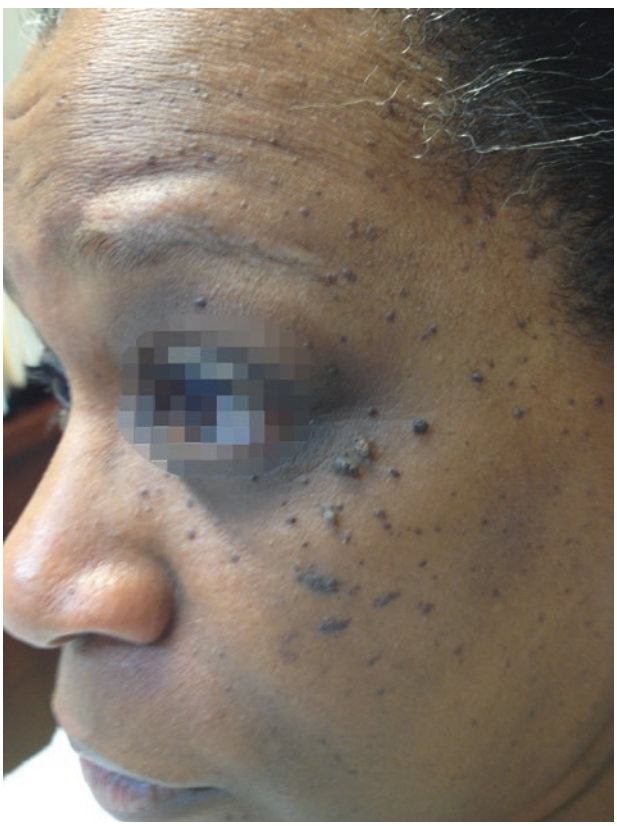

Fig. 2.234 DPN

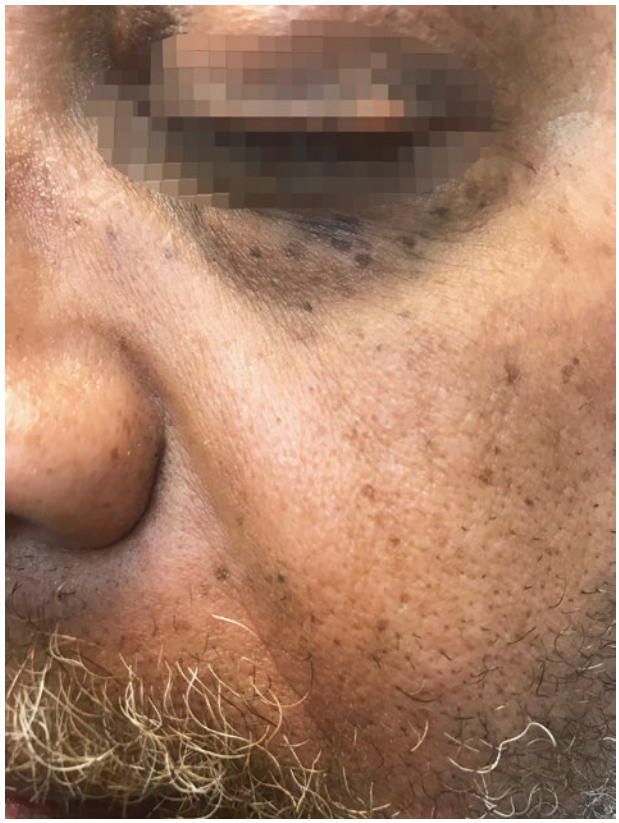

Fig. 2.235 DPN

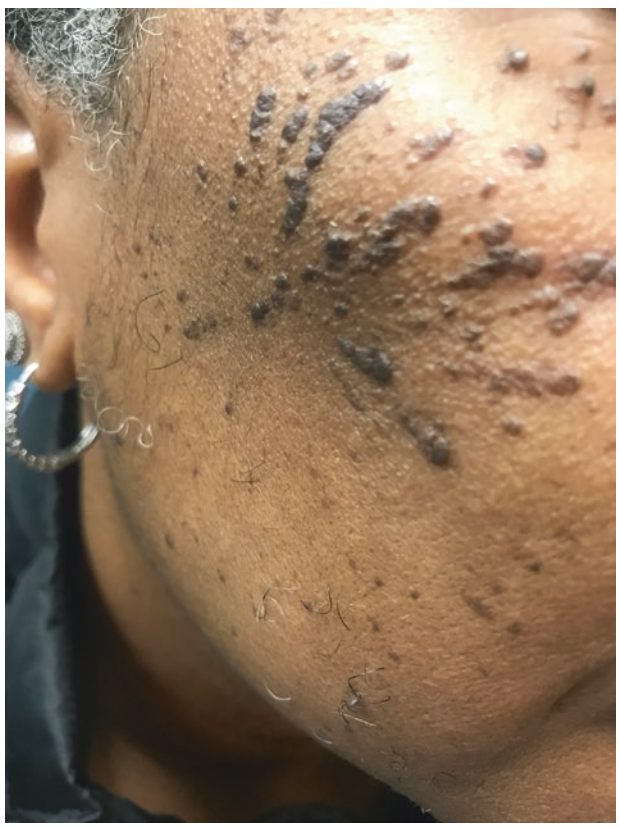

Fig. 2.236 DPN

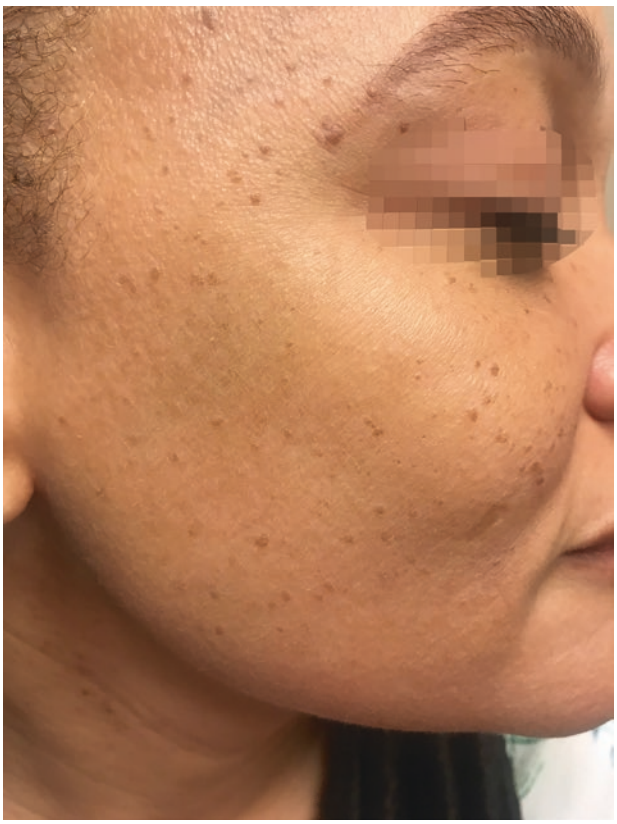

Fig. 2.237 DPN

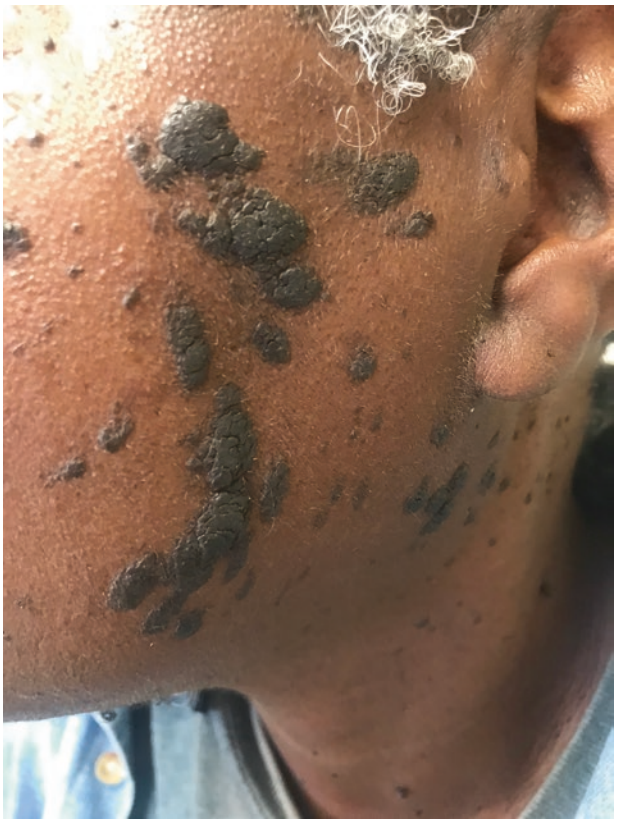

Fig. 2.239 DPN

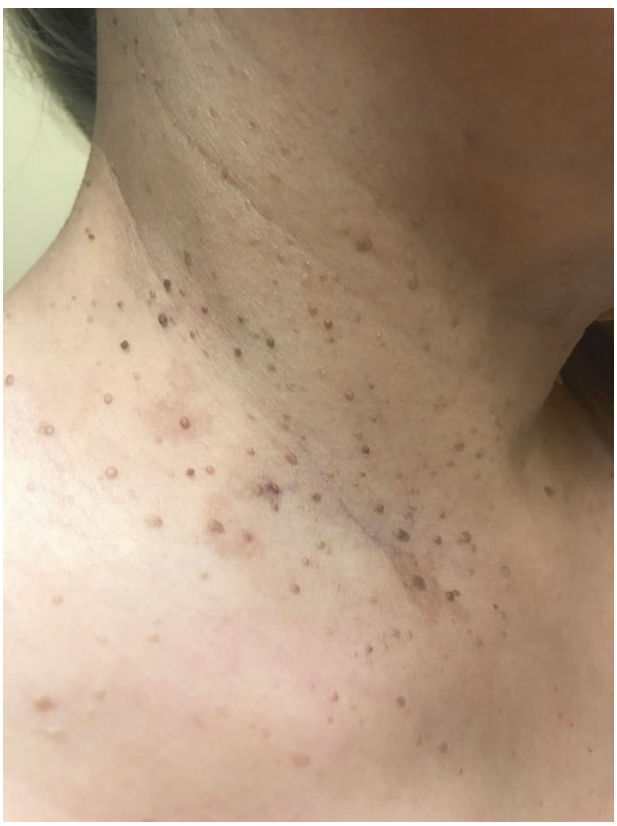

Fig. 2.238 DPN

Fig. 2.240 DPN

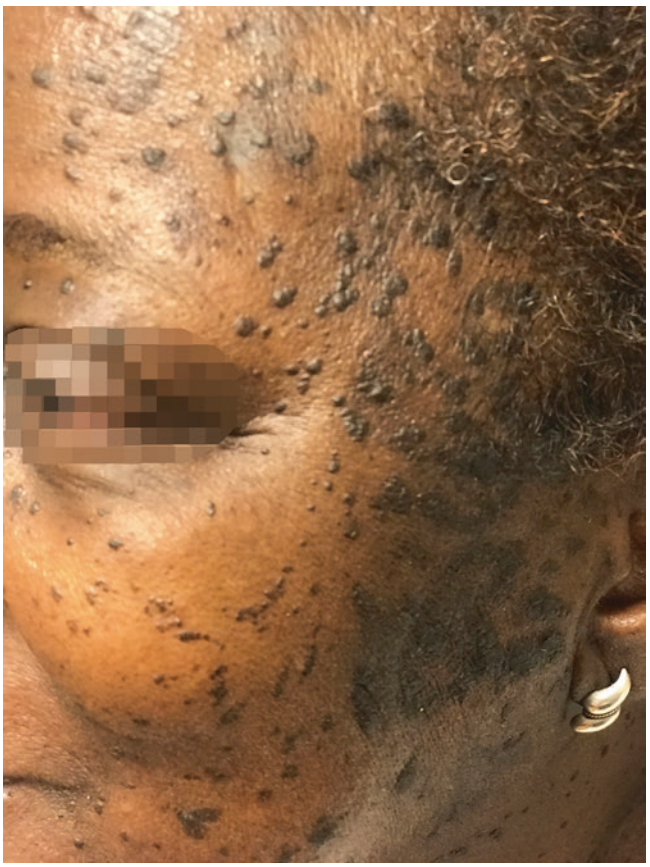

Fig. 2.241 DPN

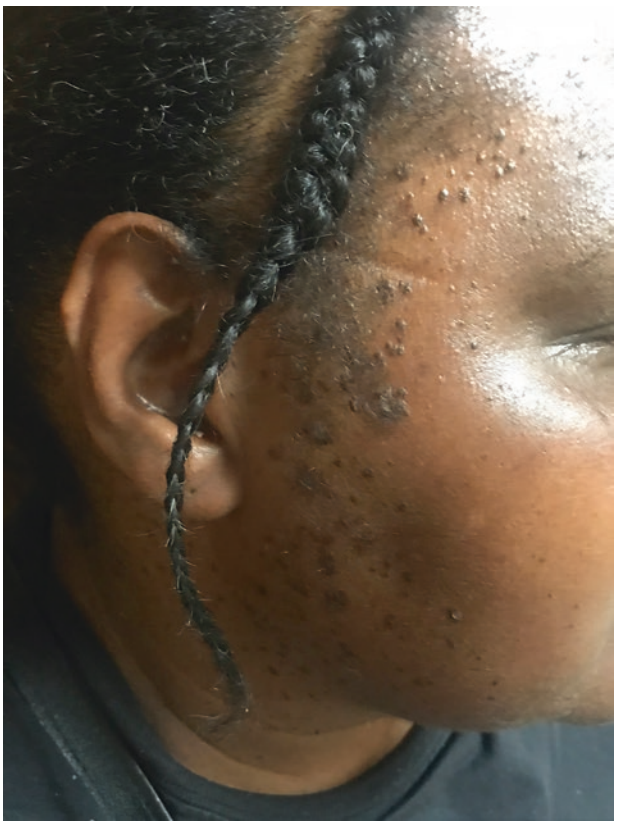

Fig. 2.243 DPN

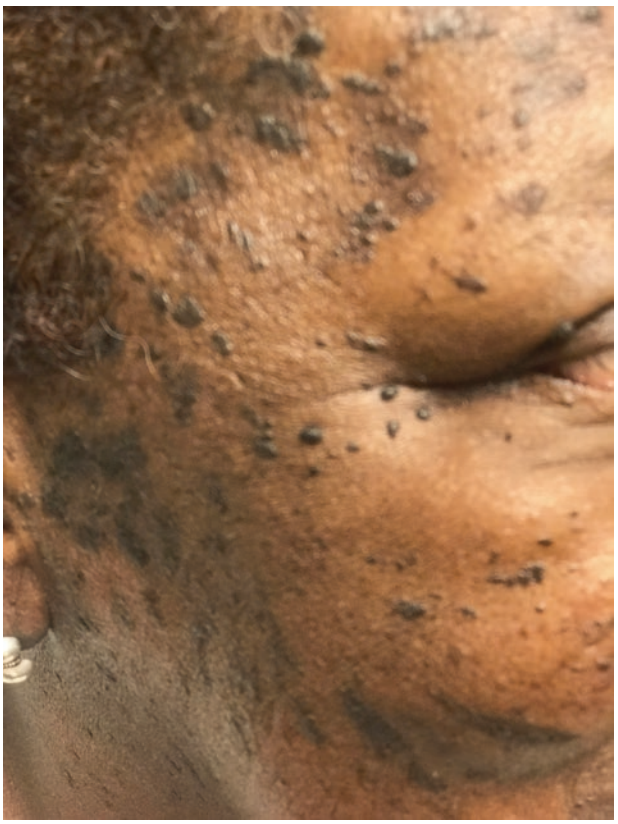

Fig. 2.242 DPN

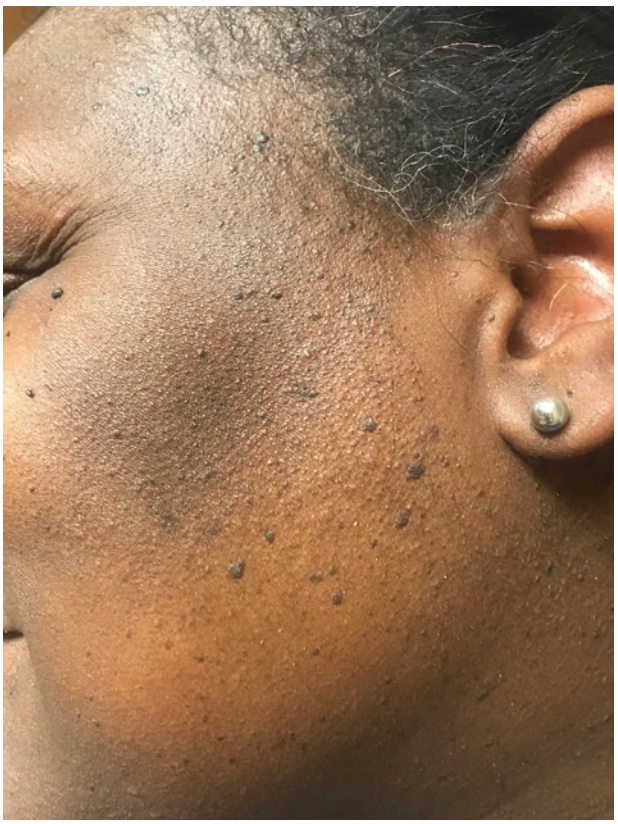

Fig. 2.244 DPN

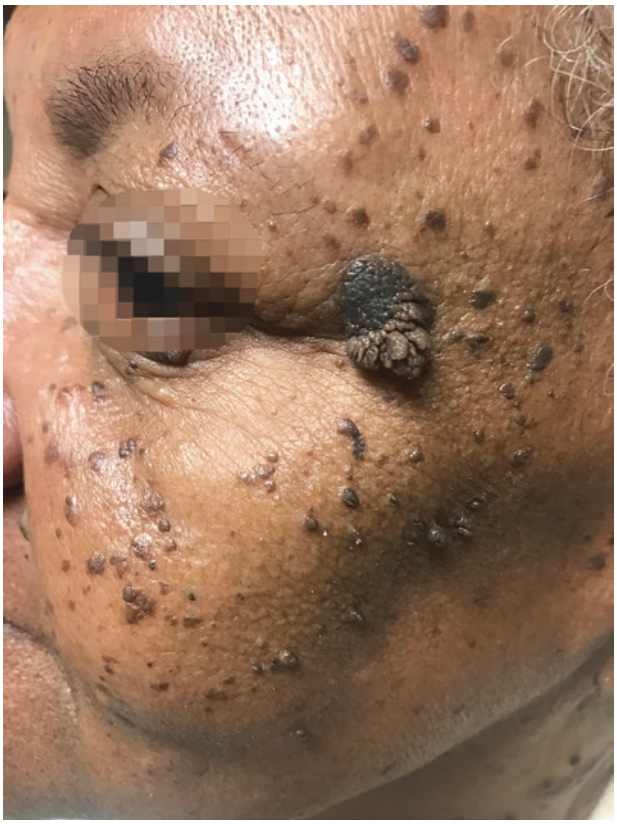

Fig. 2.245 DPN

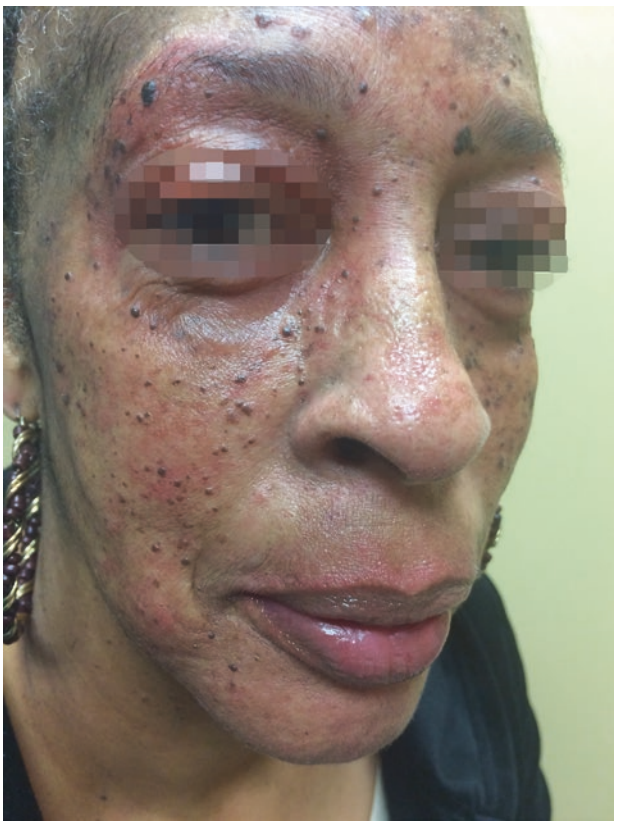

Fig. 2.246 DPN

2.8.2 Management

The main treatments for DPNs are destruction by electrodesiccation, removal by shaving, and cryotherapy. The treatment of large areas of DPNs can be challenging, and all the treatments can lead to hypopigmentation or hyperpigmentation, so we suggest testing a small number of DPNs prior to full treatments.

Suggested Reading

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