Immunological Diseases

4

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This chapter covers the diagnosis and treatment of immunologic diseases of the skin, some of which are more common in black skin. The diseases covered include systemic lupus erythematosus, photodermatoses, sarcoidosis, lichen sclerosis et atrophicus, dermatomyositis, and Sweet syndrome. The ability to diagnose these diseases with a keen eye can facilitate an appropriate therapeutic work-up of systemic symptoms or disease associations. Prompt and proper treatment of these diseases can improve cutaneous symptoms and also decrease systemic disease burden and morbidity.

4.1 Systemic Lupus Erythematosus

Systemic lupus erythematosus (SLE) is a systemic autoimmune disease of unknown etiology that is more common in women and African Americans. Cutaneous lupus erythematosus (CLE) is a common manifestation in patients with SLE. CLE may be the predominant symptom of SLE, or patients may have CLE alone.

CLE can be further subdivided into several subgroups:

- Acute cutaneous lupus erythematosus (ACLE), the most common cutaneous finding associated with SLE in black skin. ACLE is often induced by ultraviolet radiation.
- Subacute cutaneous lupus erythematosus (SCLE), which is rarely seen in black skin. It can be triggered by ultraviolet light or numerous medications, including thiazides, terbinafine, and ACE inhibitors.
- Chronic cutaneous lupus erythematosus (CCLE), which is more prevalent in black skin and results in scarring. This is also referred to as discoid lupus erythematosus (DLE) and is discussed below.
- Lupus profunda is a lobular panniculitis with scarring, which may be seen as a distinct entity or in association with DLE.

4.1.1 Presentation in Black Skin

ACLE presents with a "butterfly rash" characterized by symmetric erythema on the bilateral cheeks and nasal bridge but sparing the nasolabial folds (Figs. 4.1–4.4). It can spread to involve the forehead or ears and may be associated with facial edema. This rash usually heals without scarring. ACLE may also present in a more generalized fashion with a maculo-urticarial or papular eruption involving the extensor arms and dorsal hands, sparing the knuckles. Similar lesions may be present on the trunk.

SCLE presents with erythematous macules that develop into papulo-squamous or annular plaques. This eruption is photosensitive and classically involves exposed areas of the chest (Fig. 4.5), shoulders, and upper back. Facial involvement is uncommon. SCLE does not usually resolve with scarring, but hypopigmentation and telangiectasias may persist for months.

Lupus profunda presents with tender subcutaneous nodules that heal with atrophy. The proximal extremities, breasts, buttocks, and back are favored (Figs. 4.6–4.11).

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Fig. 4.1 Malar rash

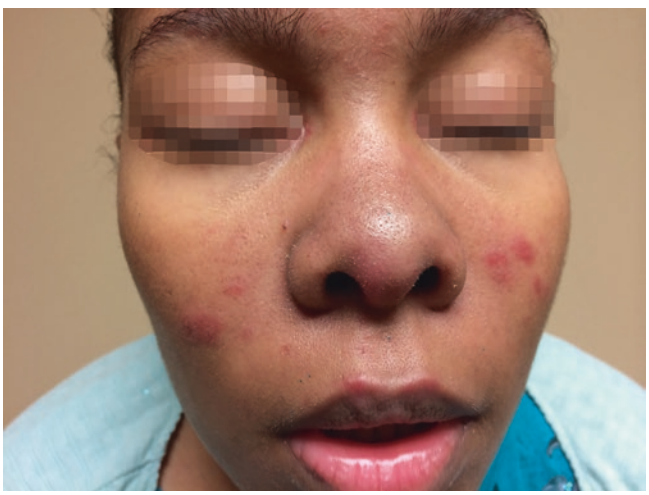


Fig. 4.2 Malar rash

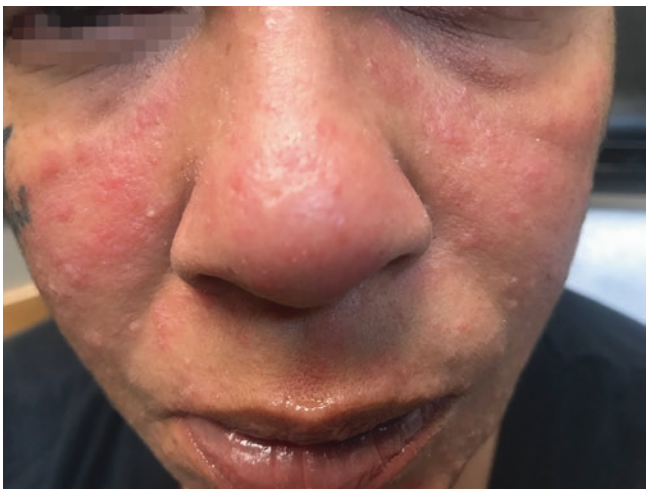


Fig. 4.3 Malar rash

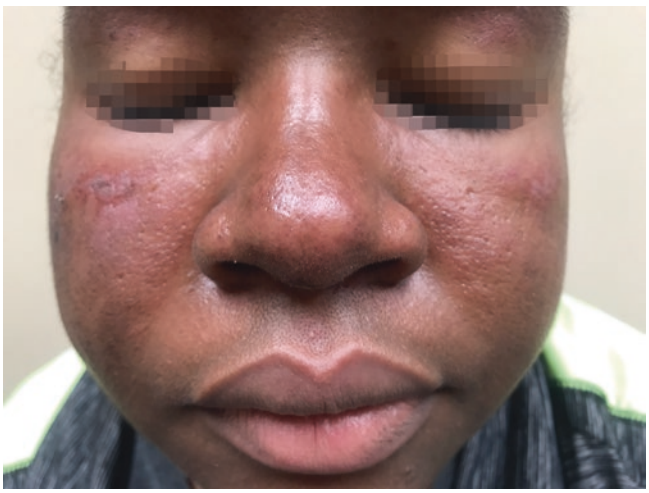


Fig. 4.4 Malar rash

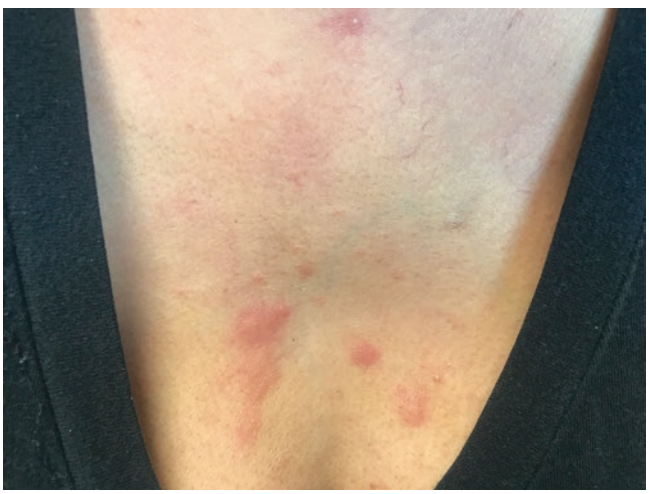


Fig. 4.5 Subacute cutaneous lupus erythematosus (SCLE)

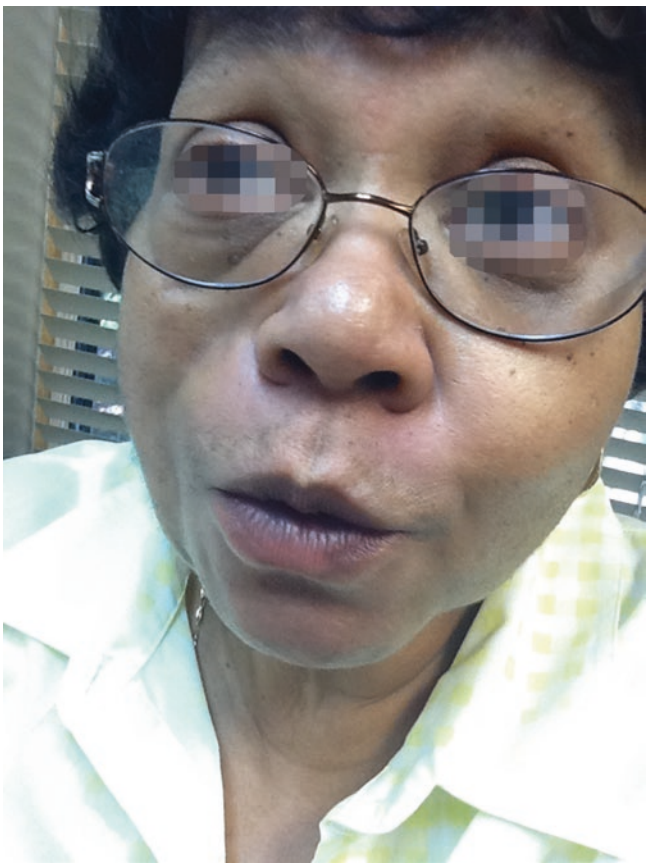


Fig. 4.6 Lupus profunda, right face

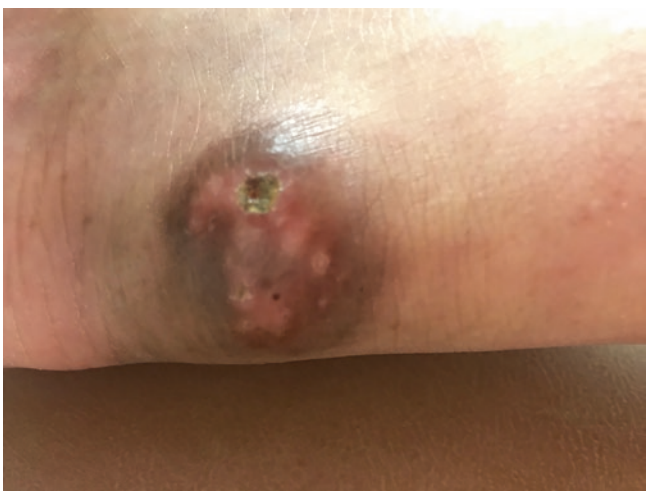


Fig. 4.7 Lupus profunda, arm

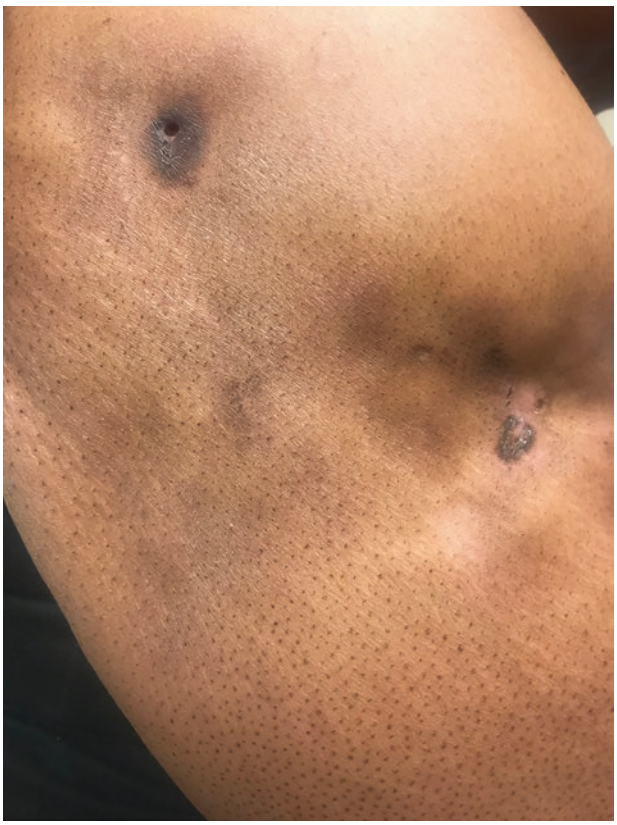


Fig. 4.8 Lupus profunda, back

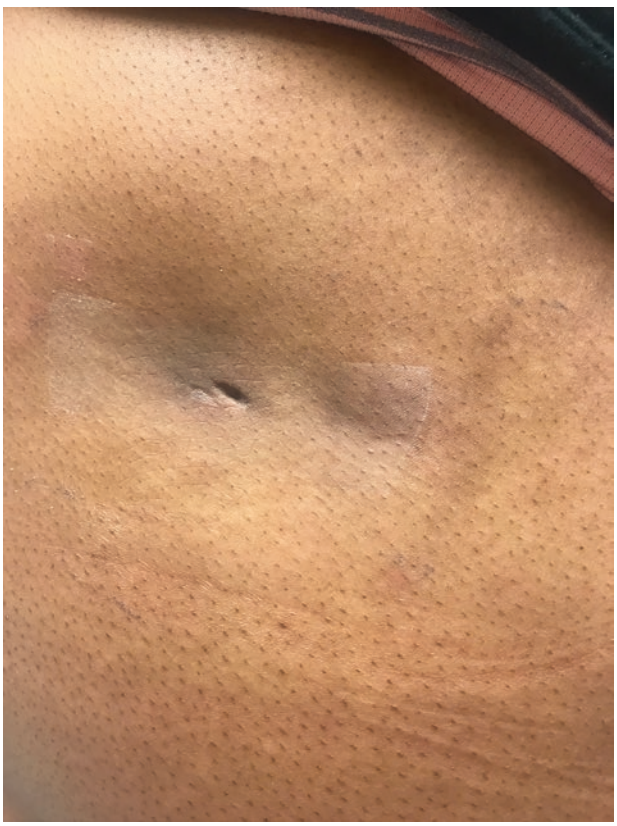


Fig. 4.9 Lupus profunda, back

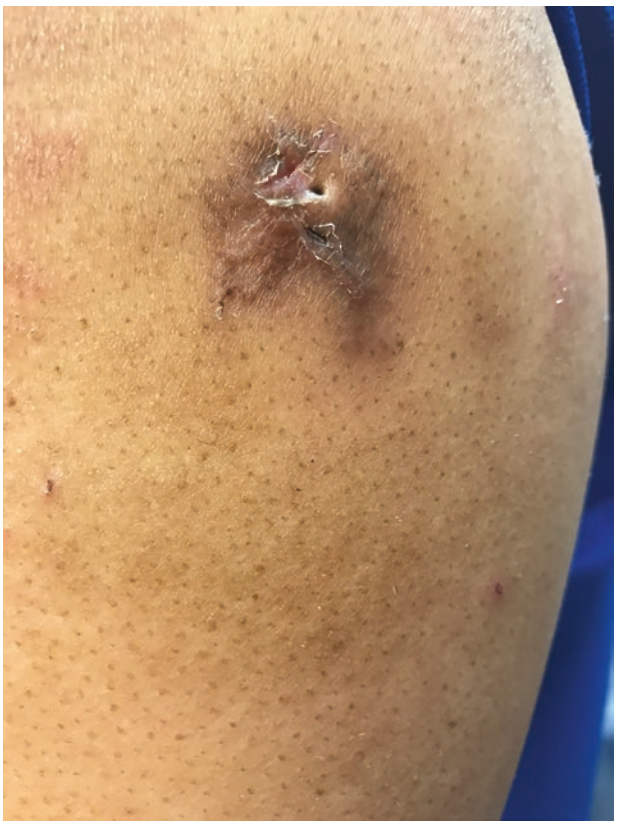


Fig. 4.10 Lupus profunda, upper arm

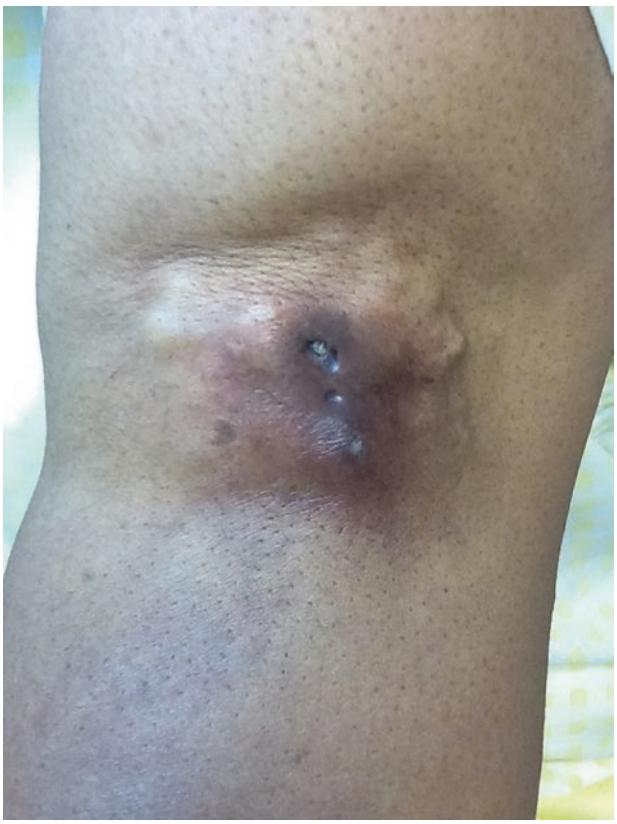


Fig. 4.11 Lupus profunda, upper arm

4.1.2 Treatment and Management

Oral antimalarials are the treatment of choice for patients with both systemic and cutaneous lupus. Treatment of systemic lupus may improve the cutaneous symptoms. High-potency topical corticosteroids usually improve cutaneous disease. Strict photoprotection is advised. Oral steroids or other immunosuppressive medications may be used in recalcitrant cases.

4.2 Discoid Lupus Erythematosus

Discoid lupus erythematosus (DLE) is the most common form of chronic cutaneous lupus erythematosus (CCLE). Most patients with DLE do not have systemic symptoms of SLE. The majority of DLE patients are black females. Men who are affected will have a close female relative who also has DLE, which hints to an X-linked nature of the gene. DLE is usually localized to the face, ears, and scalp, although in a minority of patients it is also seen below the neck, a presentation associated with an increased risk of progression to SLE.

4.2.1 Presentation in Black Skin

Discoid lupus presents with coin-shaped, erythematous plaques with hyperkeratosis. As lesions expand, hyperpigmented borders and central atrophy often develop. The face, scalp, ears, neck, and extensor arms are most commonly affected. The "carpet tack sign" refers to the keratotic spikes often observed on the inferior surface of hyperkeratotic plaques. DLE heals with scarring and may lead to scarring alopecia in the scalp, especially in black skin (Figs. 4.12–4.64).

DLE is mostly observed in the head and neck, but it may be generalized, all over the body. If there is a single lesion that one suspects of being DLE, one can look at the external ear for follicular plugging, which resembles open comedones (Figs. 4.65–4.68).

Unique presentations of DLE in black skin include linear DLE and hypertrophic DLE. Linear DLE follows the lines of Blaschko; it can happen anywhere, but usually we encounter it in the upper or lower extremities (Figs. 4.69–4.73). Hypertrophic DLE can resemble lichen planus and usually happens on the legs (Figs. 4.74–4.76).

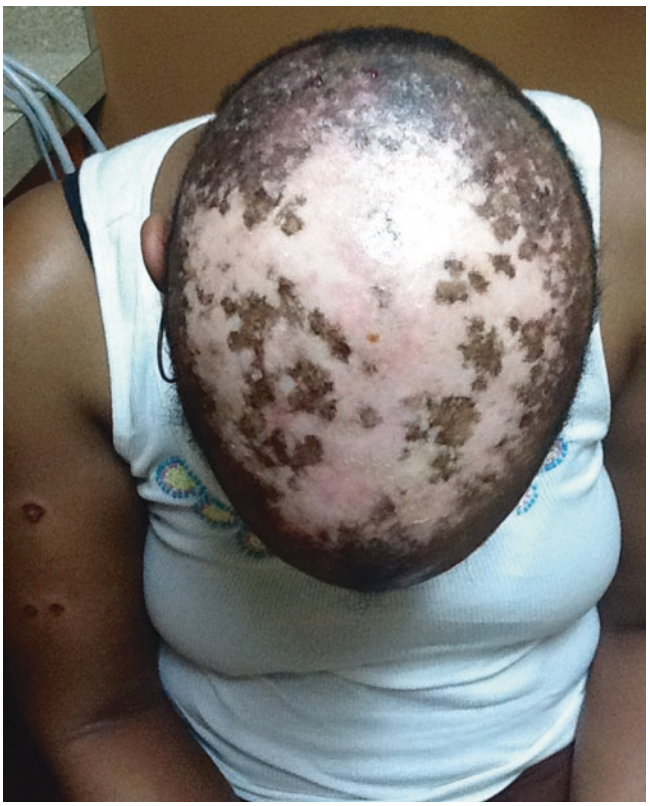


Fig. 4.12 Discoid lupus erythematosus (DLE)

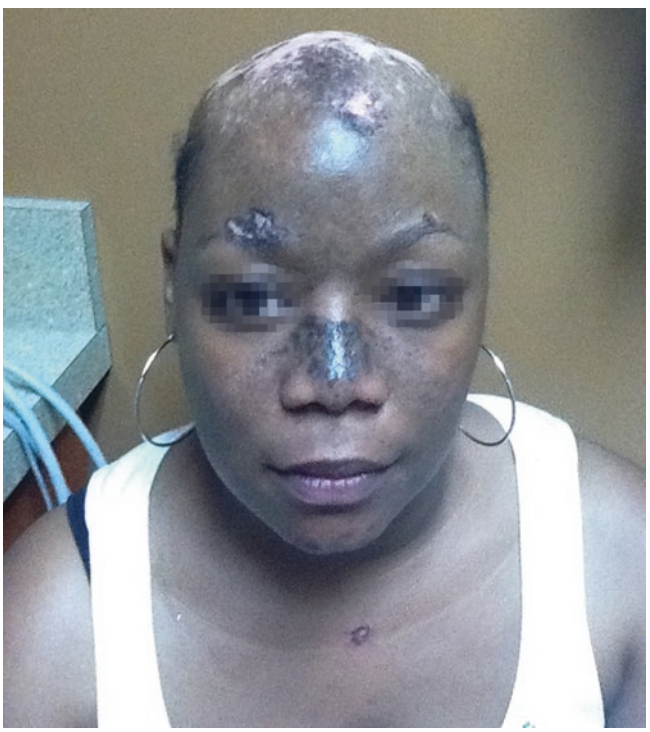


Fig. 4.13 DLE

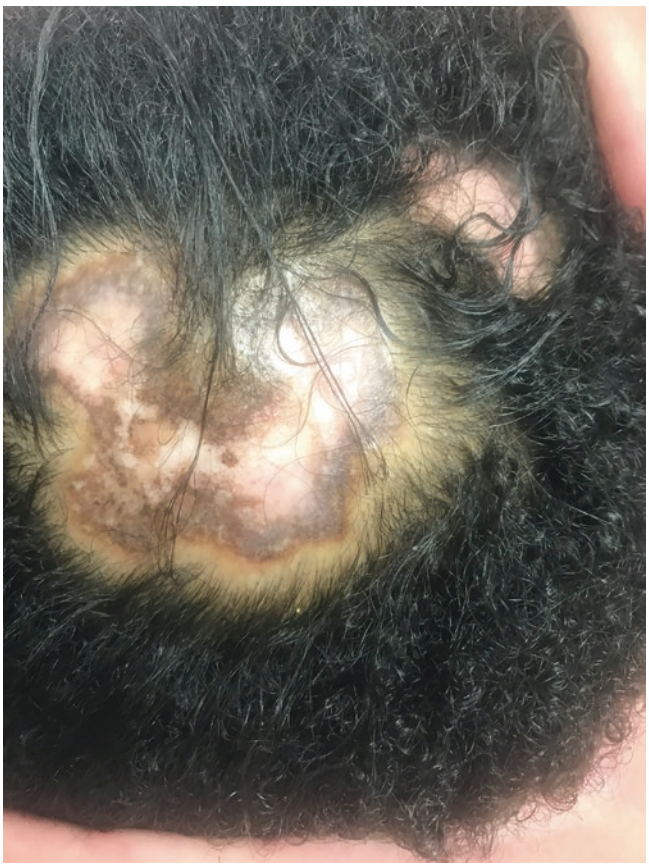


Fig. 4.14 DLE

Fig. 4.15 DLE

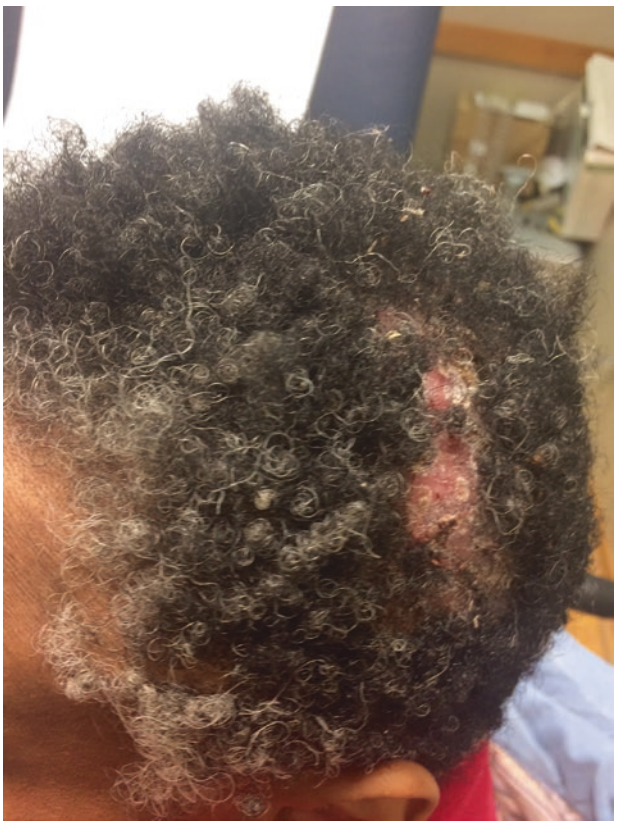


Fig. 4.16 DLE

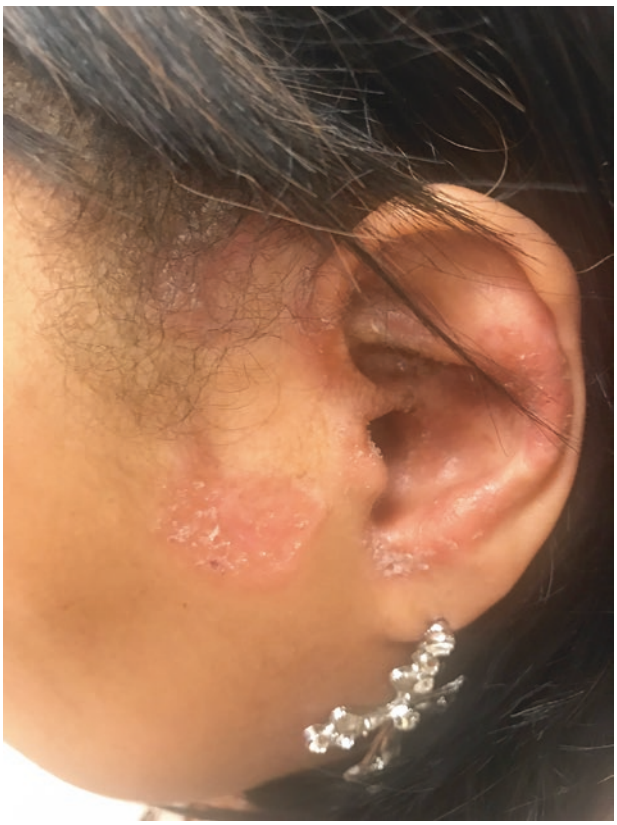


Fig. 4.18 DLE

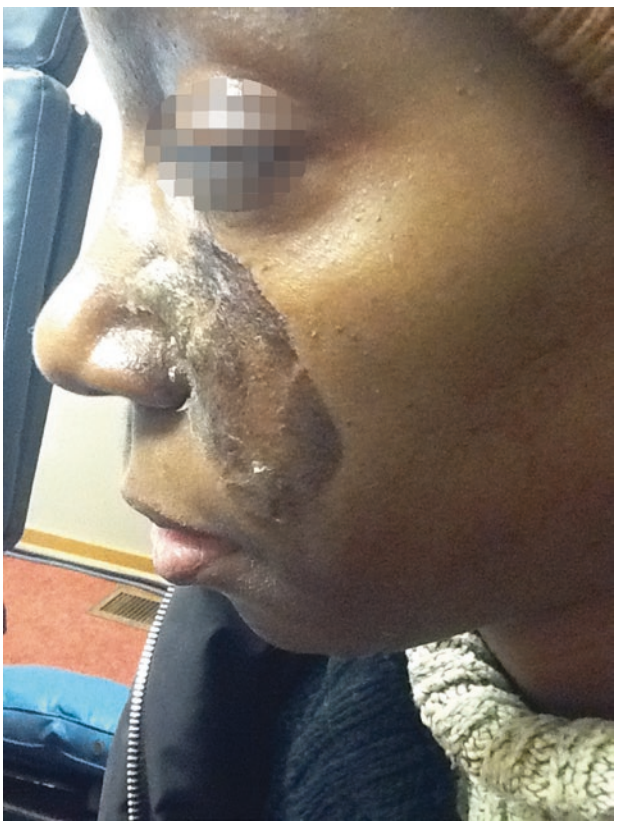


Fig. 4.17 DLE

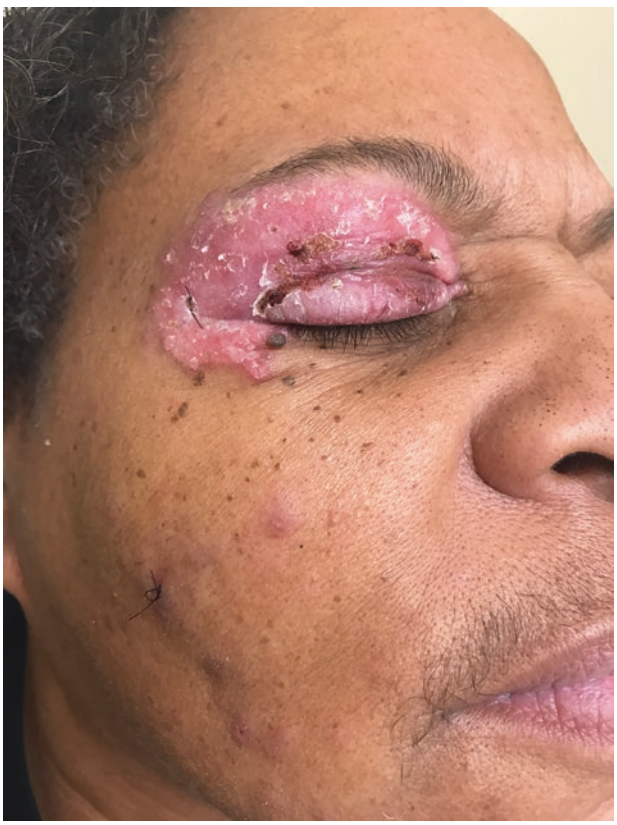
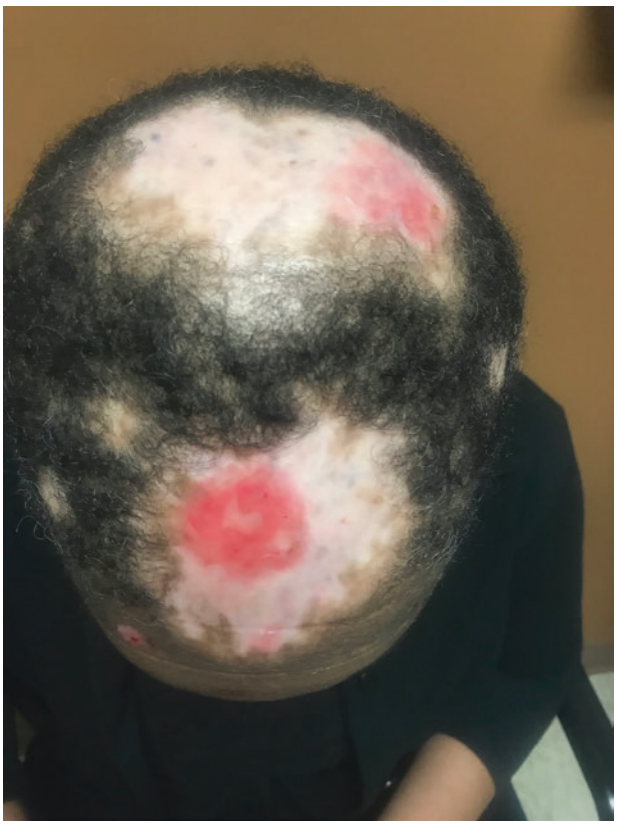


Fig. 4.19 DLE



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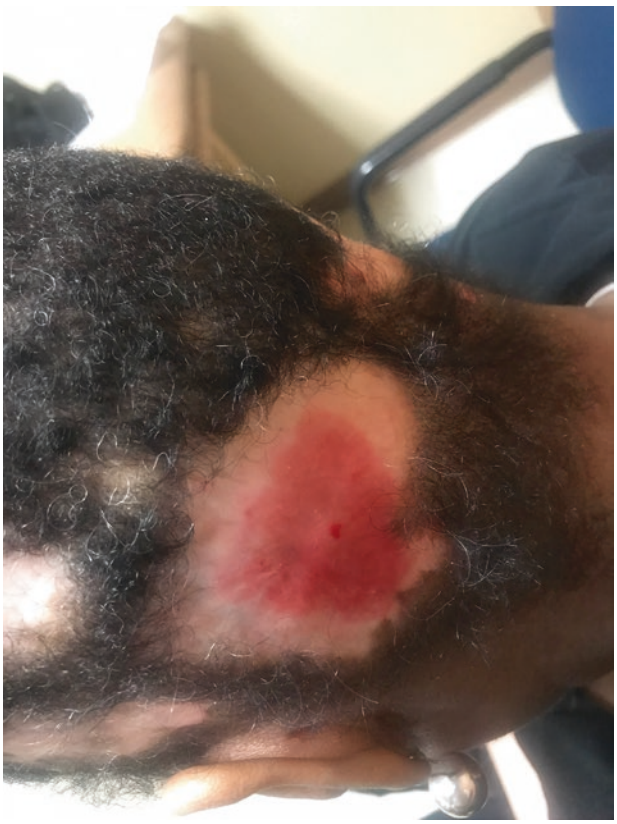

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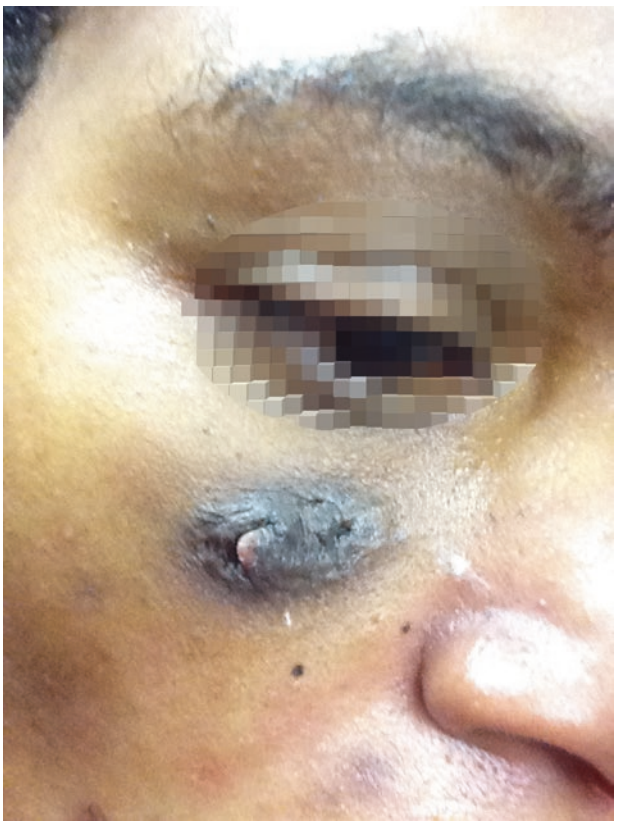

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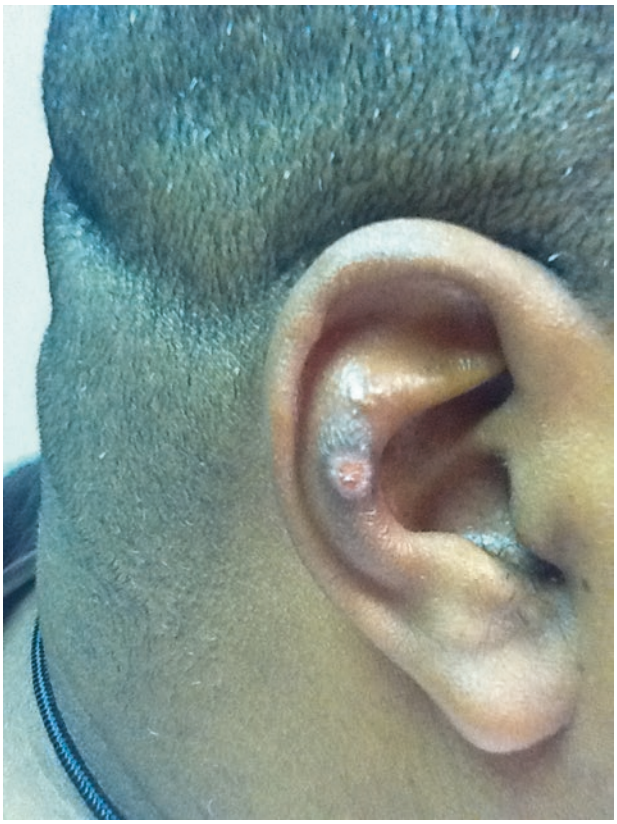

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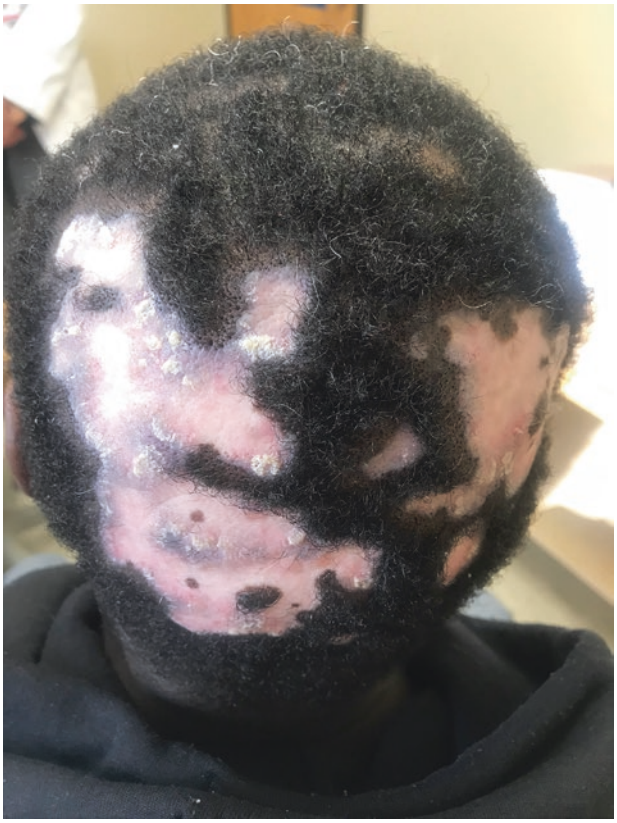

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Fig. 4.26 DLE

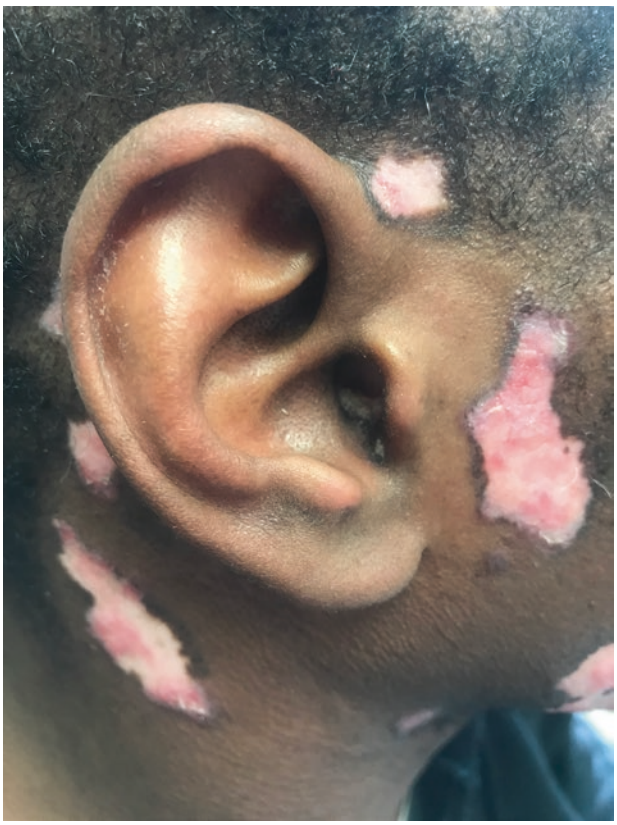

Fig. 4.25 DLE

Fig. 4.27 DLE

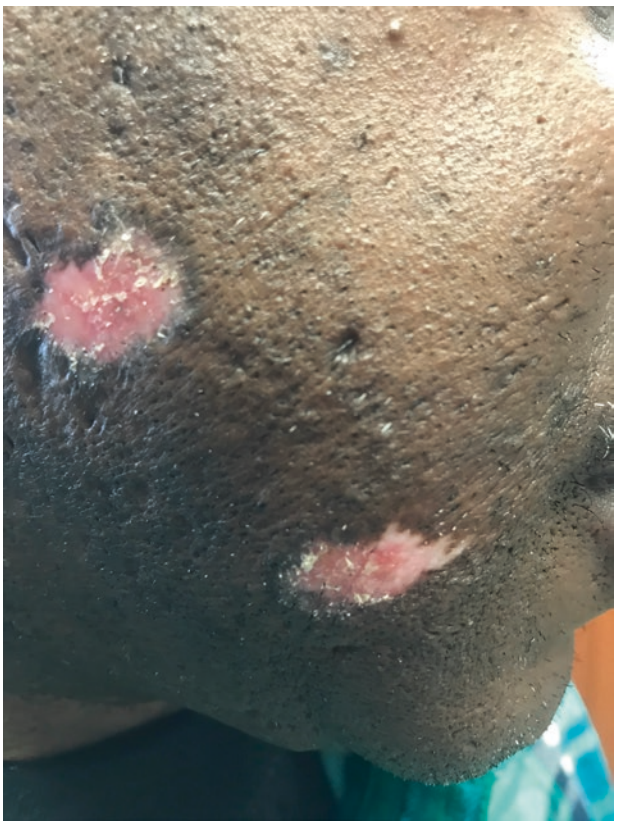

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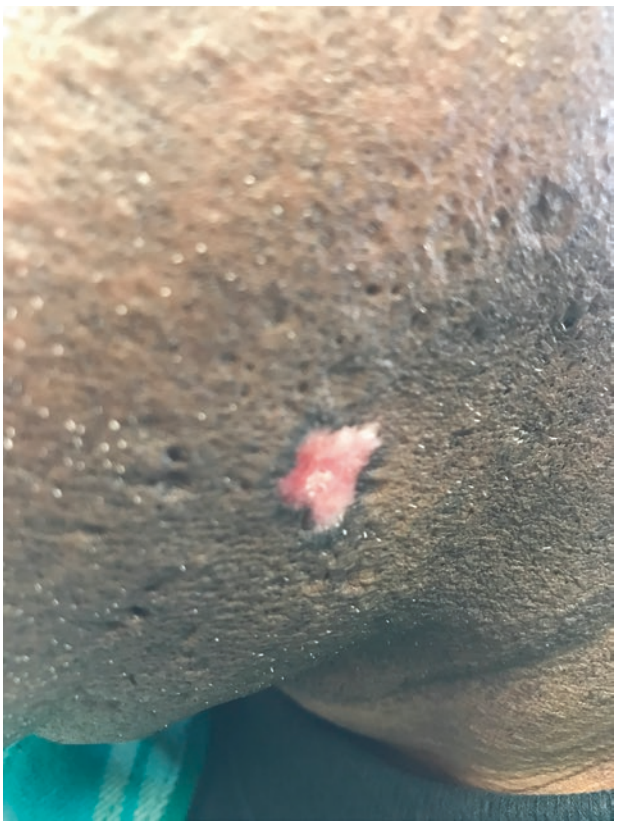
Fig. 4.30 DLE



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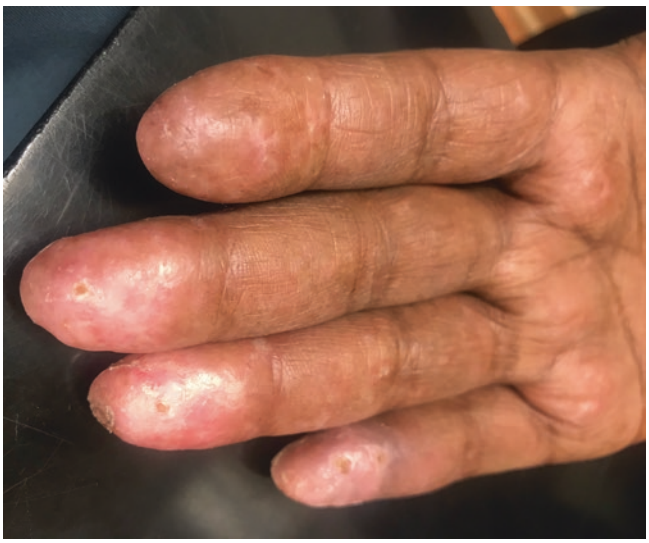
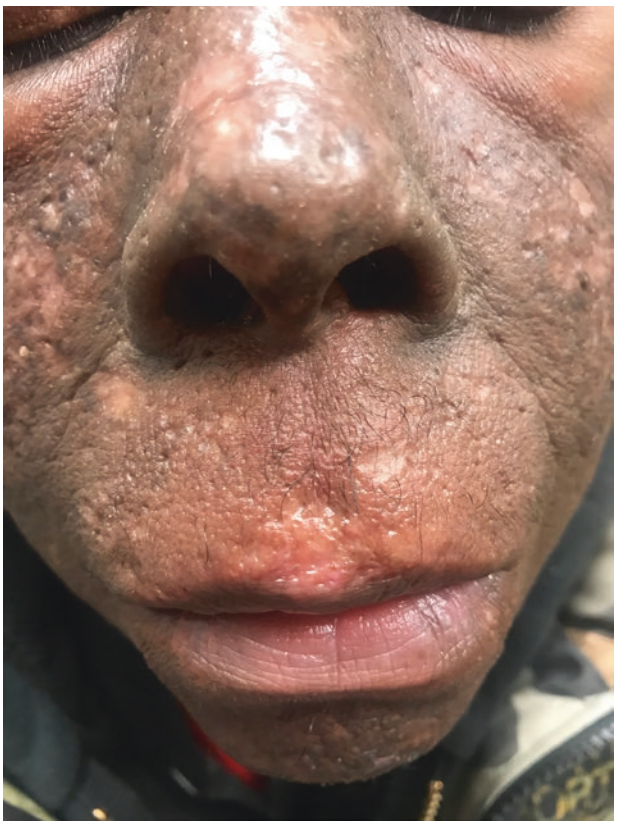


Fig. 4.31 DLE



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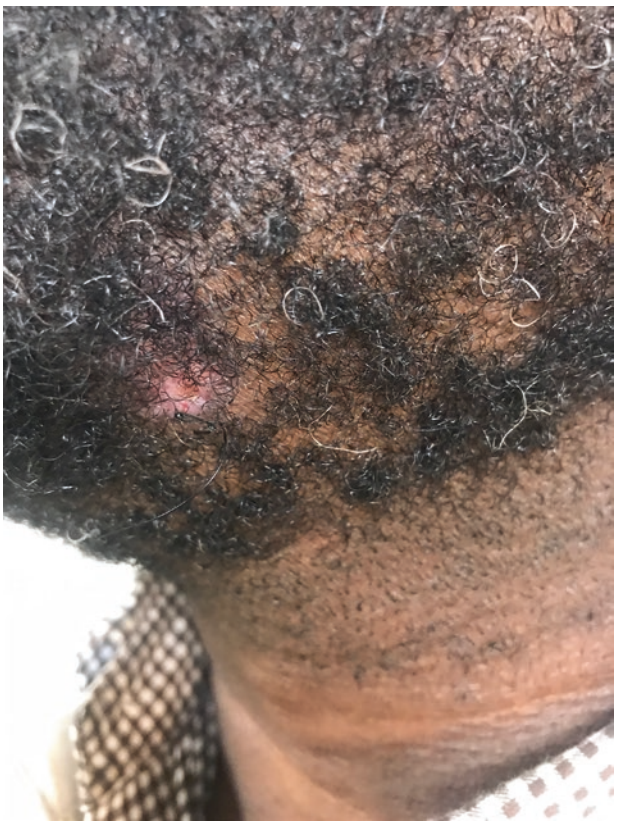

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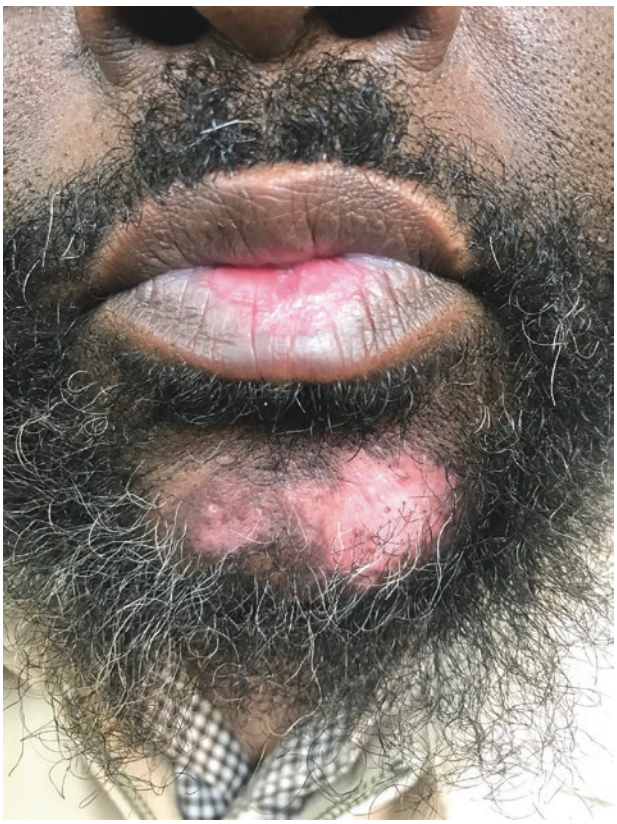


Fig. 4.33 DLE

Fig. 4.35 DLE, before treatment

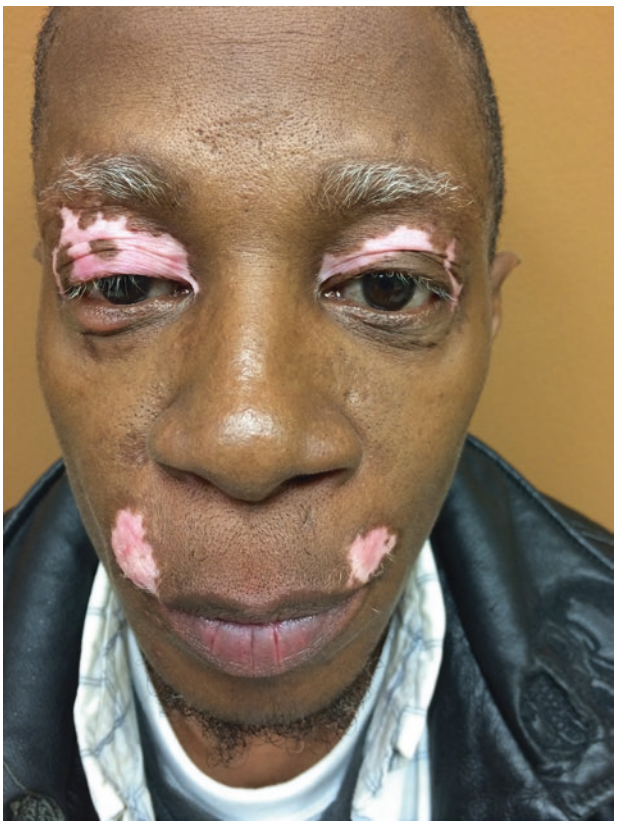


Fig. 4.36 DLE, same patient as Fig. 4.35 after treatment

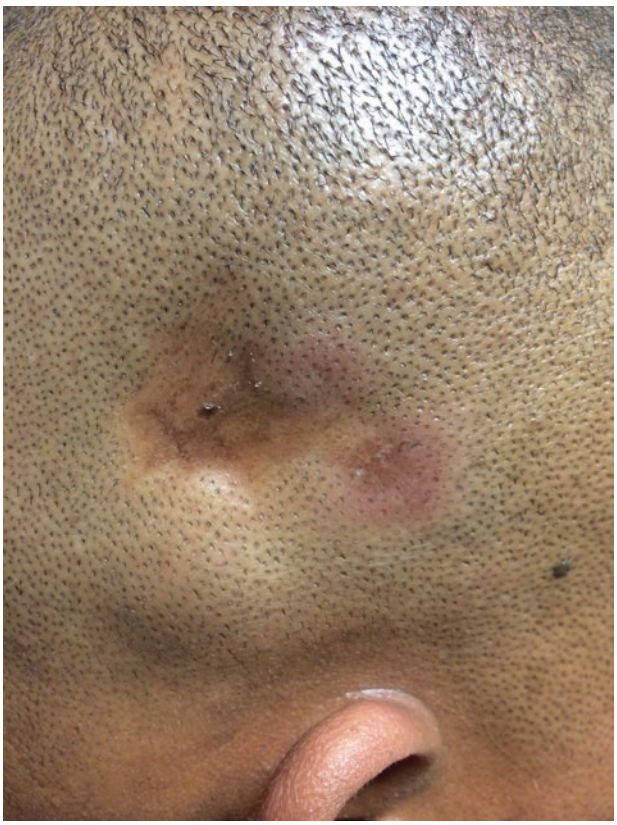
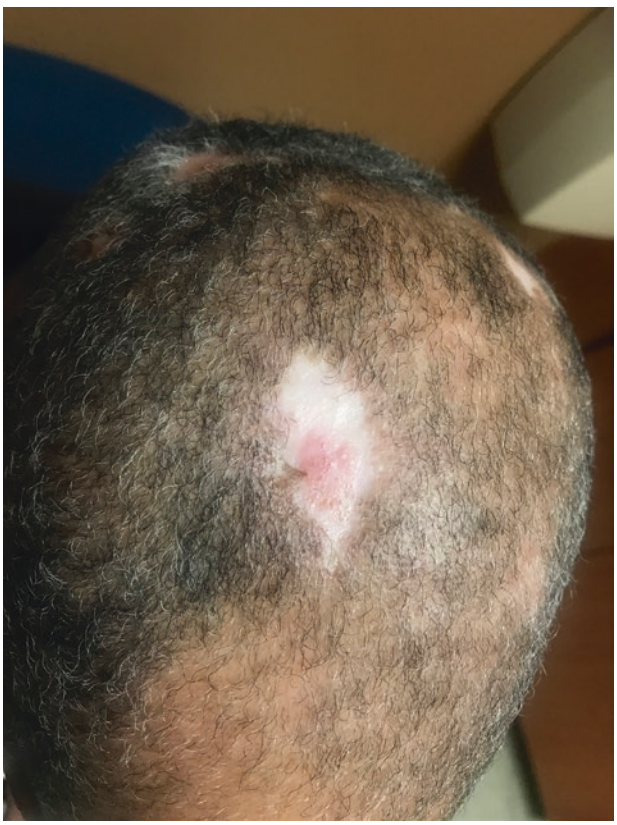


Fig. 4.38 DLE, with atrophy due to intralesional steroid



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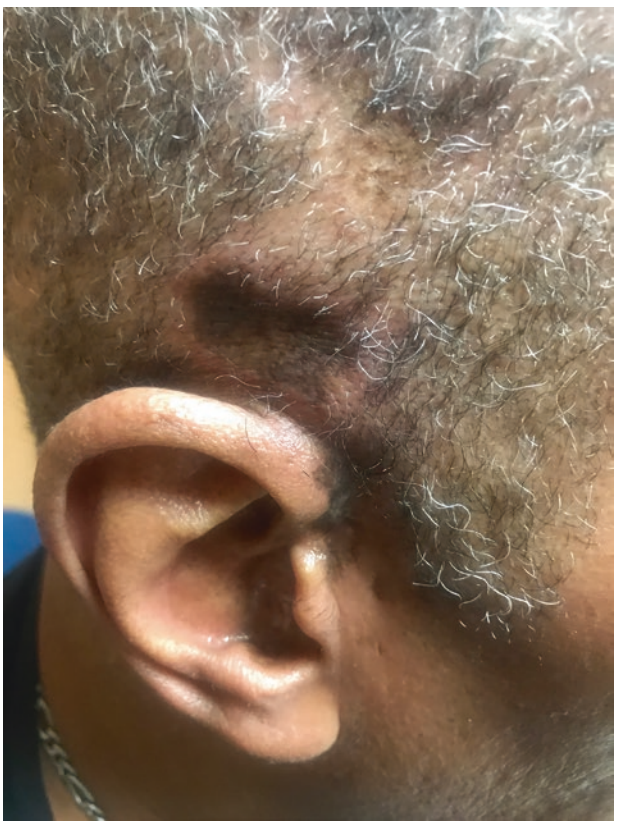

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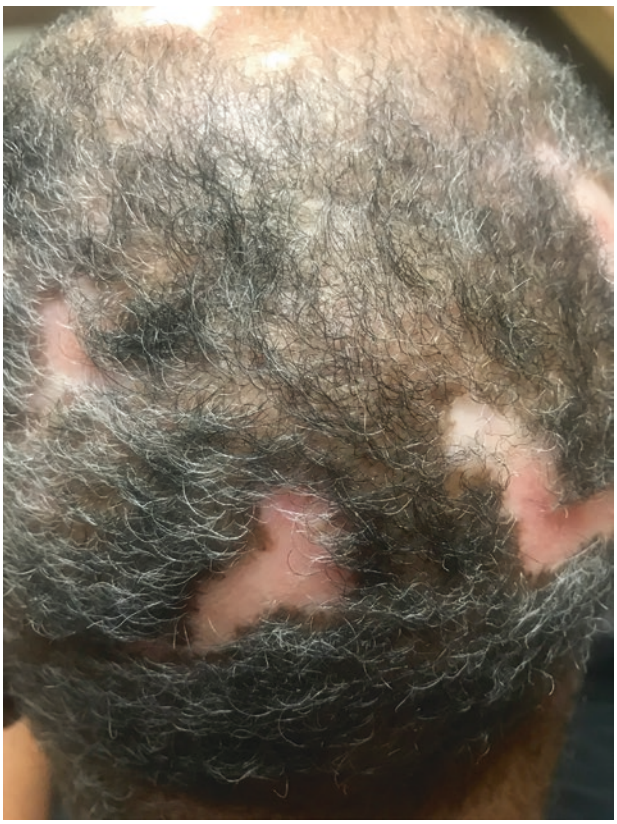

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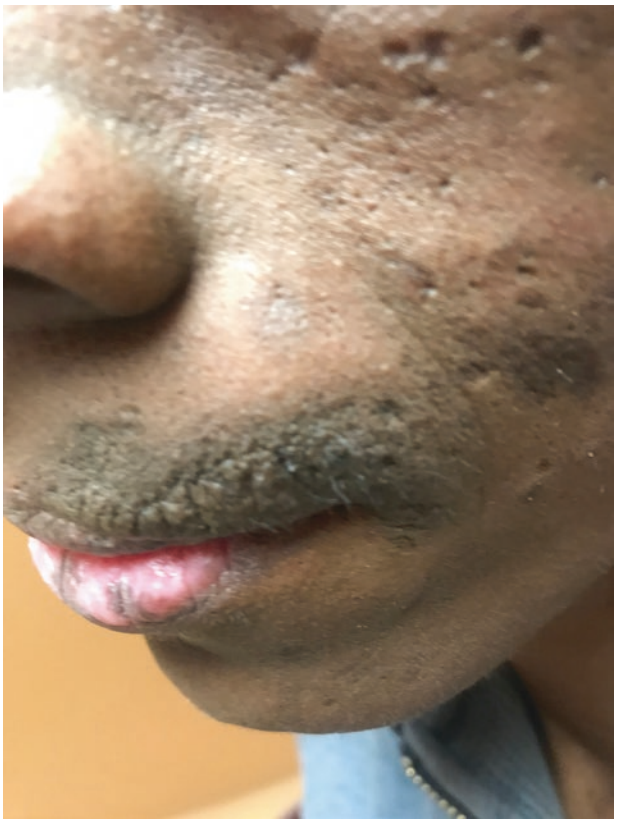

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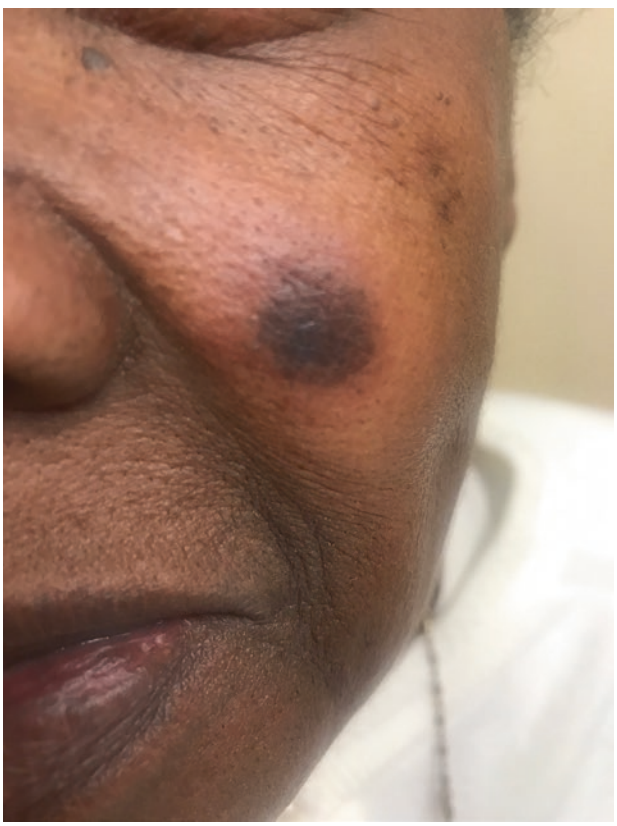

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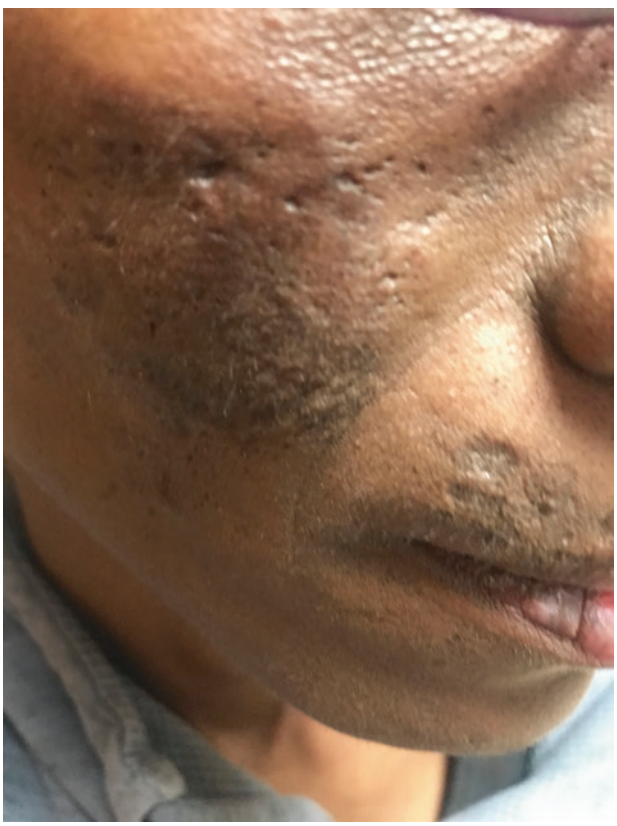

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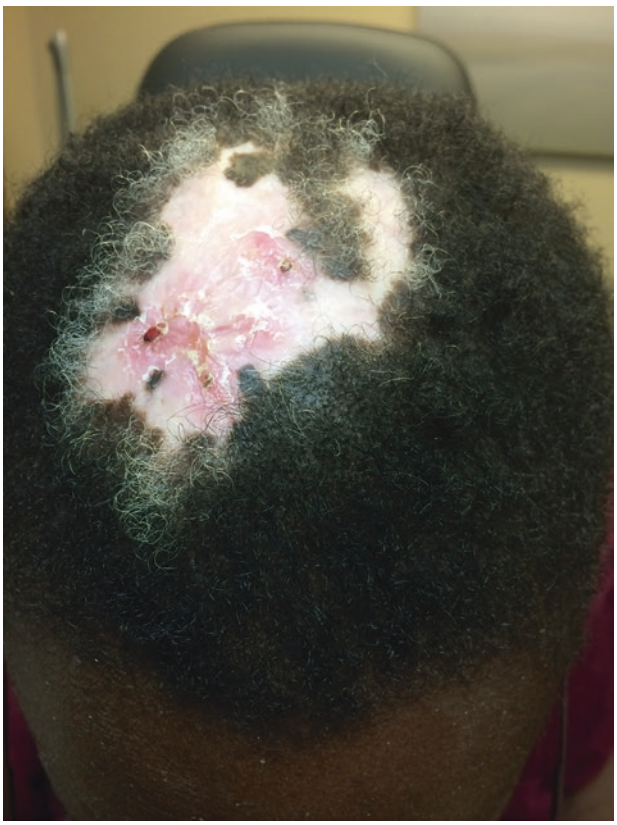
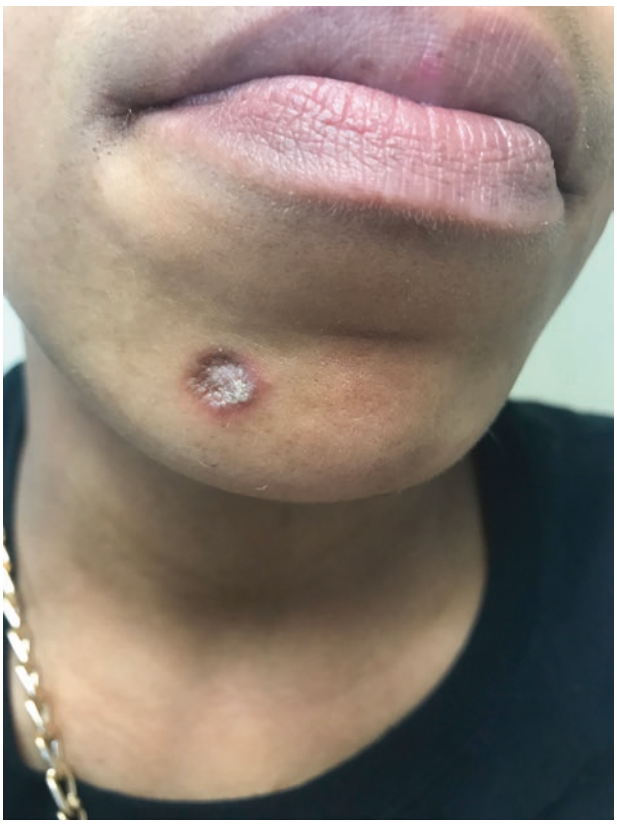


Fig. 4.44 DLE

Fig. 4.46 DLE

Fig. 4.45 DLE



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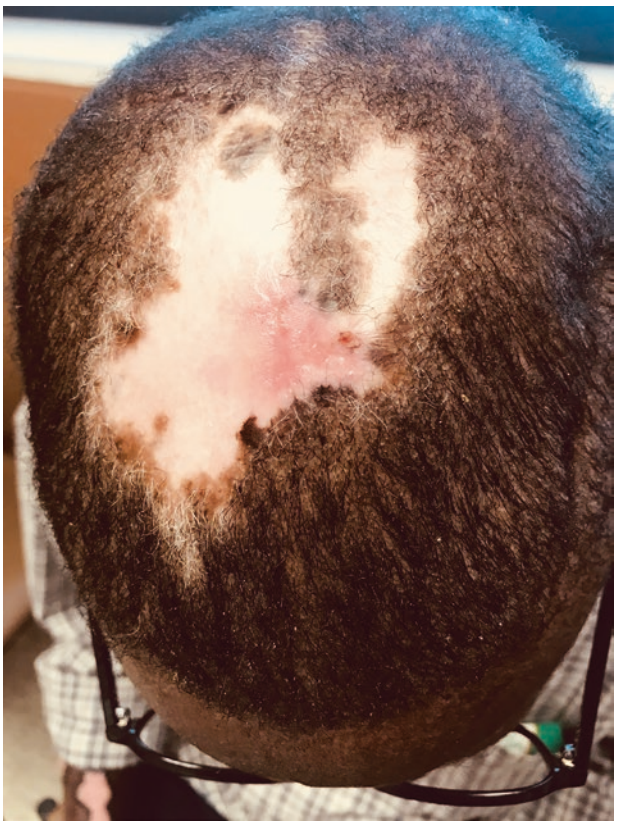

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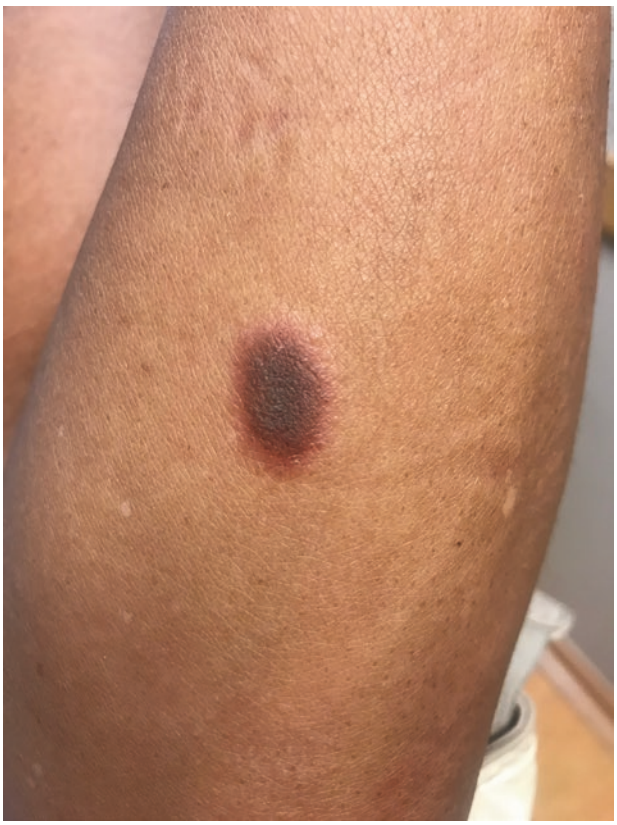

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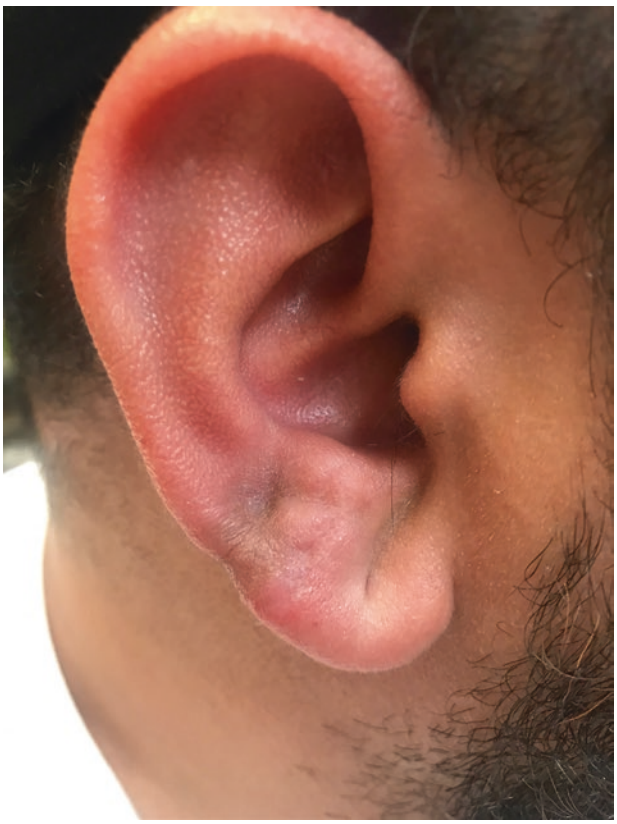

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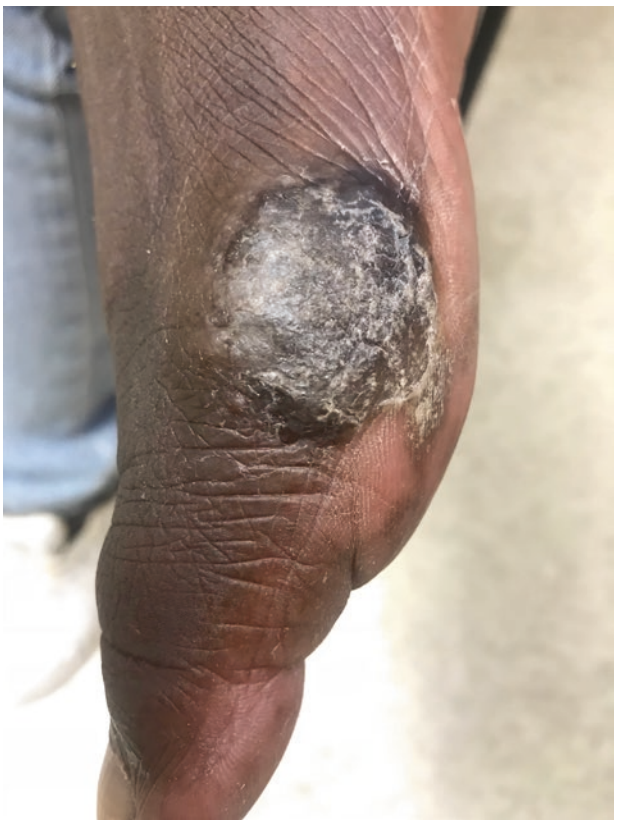

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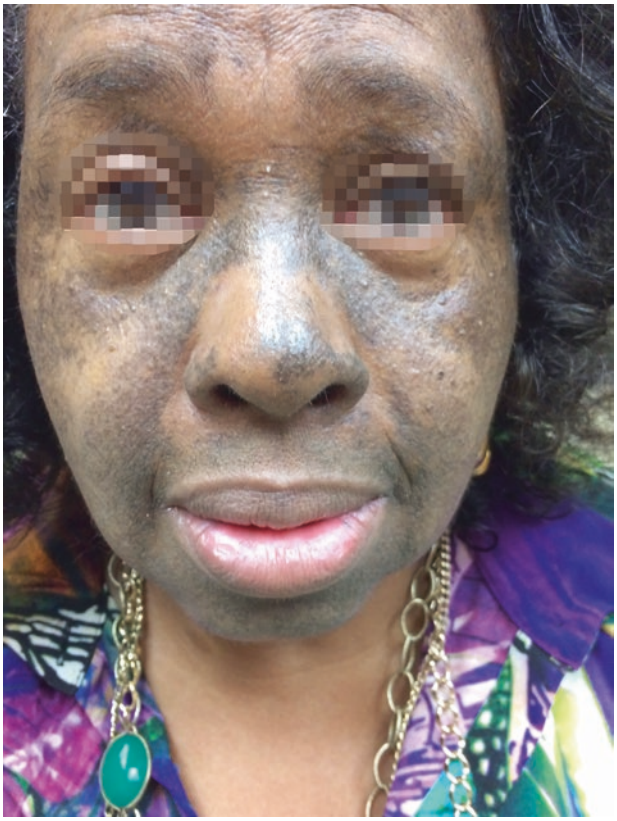
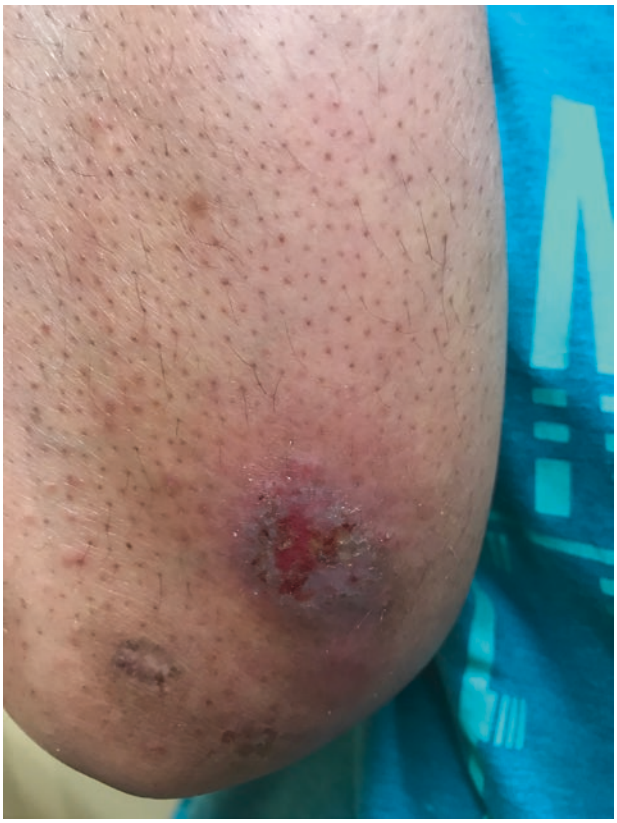


Fig. 4.52 DLE, before treatment

Fig. 4.54 DLE

Fig. 4.53 DLE, same patient as Fig. 4.52 after treatment



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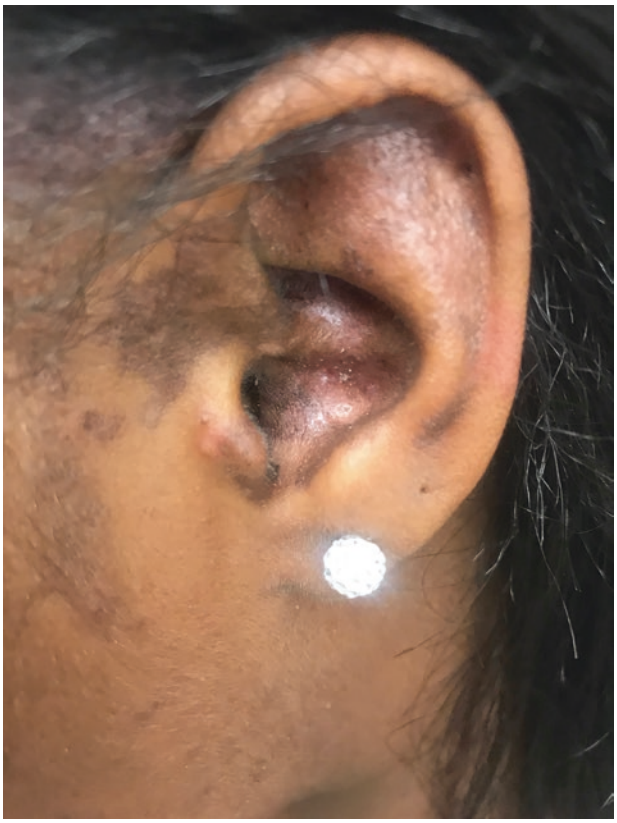

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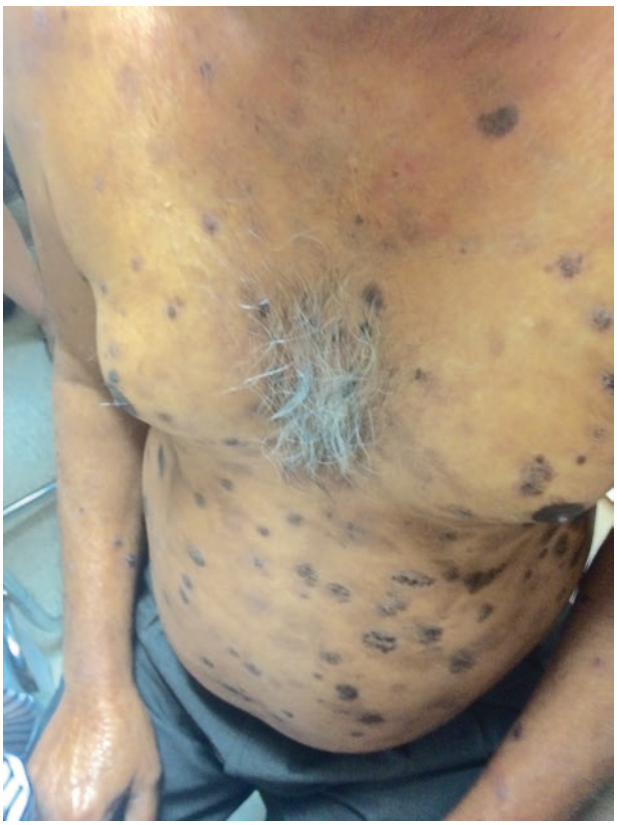


Fig. 4.58 DLE

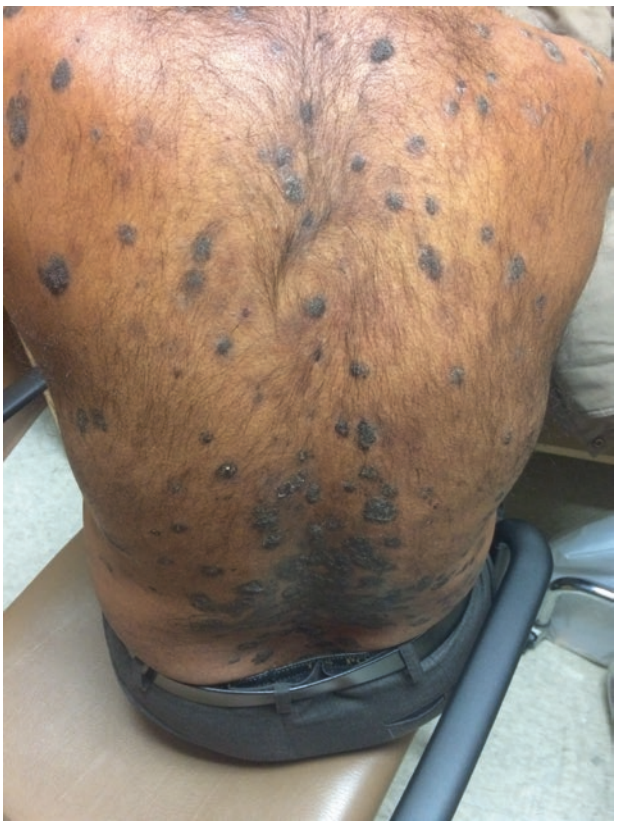


Fig. 4.57 DLE

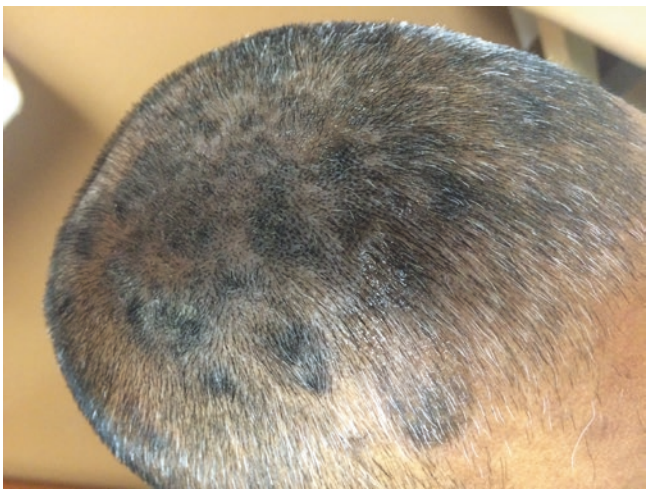


Fig. 4.59 DLE

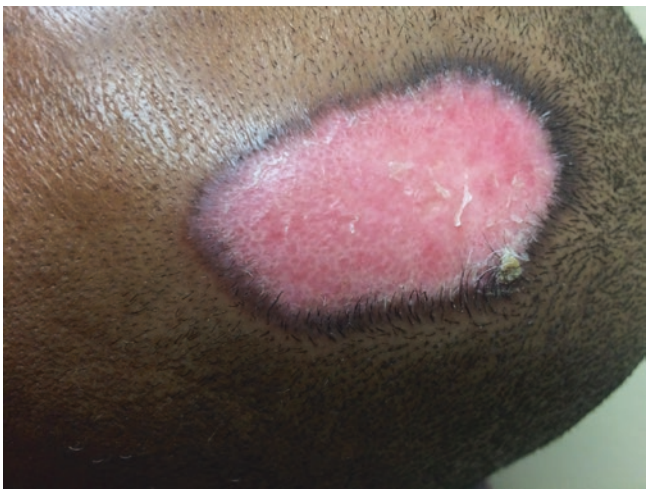
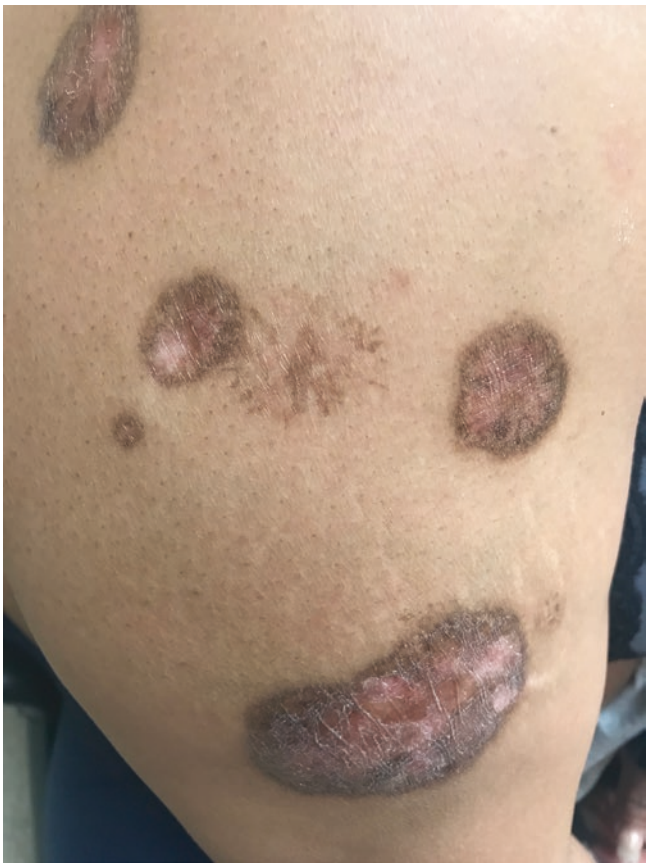


Fig. 4.60 DLE



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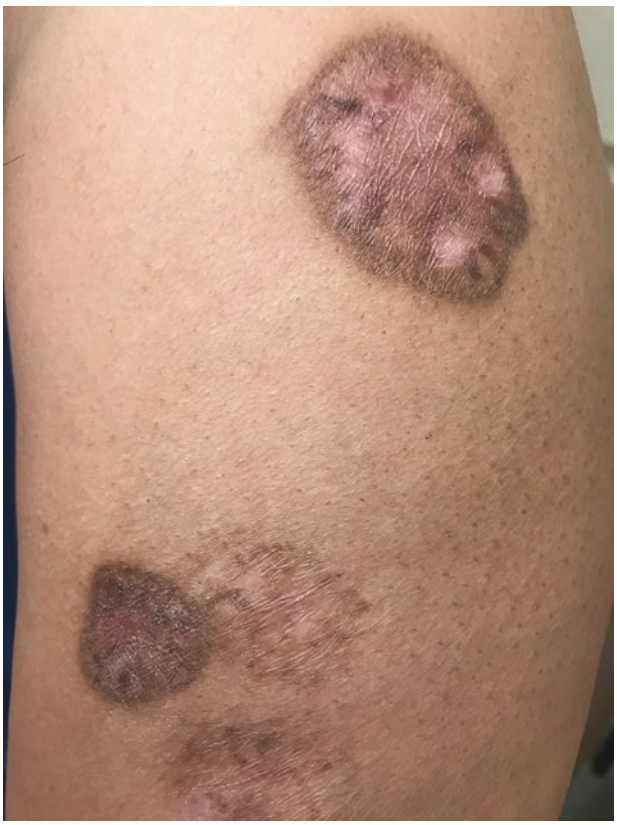

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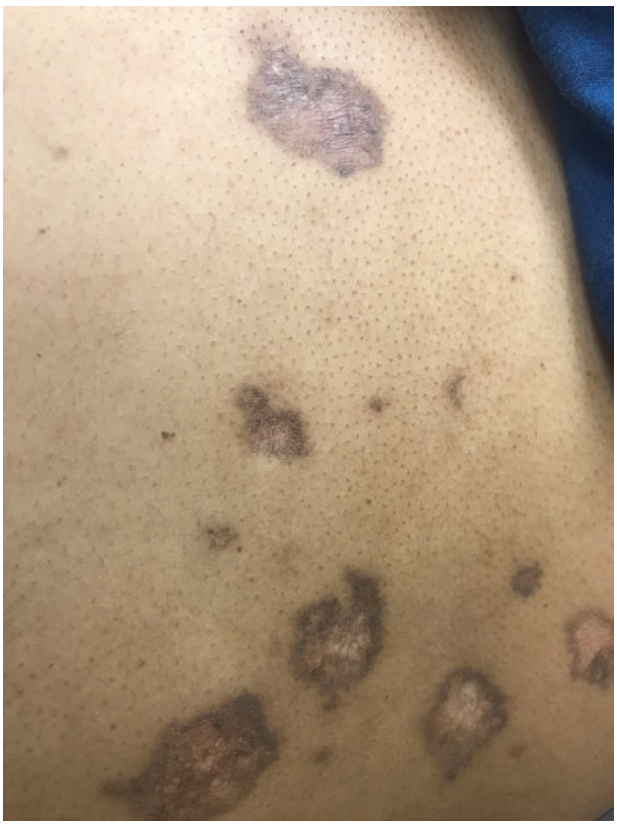

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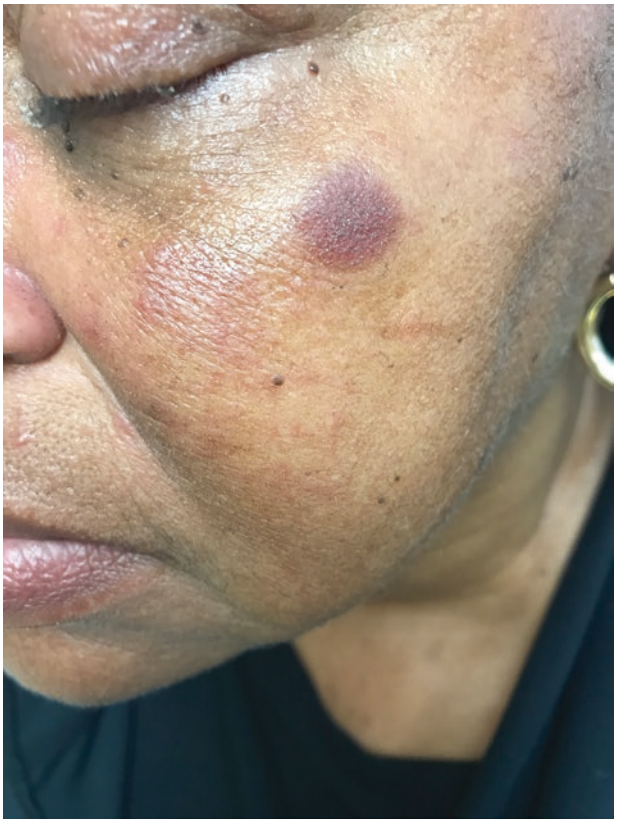

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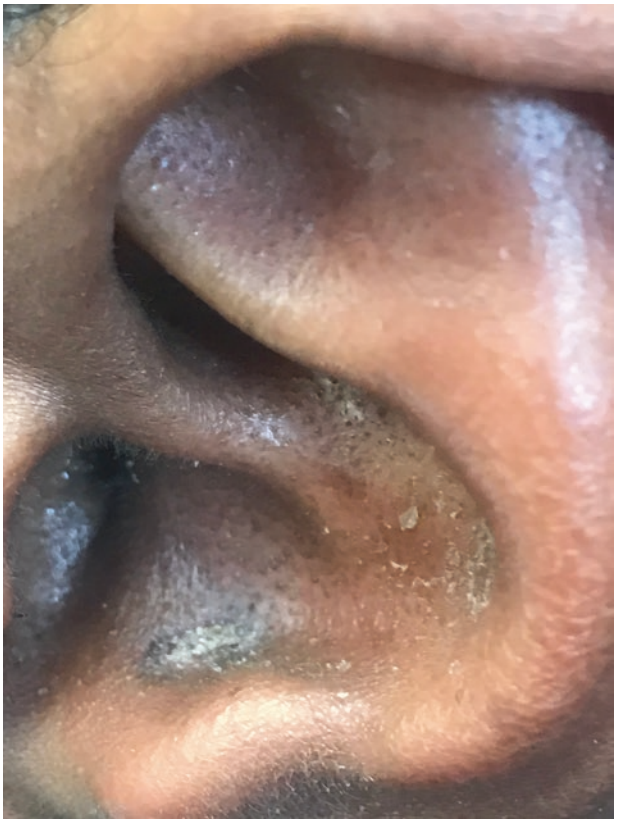

Fig. 4.66 Follicular plugging

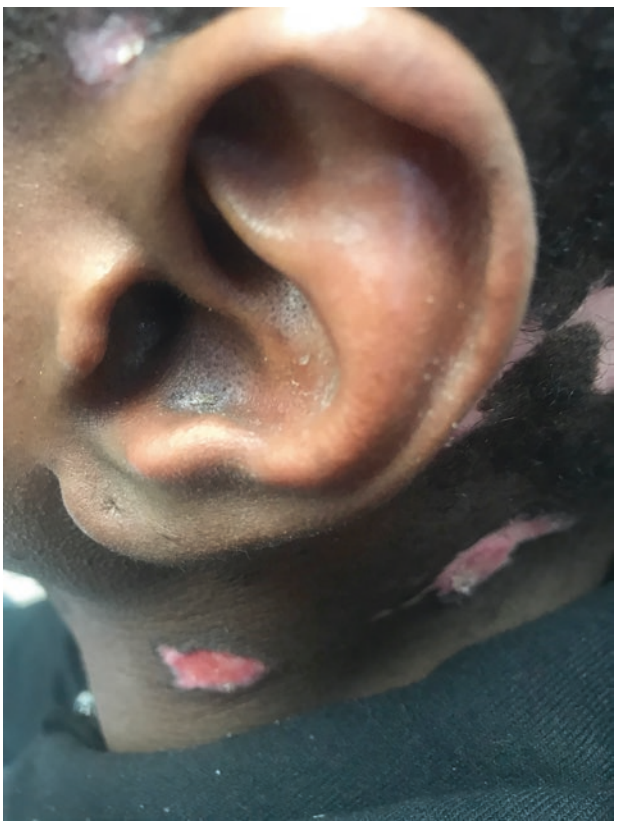

Fig. 4.65 Follicular plugging

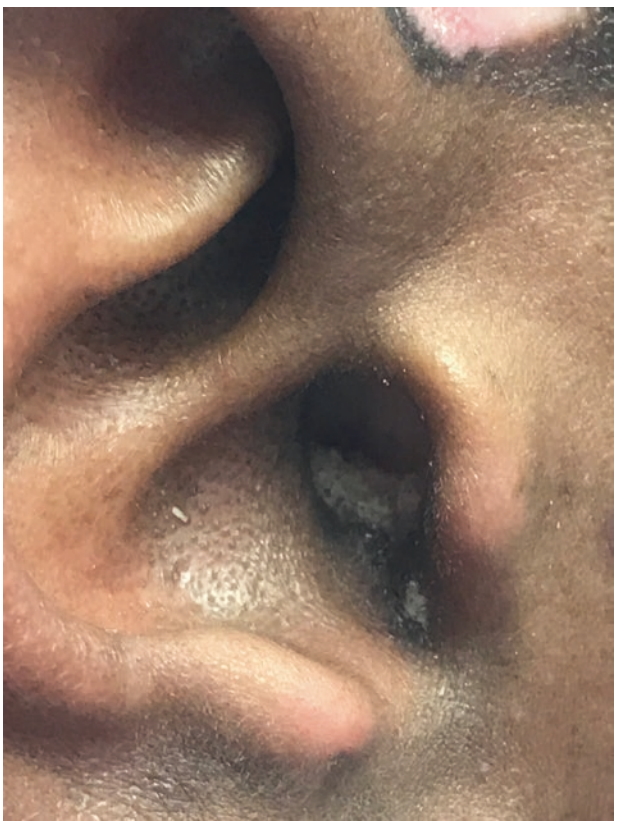

Fig. 4.67 Follicular plugging

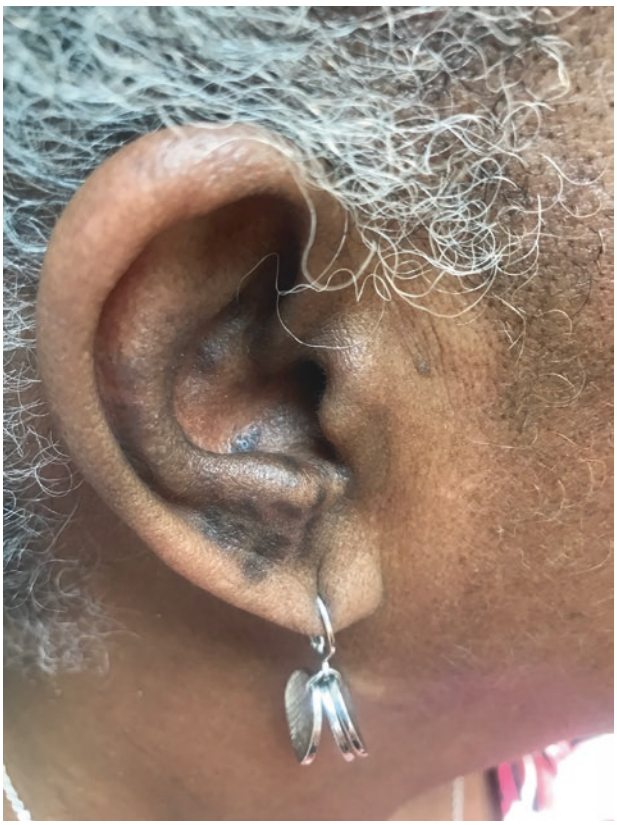

Fig. 4.68 Follicular plugging

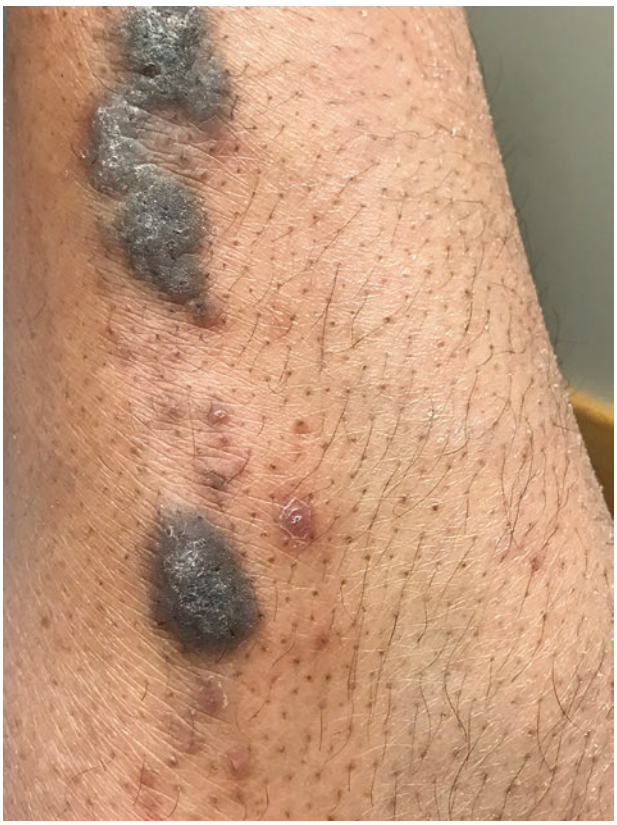

Fig. 4.70 Linear DLE

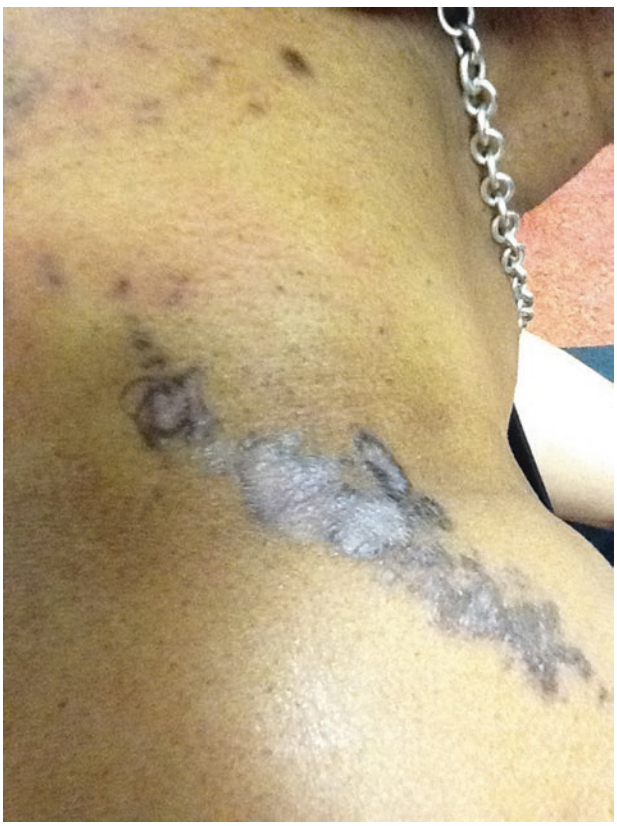

Fig. 4.69 Linear DLE

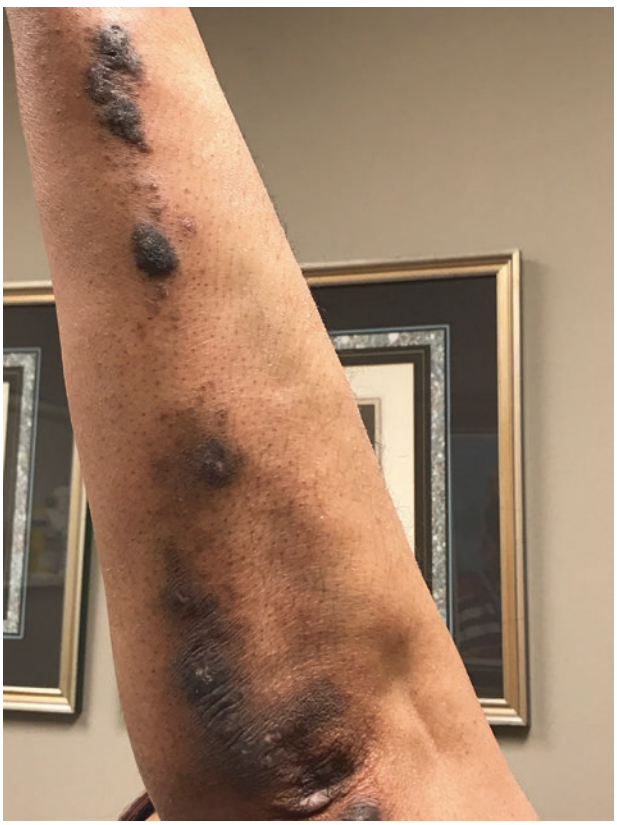

Fig. 4.71 Linear DLE

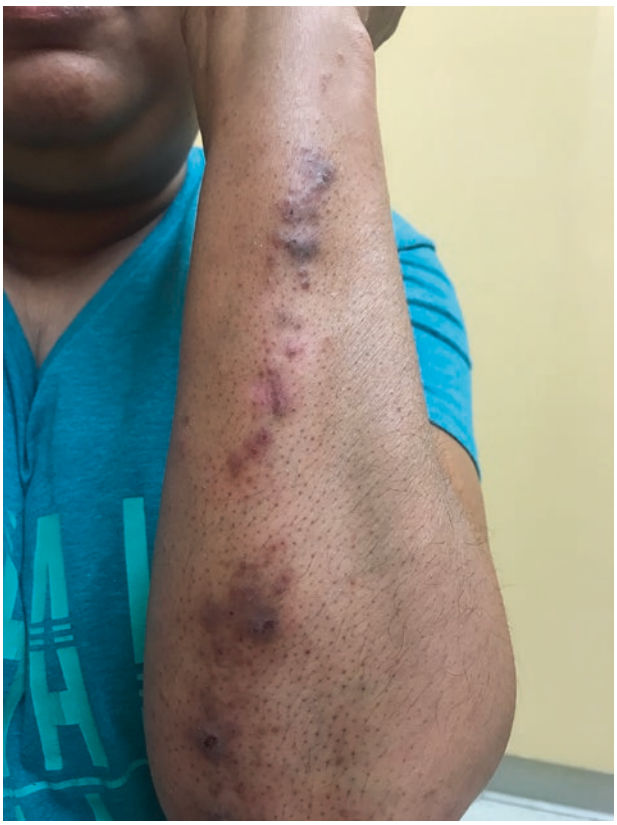

Fig. 4.72 Linear DLE

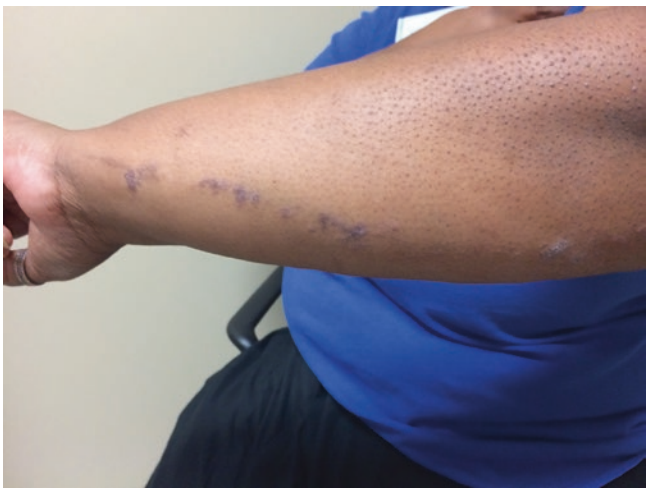

Fig. 4.73 Linear DLE

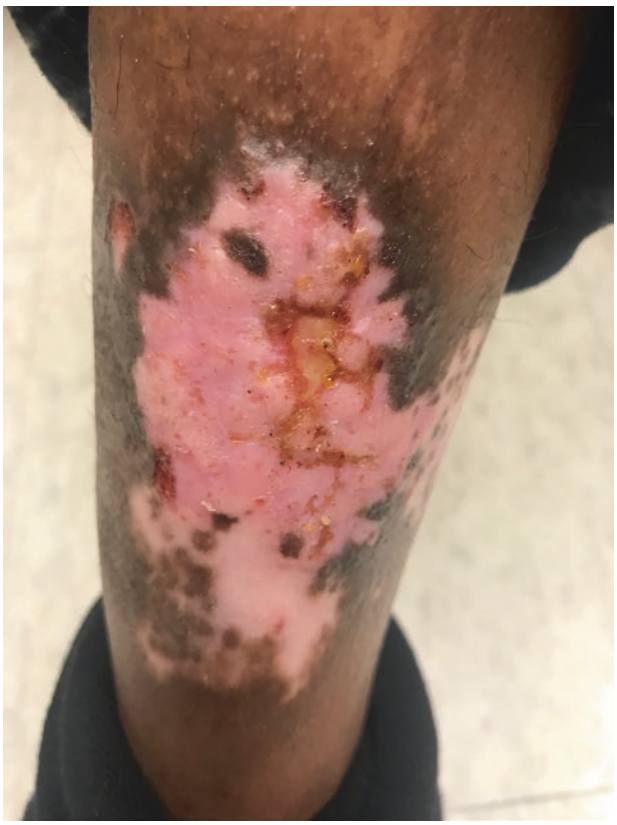

Fig. 4.74 Hypertrophic DLE

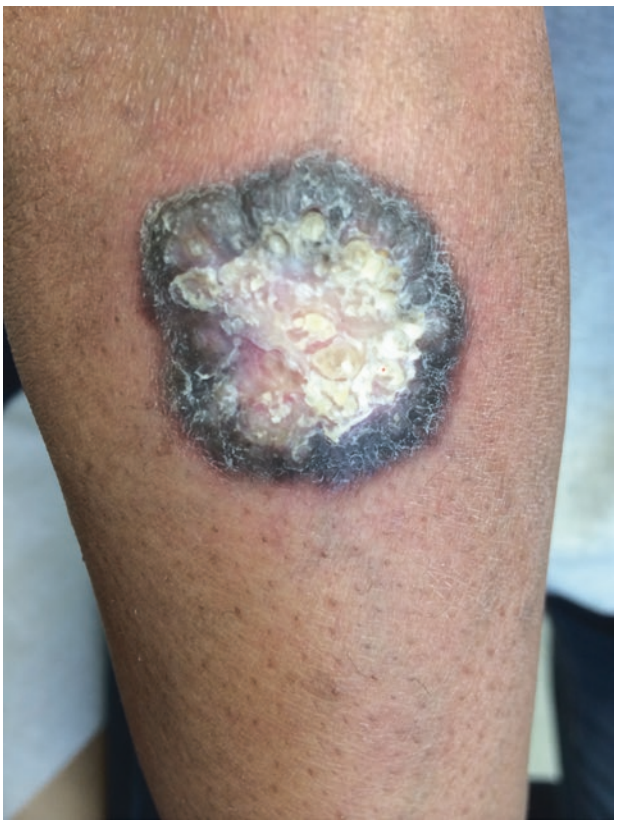

Fig. 4.75 Hypertrophic DLE

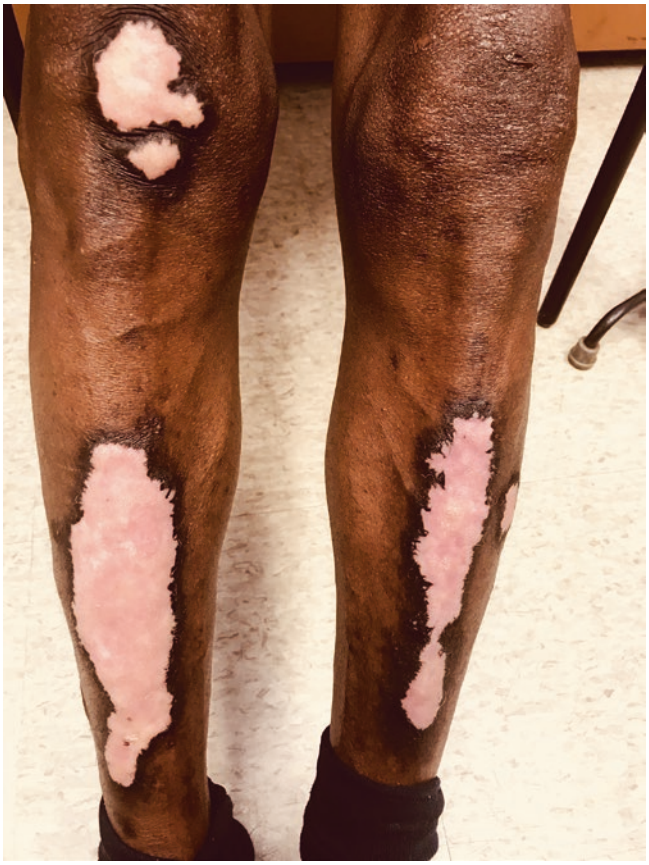

Fig. 4.76 Hypertrophic DLE

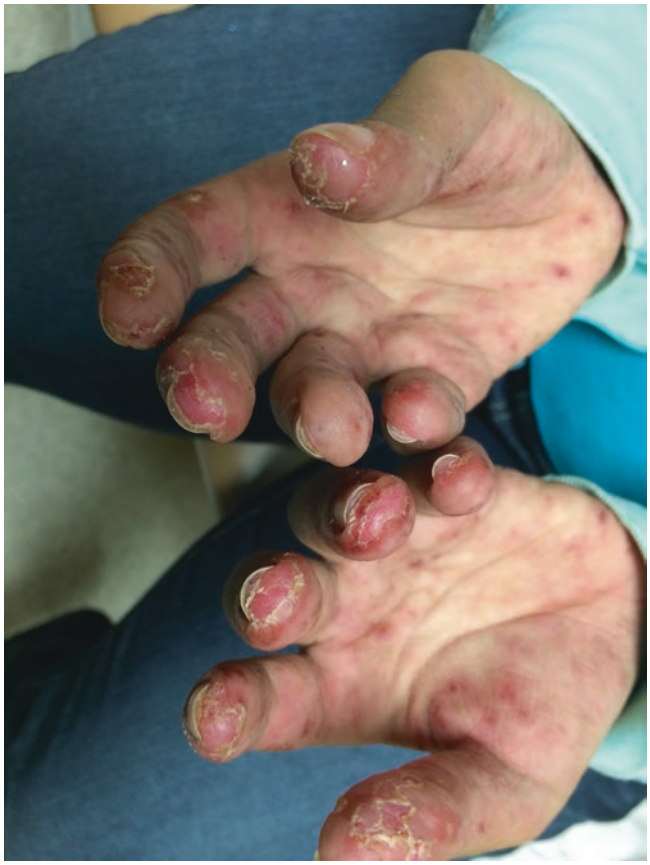

Fig. 4.77 Vasculitis due to systemic lupus erythematosus (SLE)

4.2.2 Treatment and Management

DLE is treated similarly to other forms of cutaneous lupus, using antimalarials and topical corticosteroids as the treatments of choice. Intralesional corticosteroids may be used for active lesions. Strict photoprotection is advised.

4.3 Other Skin Manifestations of SLE

In addition to the conditions described above, SLE can have many other initial presentations. These include vasculitis (Figs. 4.77–4.79), multiple (>15) dermatofibromas (Figs. 4.80–4.83), nonscarring alopecia, photodermatitis, erythema nodosum, and Raynaud phenomenon (Figs. 4.84 and 4.85), among other findings.

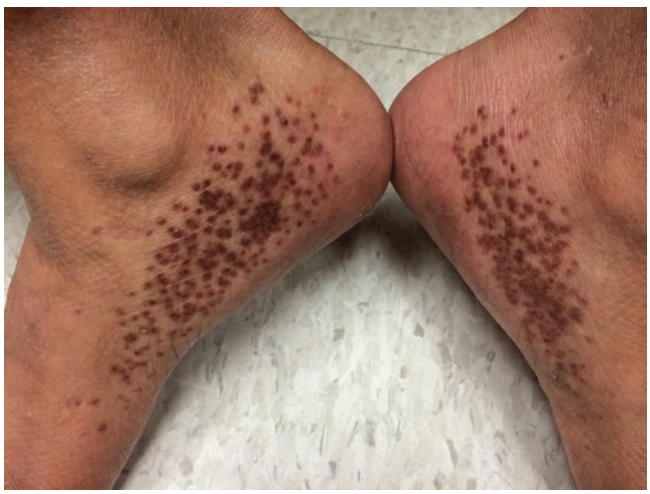

Fig. 4.78 Vasculitis due to SLE

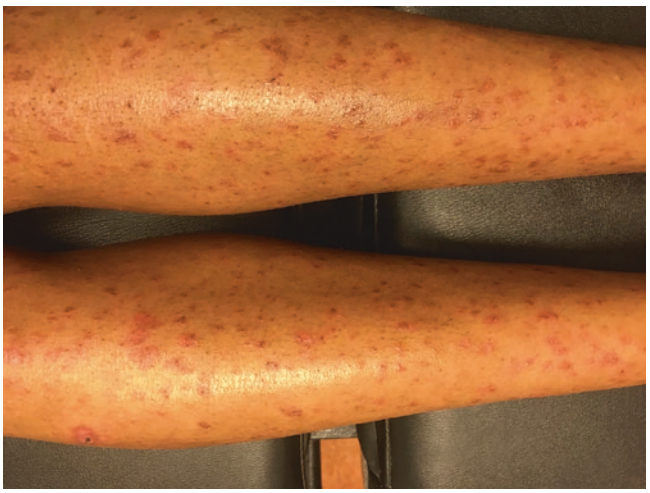

Fig. 4.79 Vasculitis due to SLE

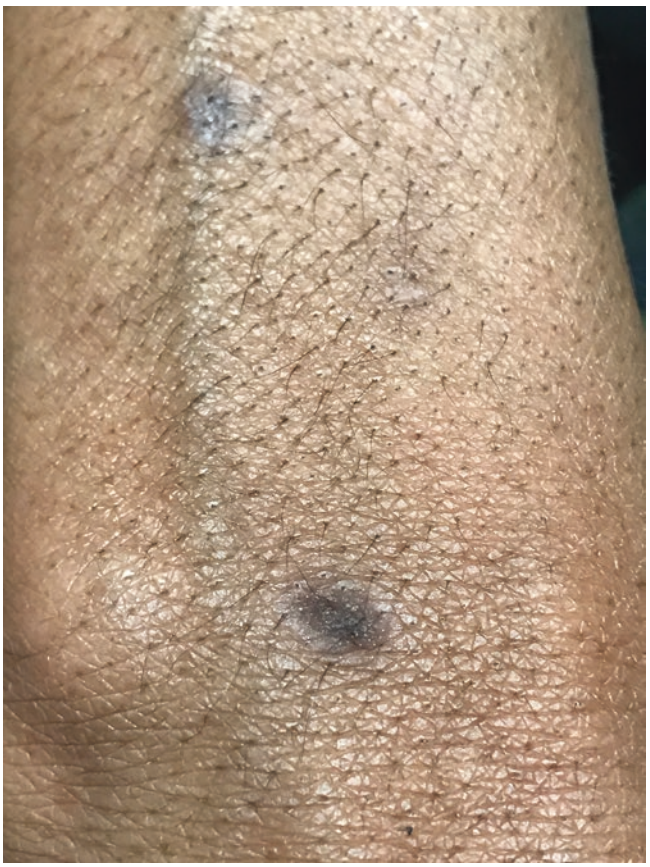

Fig. 4.80 Dermatofibroma

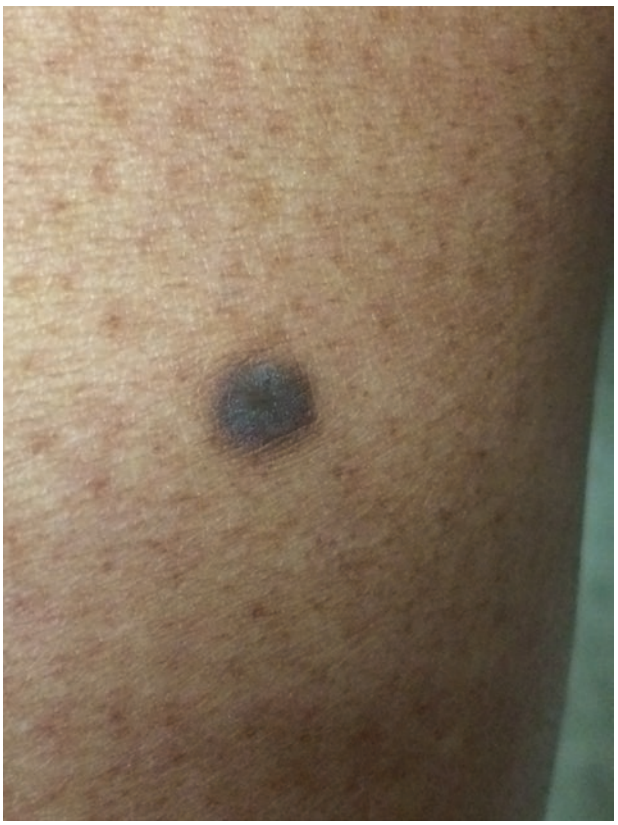

Fig. 4.81 Dermatofibroma

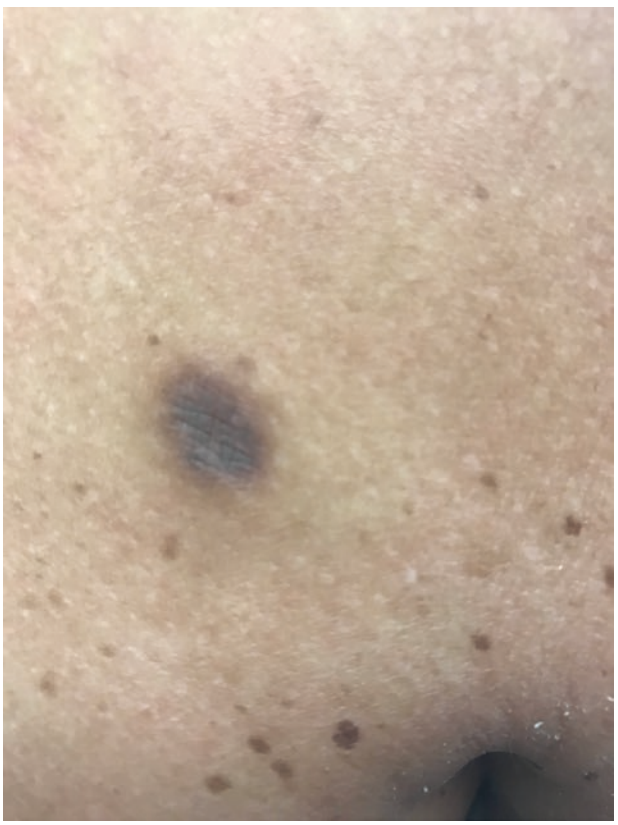

Fig. 4.82 Dermatofibroma

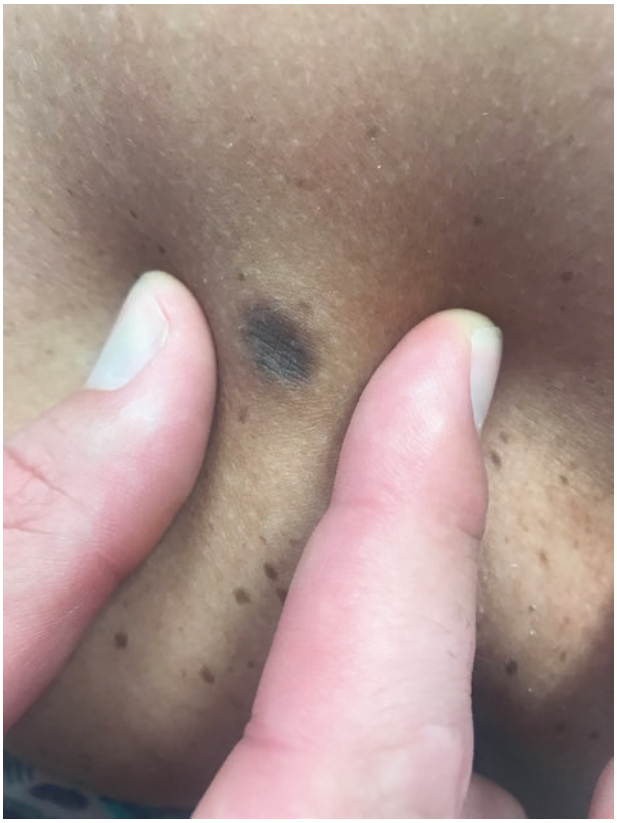

Fig. 4.83 Dimple sign

Fig. 4.85 Secondary Raynaud phenomenon

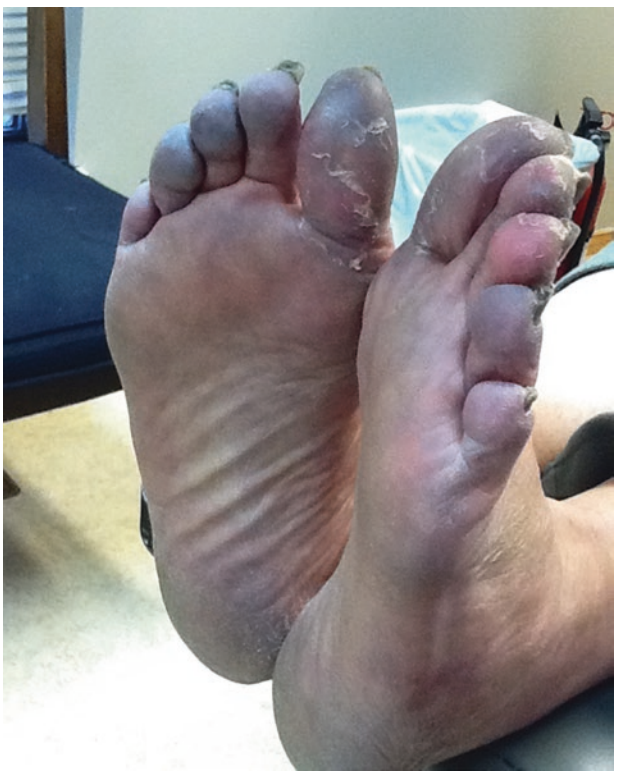

Fig. 4.84 Secondary Raynaud phenomenon

4.4 Photodermatitis

Photodermatoses are caused by an abnormal reaction to sunlight, usually the ultraviolet (UV) component. They are seen in males and females of all races. Time of onset is variable; both environmental and genetic factors play a role. They can be subdivided into immunologically mediated, chemical- or drug-induced, photoaggravated, and genetic diseases. Overall, the incidence of photodermatoses in black skin and Caucasians is similar, but polymorphous light eruption (PMLE) occurs more commonly in black skin, whereas phototoxicity is more common in Caucasians.

PMLE is the most common immunologically mediated photodermatosis. Etiology is unknown. It can occur at any age. It is seen most commonly from March to June and in people travelling to sunny locations. Many patients develop tolerance to UV radiation throughout the summer, hence the usual presentation in spring to early summer.

Chronic actinic dermatitis (CAD) is an idiopathic photodermatitis. In patients with darker skin, CAD is more prevalent in men and presents at any age.

Phototoxic skin reactions are inflammatory skin reactions induced photochemically in exposed areas of skin.

4.4.1 Presentation in Black Skin

PMLE presents a few hours to days after sun exposure. Pruritic, patchy erythema develops into distinct lesions that may be macular, papular, urticarial, papulovesicular, multiforme, or plaquelike (Figs. 4.86–4.100). Pin-head papules are the most common presentation in patients with dark skin. Lesions favor sun-exposed areas (the upper chest, upper arms, dorsal hands, thighs, and lateral face), but the face and dorsal hands may be spared subsequent to natural hardening. Skin lesions may be reproduced using experimental provocation. Skin lesions resolve spontaneously without scarring within a few days of ceasing sun exposure.

CAD presents with lichenified, inflamed erythematous papules or plaques in sun-exposed areas. The lesions may extend onto covered areas of skin. Excoriations may be present, as pruritis is significant. The most common locations include the forehead, cheeks, ears, nape of the neck, and dorsal hands (Figs. 4.101–4.109). Spontaneous resolution has been reported in up to 10% of patients after 5 years, 20% after 10 years, and 50% after 15 years.

Phototoxic/photoallergic eruptions present with symptoms similar to sunburn; acute dermatitis with erythema, edema, and vesicles is classically observed. Post-inflammatory hyperpigmentation may develop (Figs. 4.110 and 4.111).

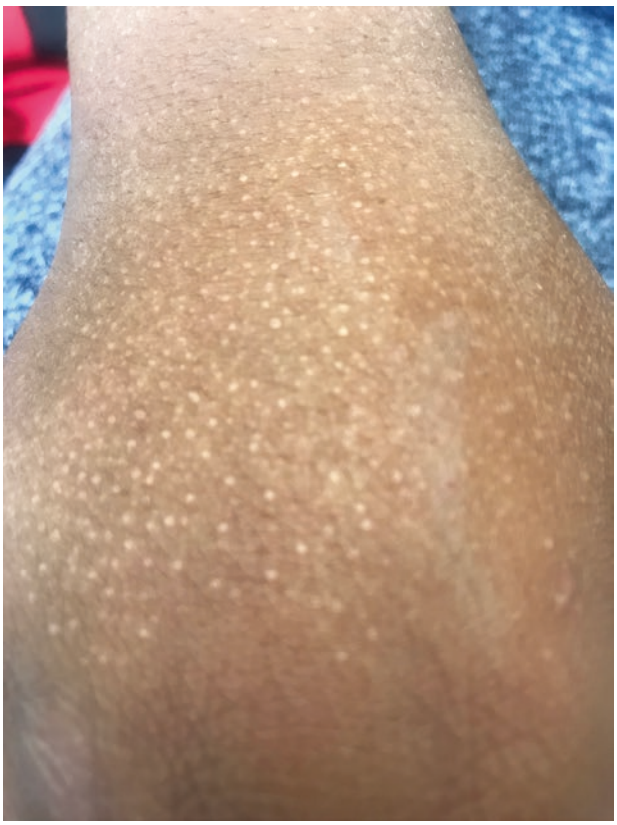

Fig. 4.87 PMLE

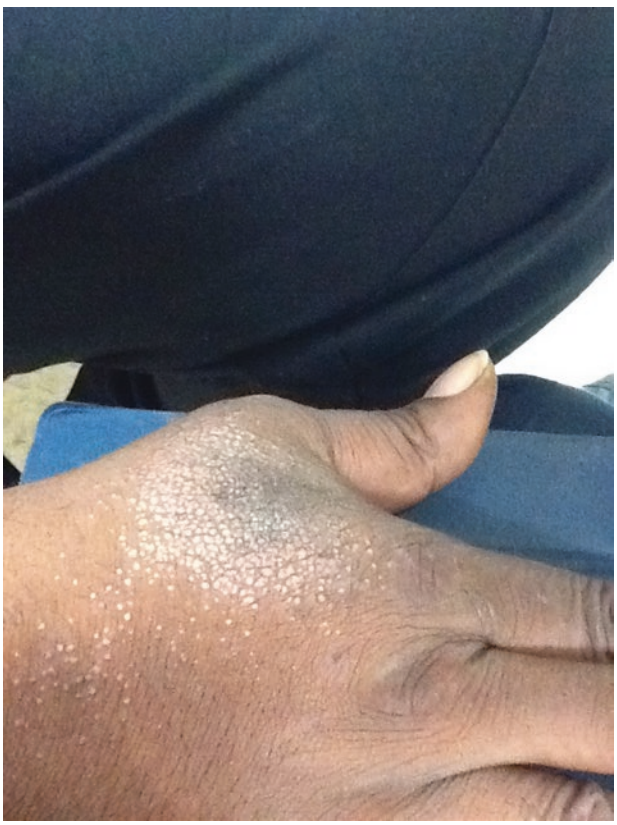

Fig. 4.86 Polymorphous light eruption (PMLE)

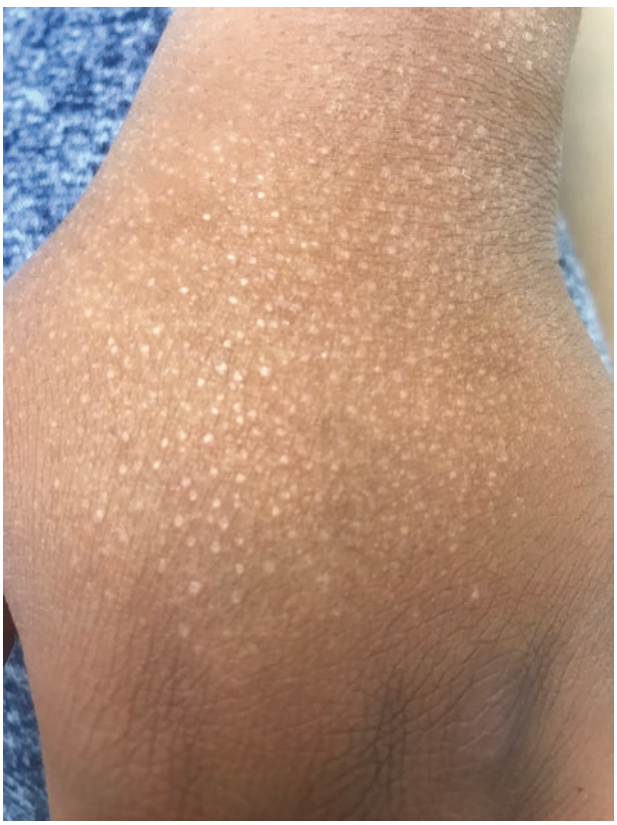

Fig. 4.88 PMLE

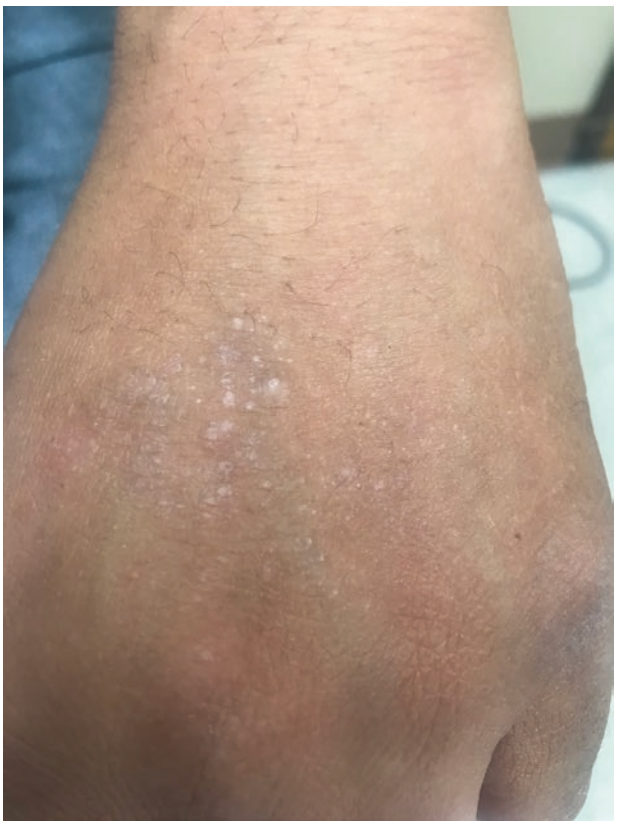

Fig. 4.89 PMLE

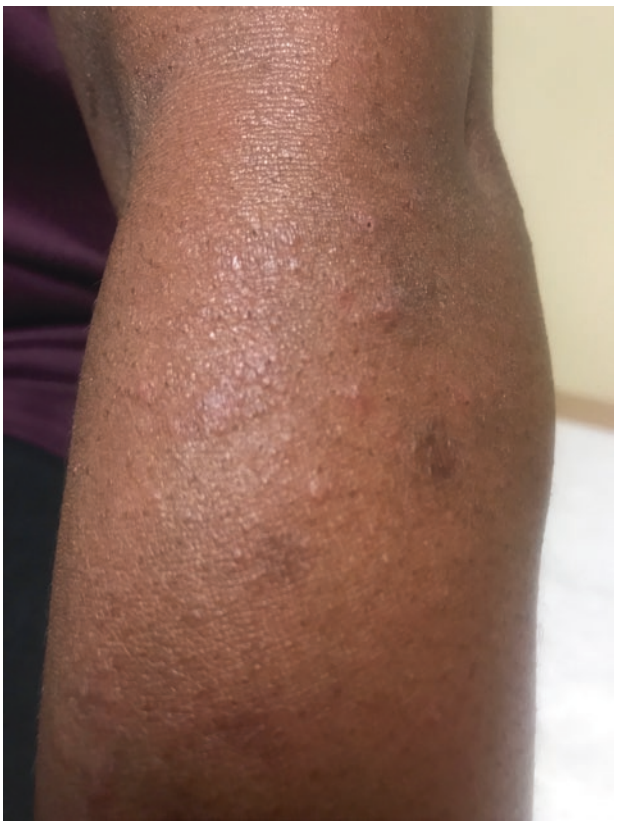

Fig. 4.90 PMLE

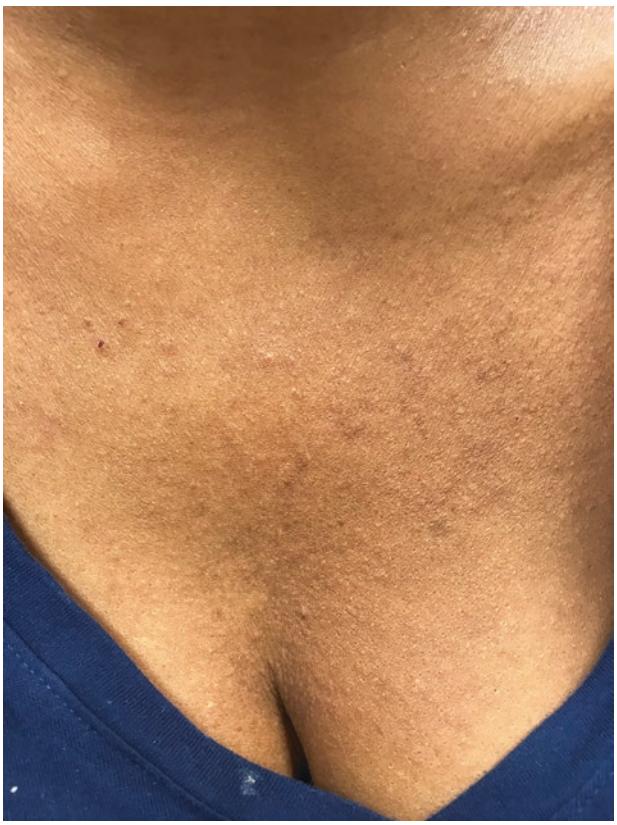

Fig. 4.91 PMLE

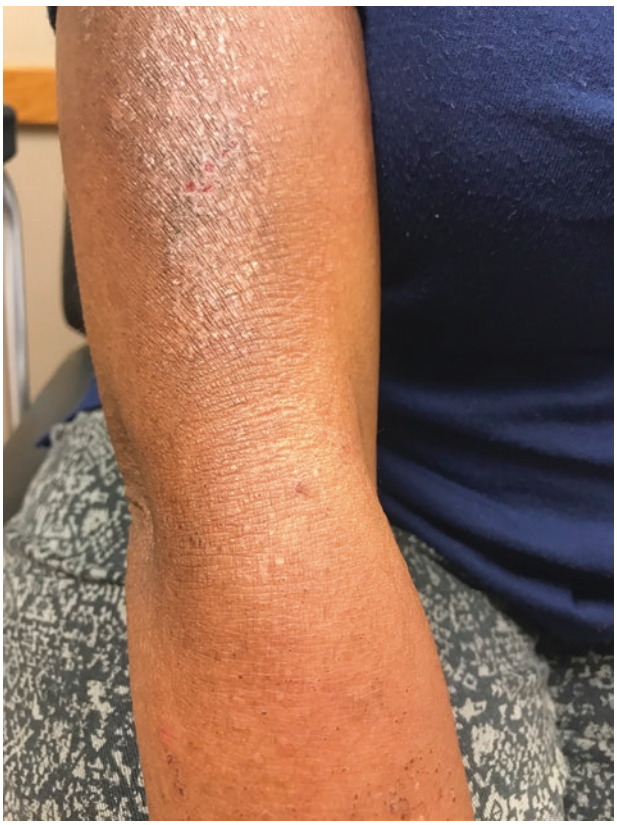

Fig. 4.92 PMLE

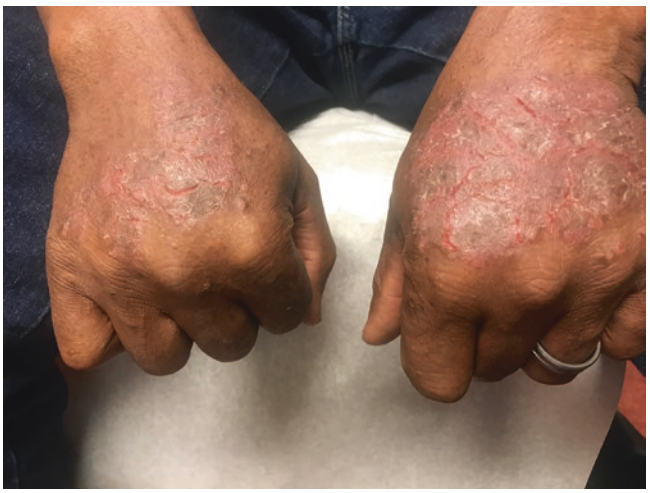

Fig. 4.93 PMLE

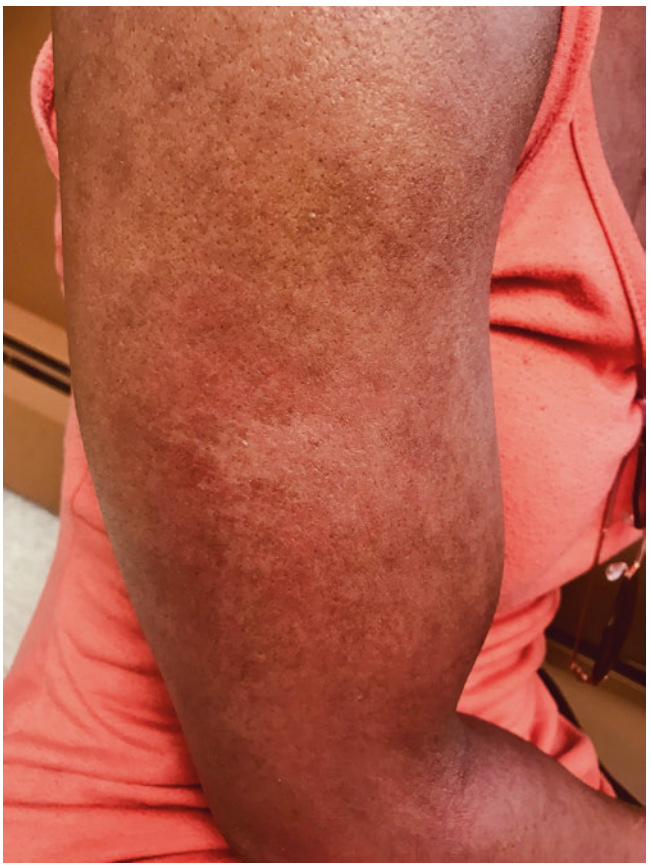

Fig. 4.94 PMLE

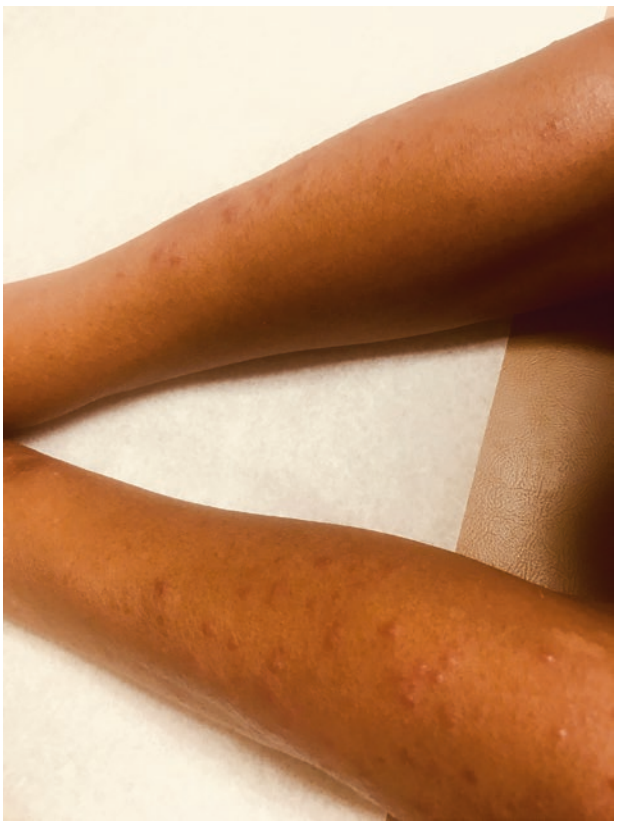

Fig. 4.95 PMLE

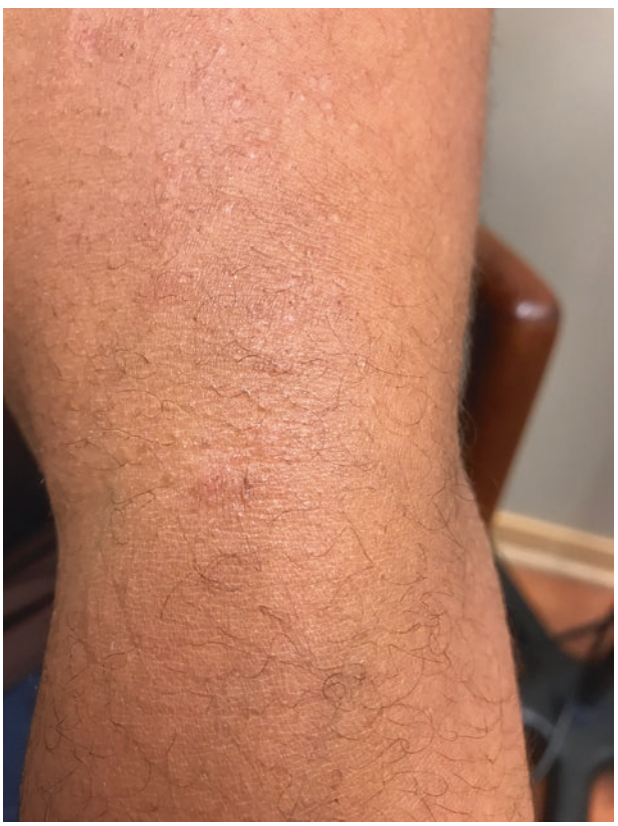

Fig. 4.96 PMLE

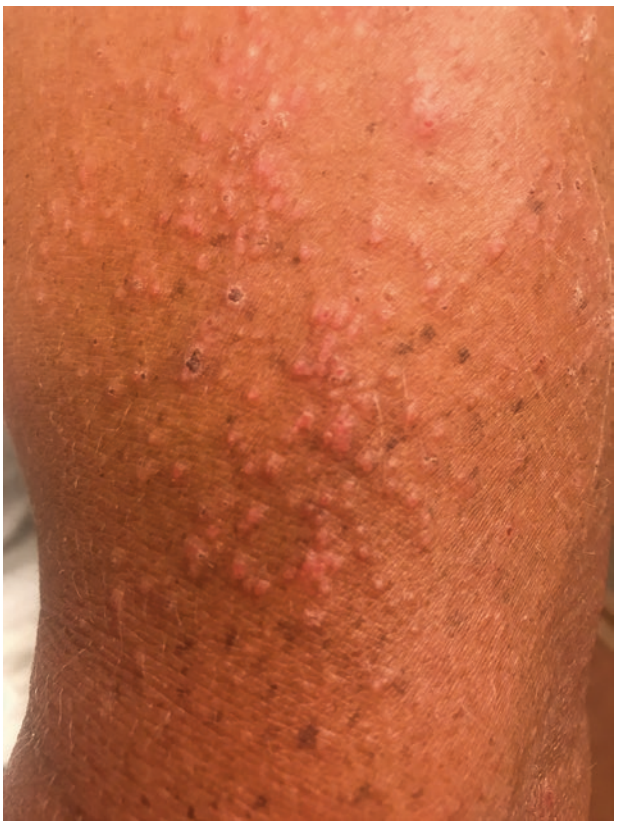

Fig. 4.97 PMLE

Fig. 4.98 PMLE

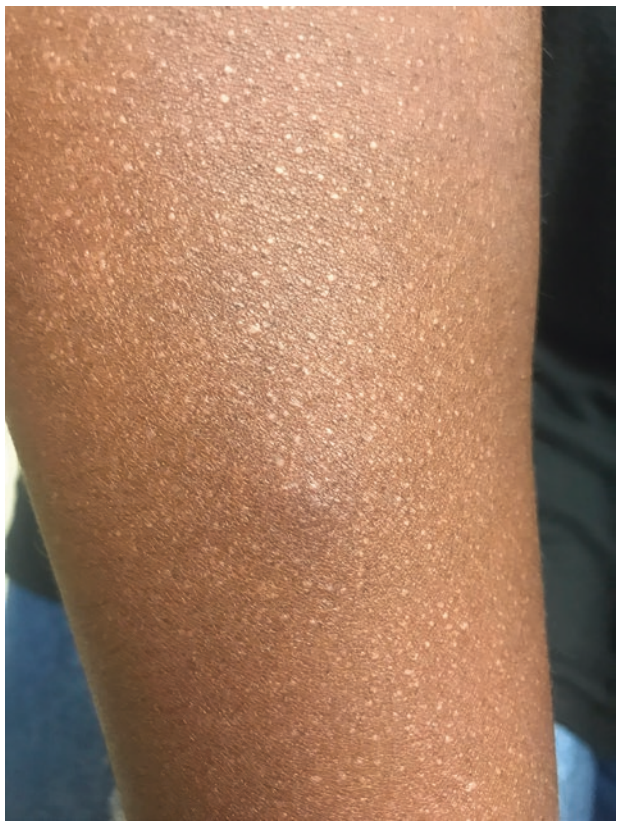

Fig. 4.99 PMLE

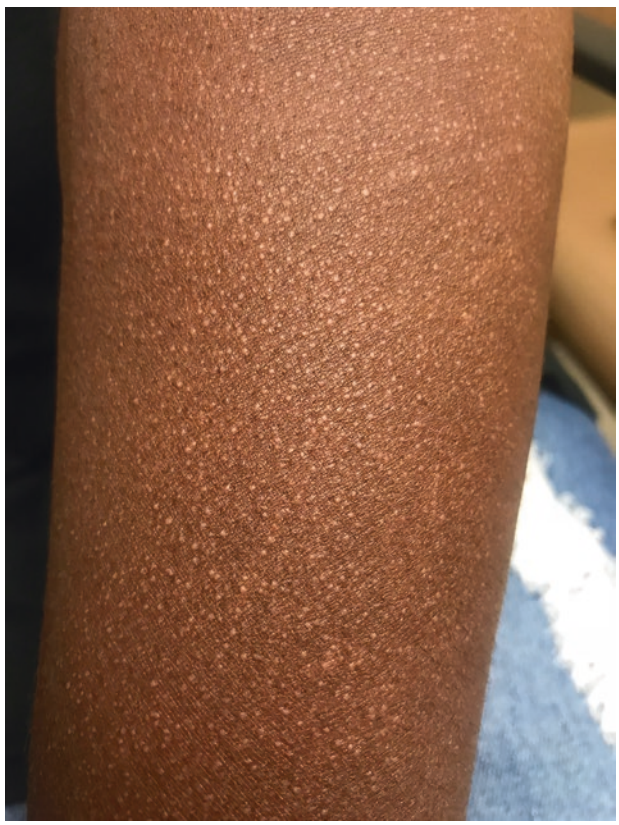

Fig. 4.100 PMLE

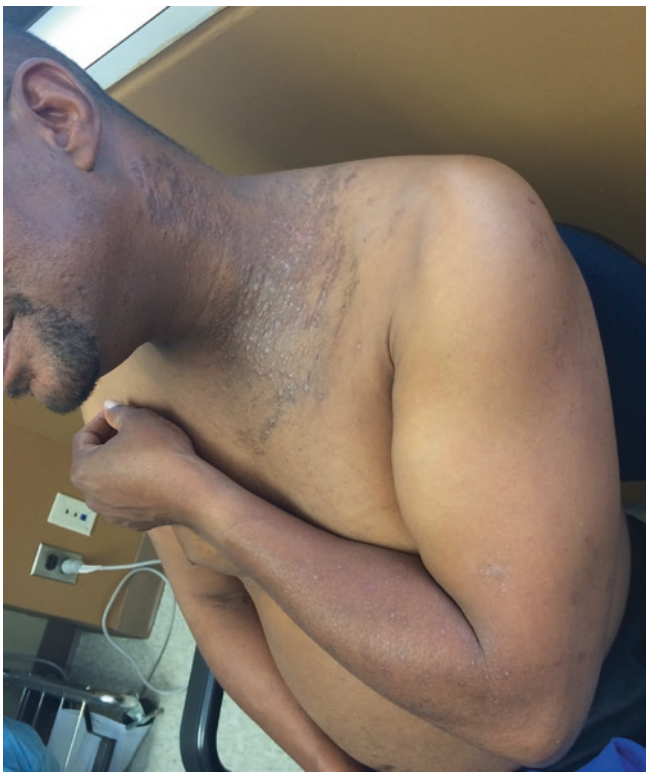

Fig. 4.101 Chronic actinic dermatitis (CAD)

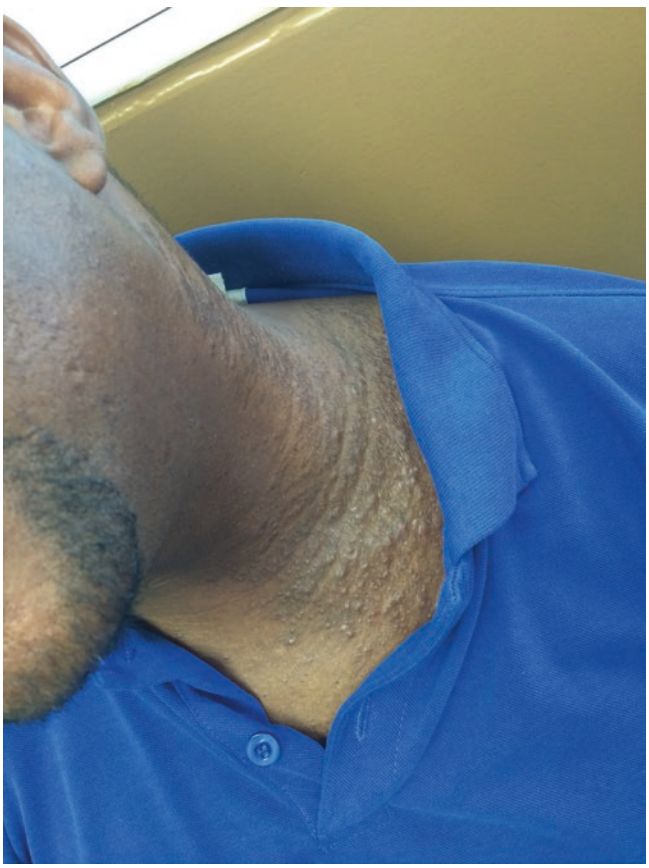

Fig. 4.102 CAD

Fig. 4.103 CAD

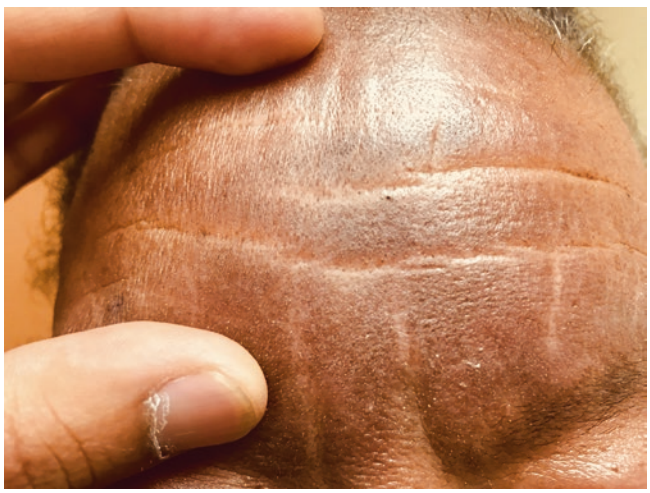

Fig. 4.104 CAD, close-up of Fig. 4.103

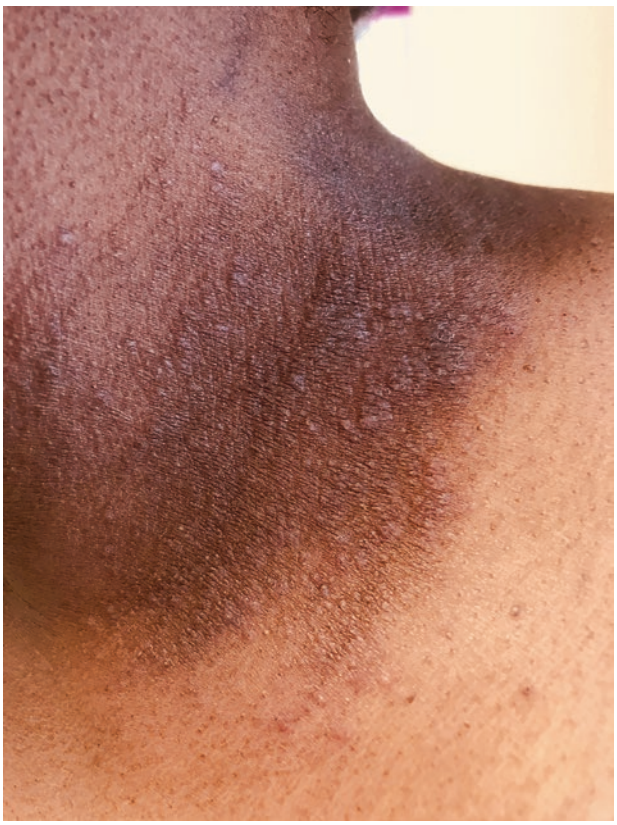

Fig. 4.105 CAD

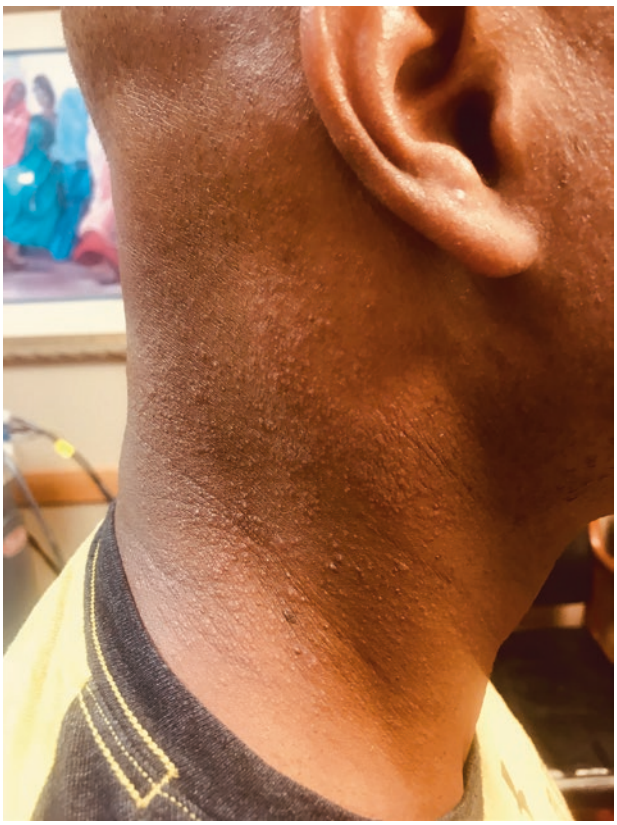

Fig. 4.107 CAD

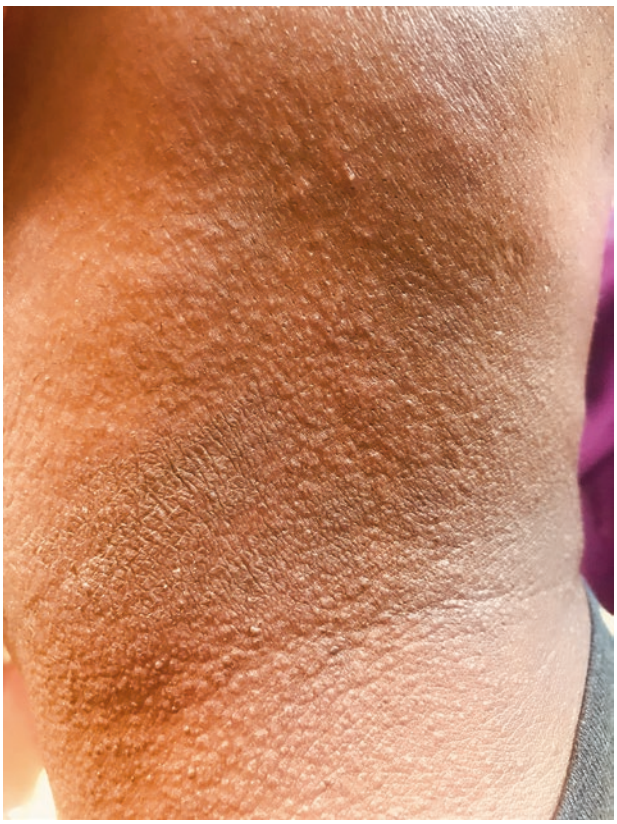

Fig. 4.106 CAD

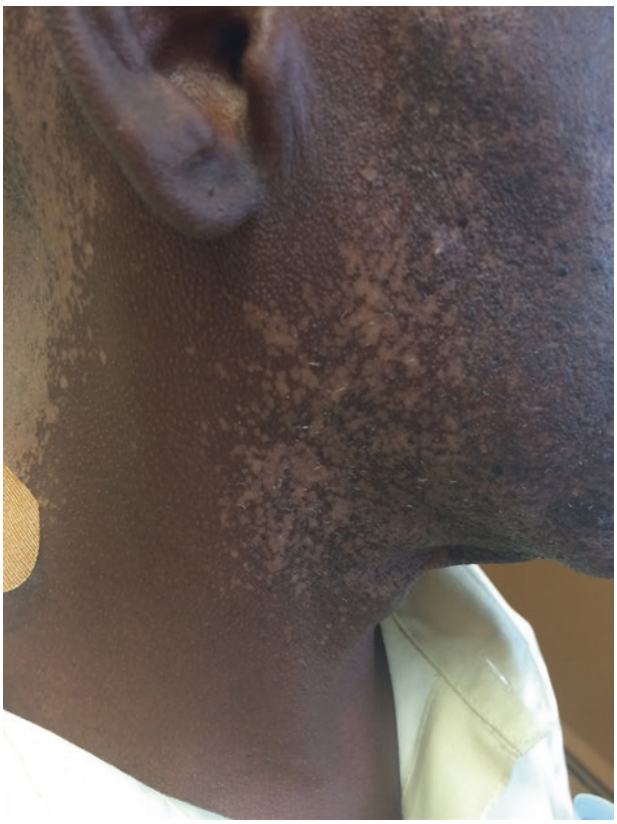

Fig. 4.108 CAD

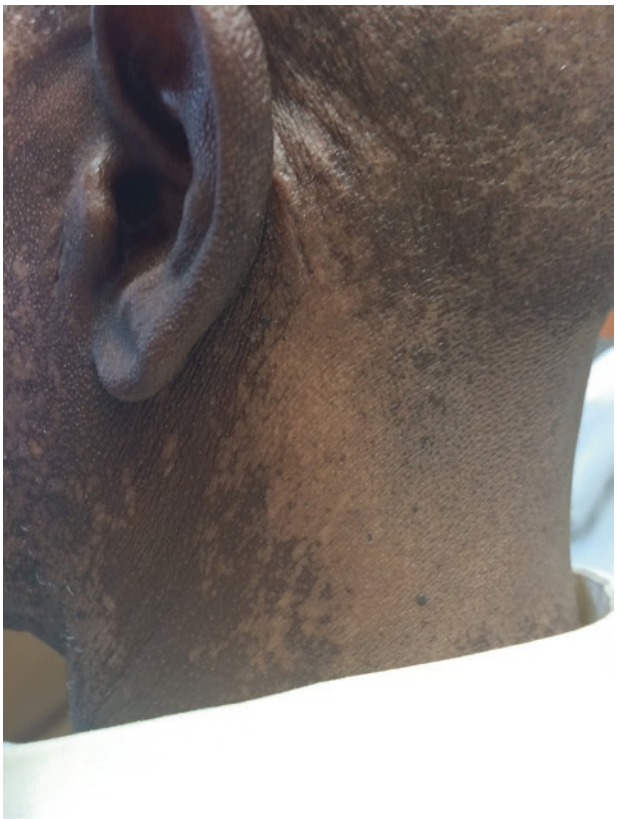

Fig. 4.109 CAD

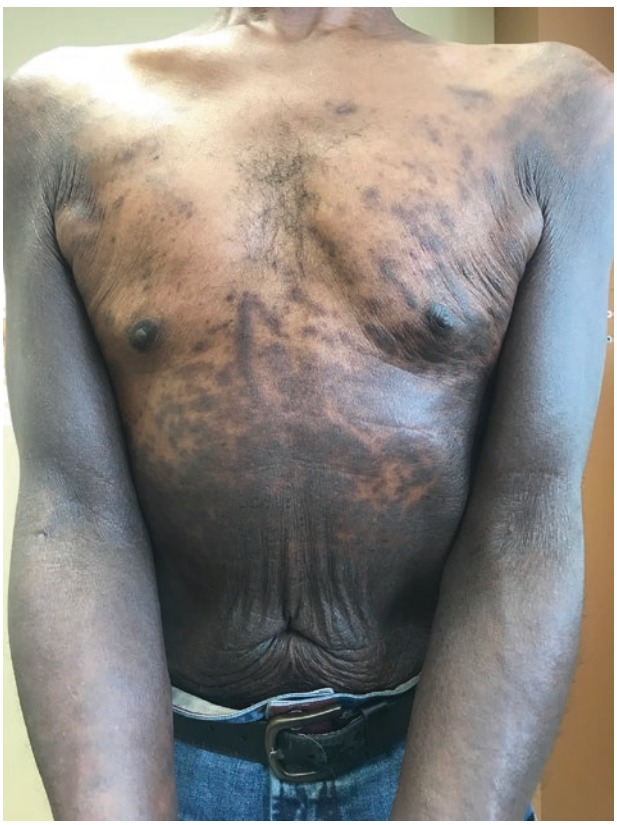

Fig. 4.110 Photoallergic reaction secondary to doxycycline

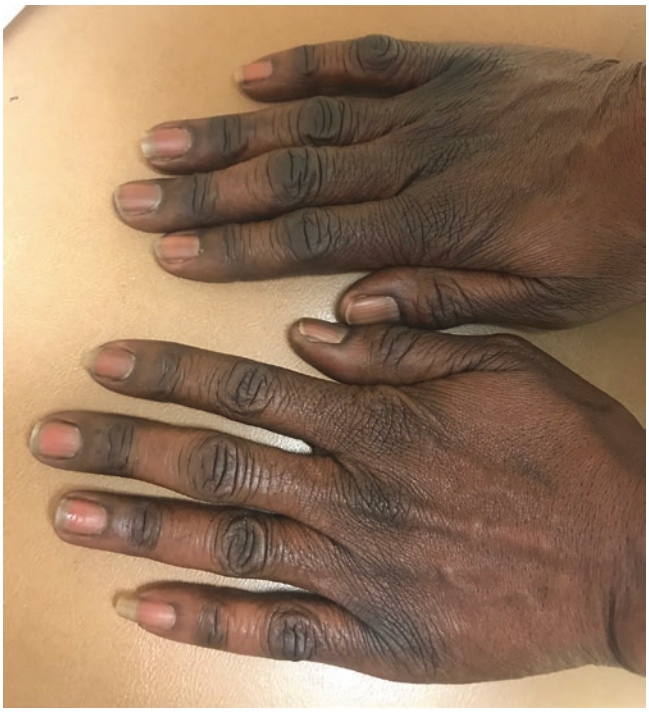

Fig. 4.111 Photoallergic reaction secondary to Bactrim (sulfamethoxazole and trimethoprim)

4.4.2 Treatment and Management

PMLE will resolve with cessation of sun exposure. Symptomatic treatment of PMLE can be accomplished with topical steroids and oral antihistamines. Prevention with application of broad-spectrum sunscreen is most important. Hardening can be accelerated with phototherapy leading up to sunny months.

Avoiding UV exposure is the first step in treatment of CAD. Intensive sun protection is crucial. Performing leisure activities in the evening or at night, wearing UV-protective clothing, and wearing full-coverage tinted makeup can help. PUVA is the treatment of choice, but other options include systemic steroids, azathioprine, and cyclosporine.

Discontinuation of photosensitizing medications is crucial to prevent reoccurrence of phototoxic eruption.

4.5 Sarcoidosis

Sarcoidosis is a systemic granulomatous disease of unknown etiology, which is characterized by noncaseating granulomas. Many environmental and genetic factors play a role in the etiology. Sarcoidosis is more common in women and blacks. Blacks often present with more severe disease that has a worse prognosis than sarcoidosis in Caucasians. The lungs, lymph nodes, eyes, and skin are the organs most often involved. Cutaneous manifestations are present in 20% of patients and are the presenting symptom in one third of these patients.

Sarcoidosis has a wide range of cutaneous manifestations, including "specific" eruptions, characterized by noncaseating granulomas, or "nonspecific" eruptions that develop as a result of a reactive process without granuloma formation.

Erythema nodosum (EN) is the most common nonspecific manifestation of cutaneous sarcoidosis. EN is typically associated with a better prognosis and transient disease.

4.5.1 Presentation in Black Skin

Papular sarcoidosis favors the face, specifically the periorbital region, and starts as 1–5-mm papules that are initially orange to yellow-brown and turn red to violaceous before involuting to faint macules. Papular sarcoidosis is associated with a good prognosis; lesions usually heal without scarring. Maculopapular sarcoidosis is most commonly seen on the neck, extremities, and mucous membranes. Overall, this type of cutaneous sarcoidosis has a good prognosis. Plaque sarcoidosis presents on the back, buttocks, face, and extensor extremities and is associated with a chronic disease course. Plaques may become hypopigmented in skin of color. Lupus pernio presents with chronic, indurated violaceous to red-brown plaques, usually on the nose or cheeks. The plaques enlarge to become confluent and progressively disfiguring (Figs. 4.112–4.135).

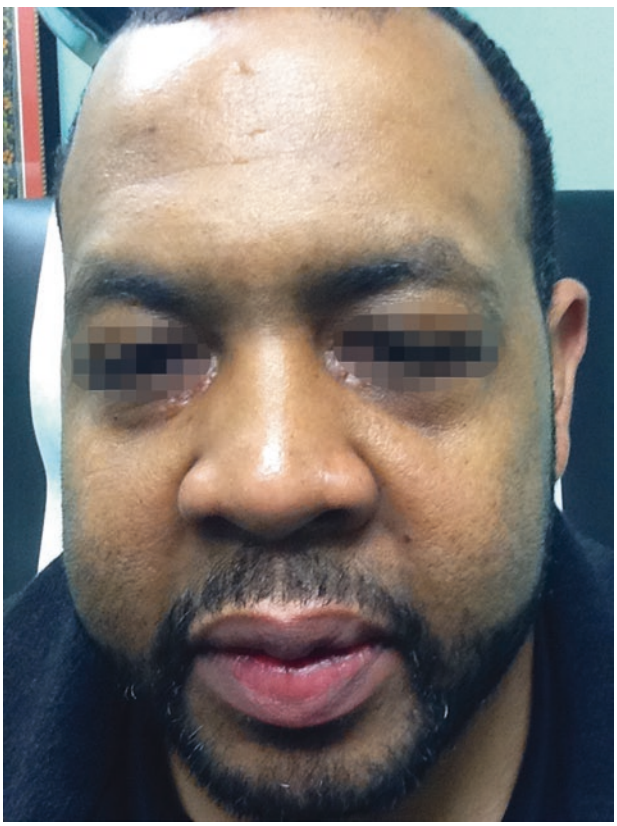

Fig. 4.112 Sarcoidosis

Other variants include osseus sarcoidosis (Fig. 4.136), hypotrophic sarcoidosis (Figs. 4.137–4.139), and annular sarcoidosis (Figs. 4.140 and 4.141). Annular sarcoidosis presents with annular papules or plaques on the face, most commonly the forehead. It can heal with scarring and hair loss. Figures 4.142–4.145 show hypopigmented sarcoidosis. Other types of cutaneous sarcoidosis include ulcerative, icthyosiform, psoriasiform, lichenoid, verrucous, and angiolupoid.

4.5.2 Treatment and Management

Treatment of cutaneous sarcoid is recommended for disease that is disfiguring or symptomatic. Topical and intralesional corticosteroids are the treatment of choice for cutaneous disease. Oral corticosteroids may be used for recalcitrant lesions or those that are rapidly progressive. Alternative therapies include methotrexate, antimalarials, and tumor necrosis factor-alpha (TNF-α) inhibitors.

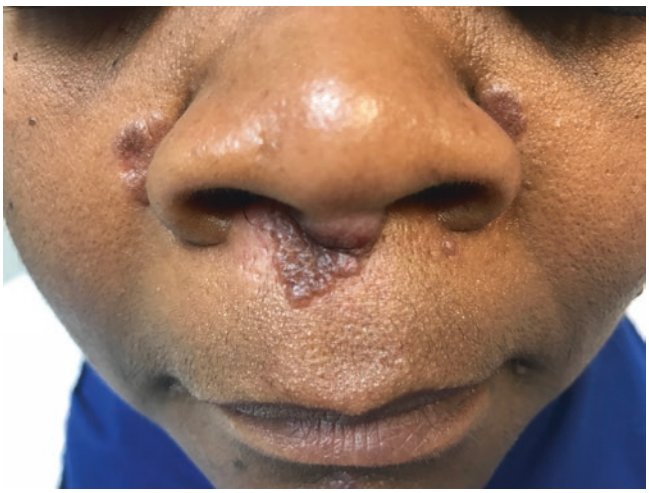

Fig. 4.113 Sarcoidosis

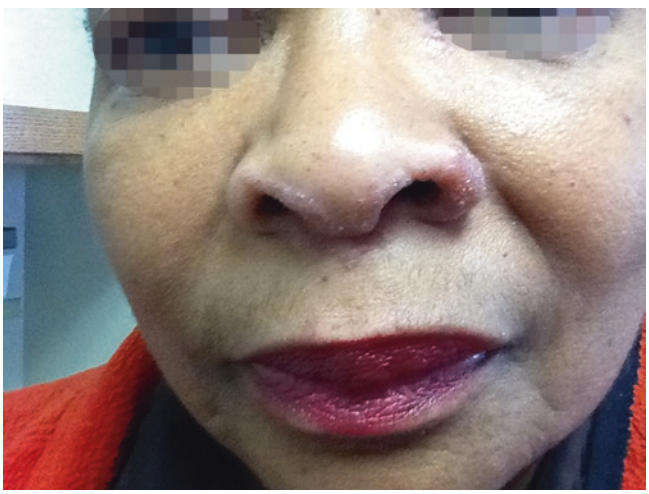

Fig. 4.114 Sarcoidosis

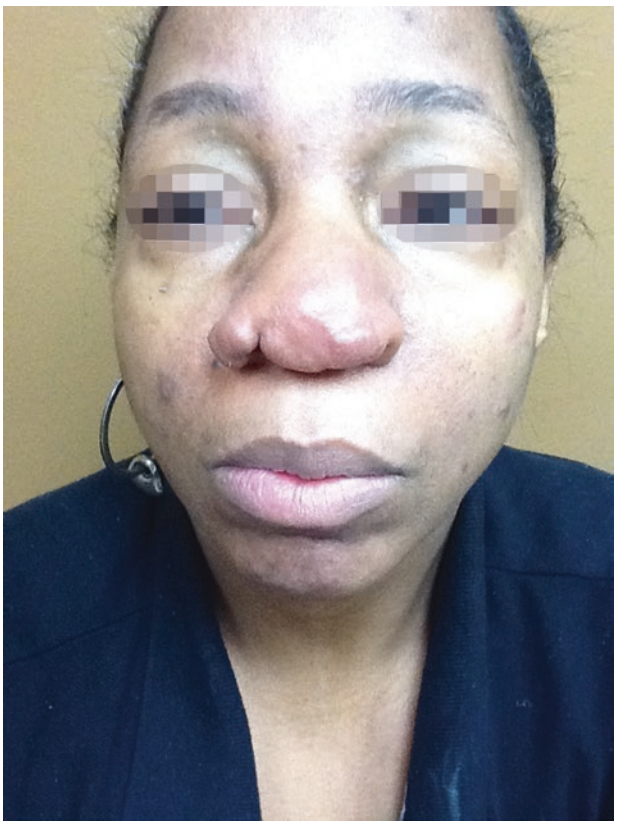

Fig. 4.115 Sarcoidosis with destruction of nose

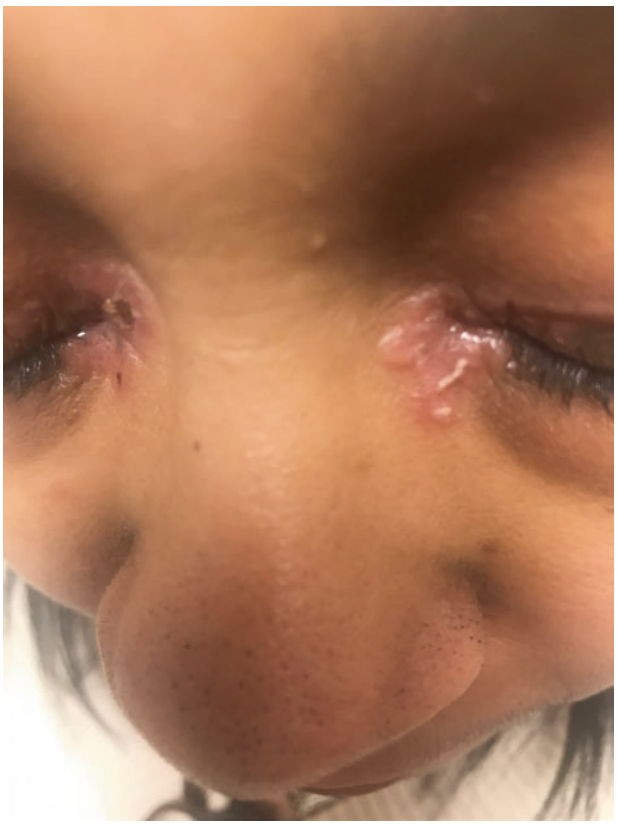

Fig. 4.117 Sarcoidosis

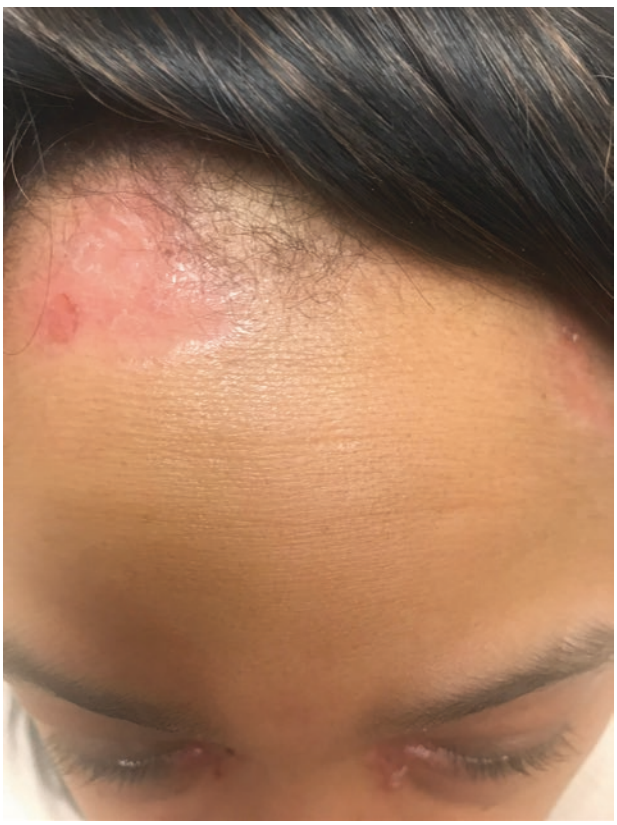

Fig. 4.116 Sarcoidosis

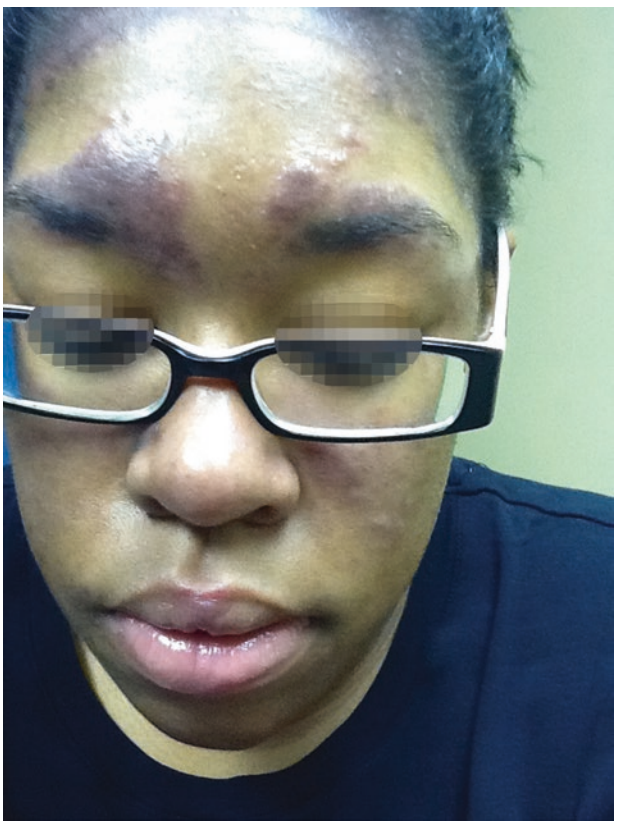

Fig. 4.118 Sarcoidosis mimicking psoriasis

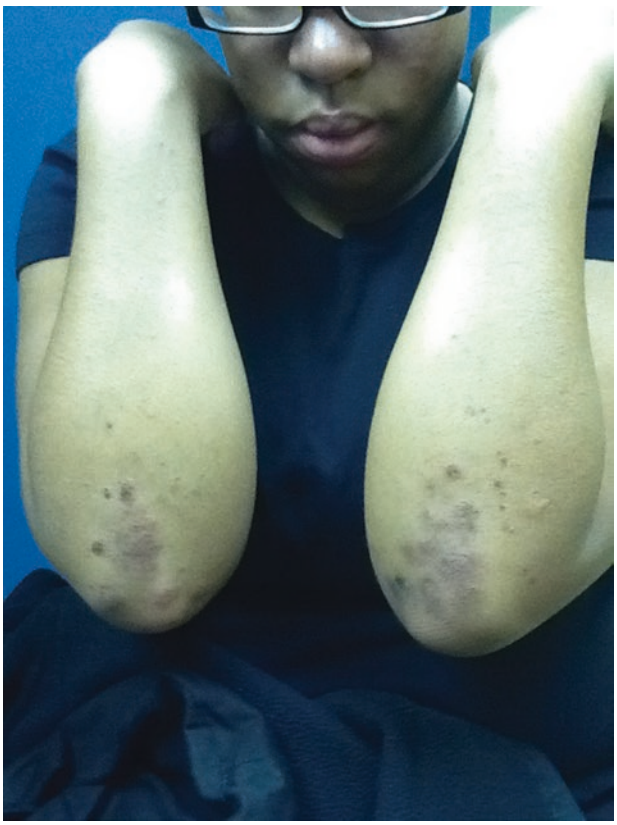

Fig. 4.119 Sarcoidosis mimicking psoriasis

Fig. 4.121 Sarcoidosis

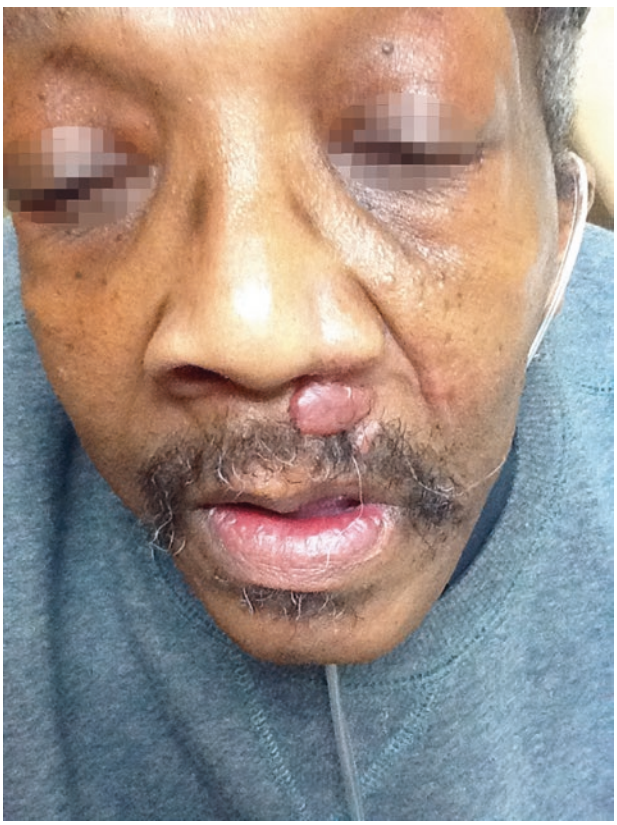

Fig. 4.120 Sarcoidosis

Fig. 4.122 Sarcoidosis

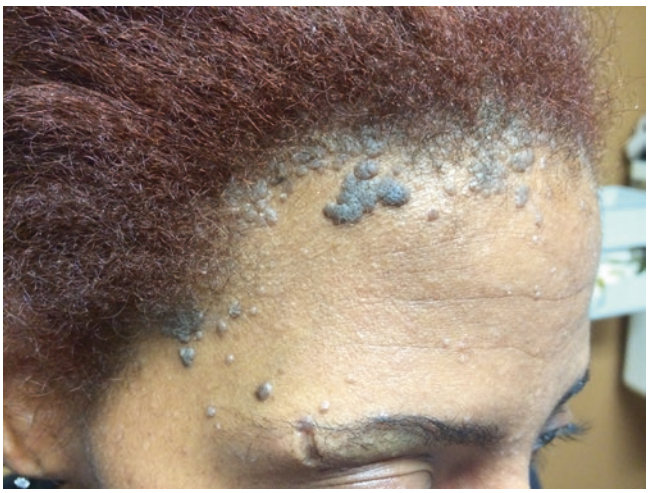

Fig. 4.123 Sarcoidosis mimicking psoriasis

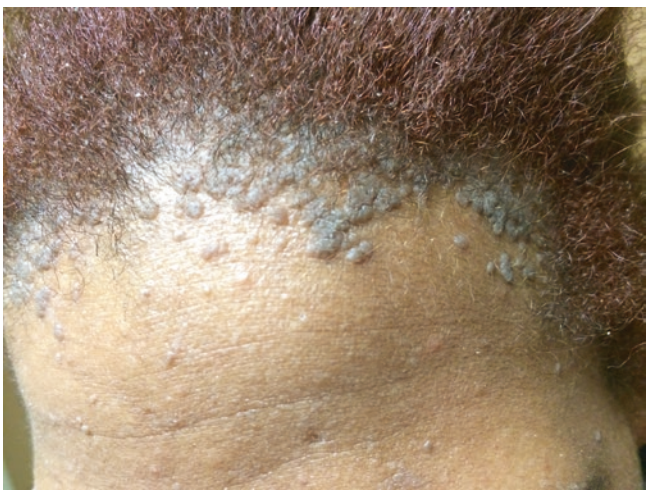

Fig. 4.124 Sarcoidosis mimicking psoriasis

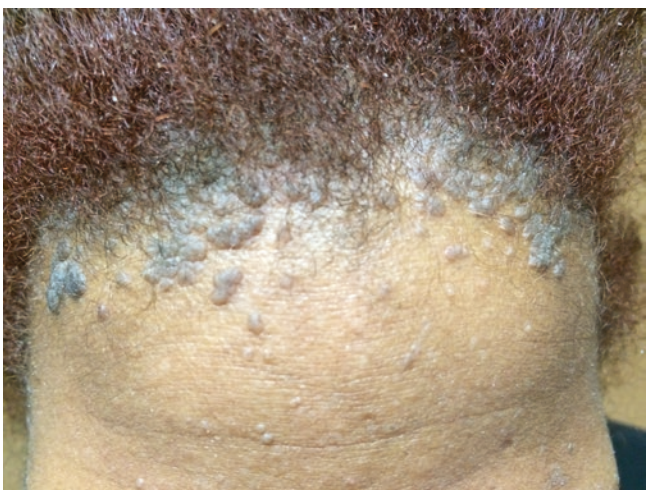

Fig. 4.125 Sarcoidosis mimicking psoriasis

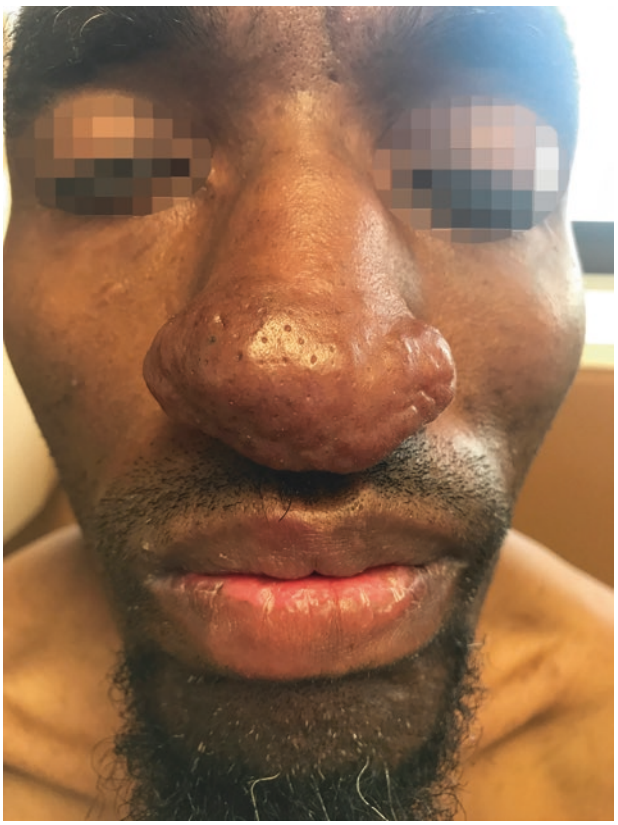

Fig. 4.126 Sarcoidosis

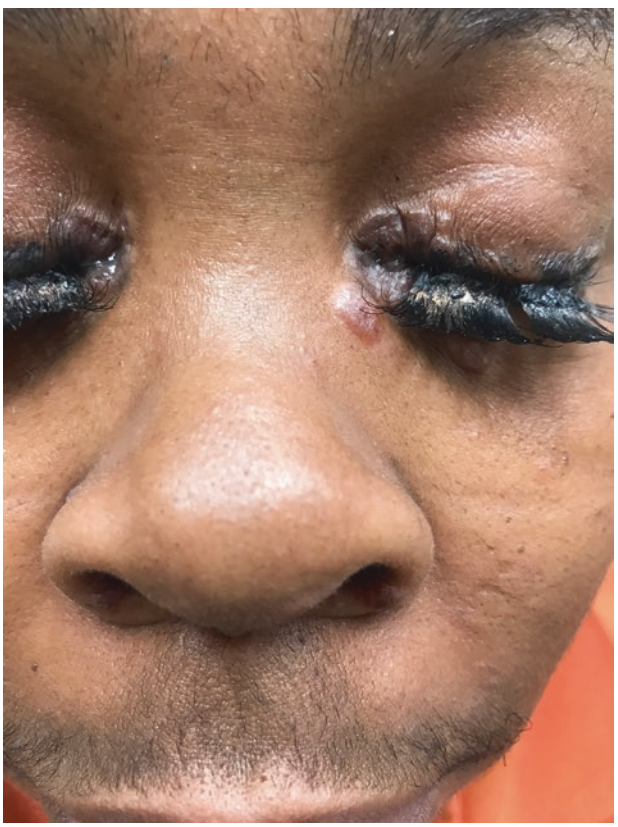

Fig. 4.127 Sarcoidosis

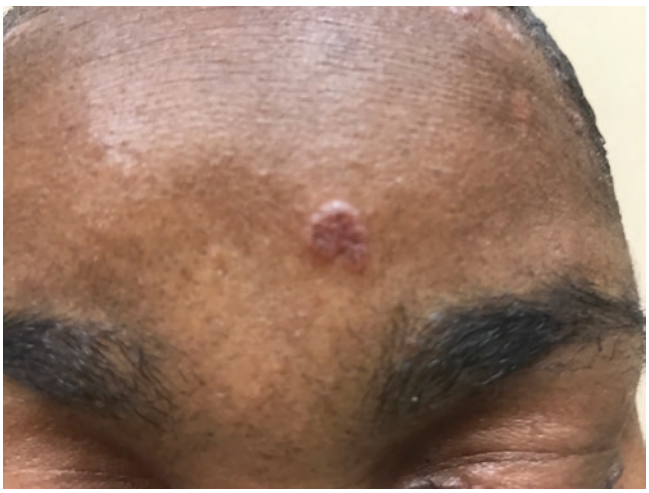

Fig. 4.128 Sarcoidosis

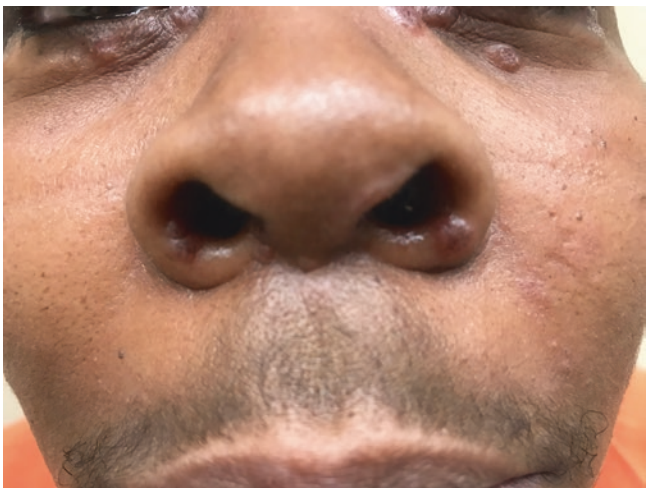

Fig. 4.129 Sarcoidosis

Fig. 4.130 Sarcoidosis

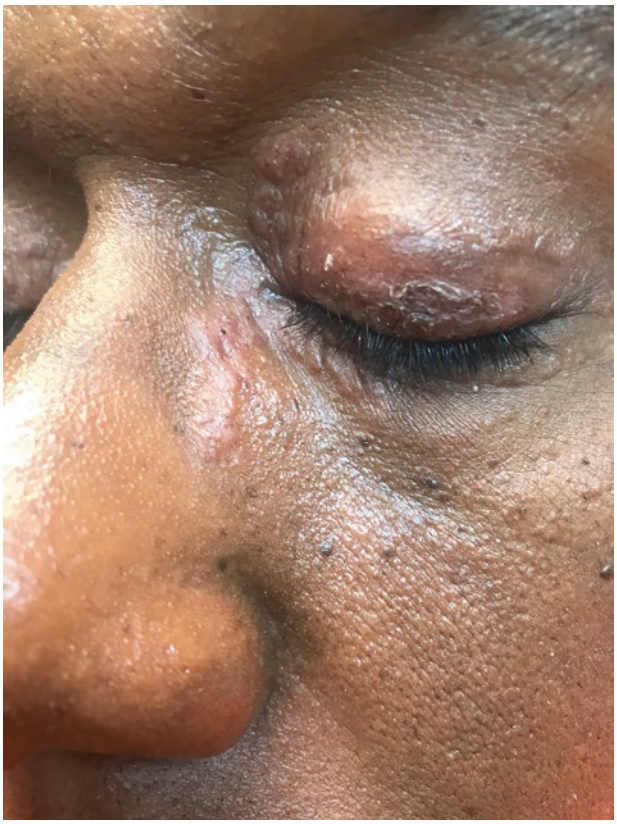

Fig. 4.131 Sarcoidosis

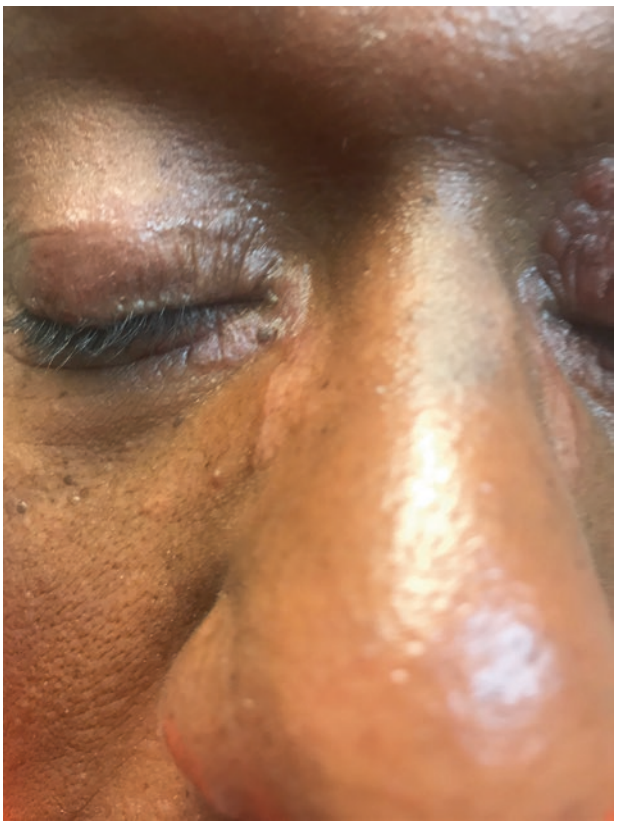

Fig. 4.132 Sarcoidosis

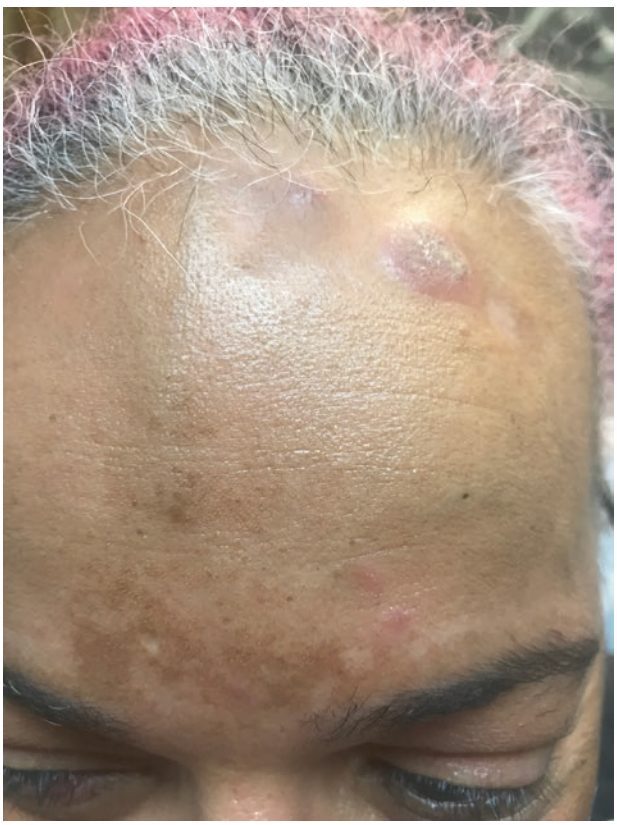

Fig. 4.133 Sarcoidosis

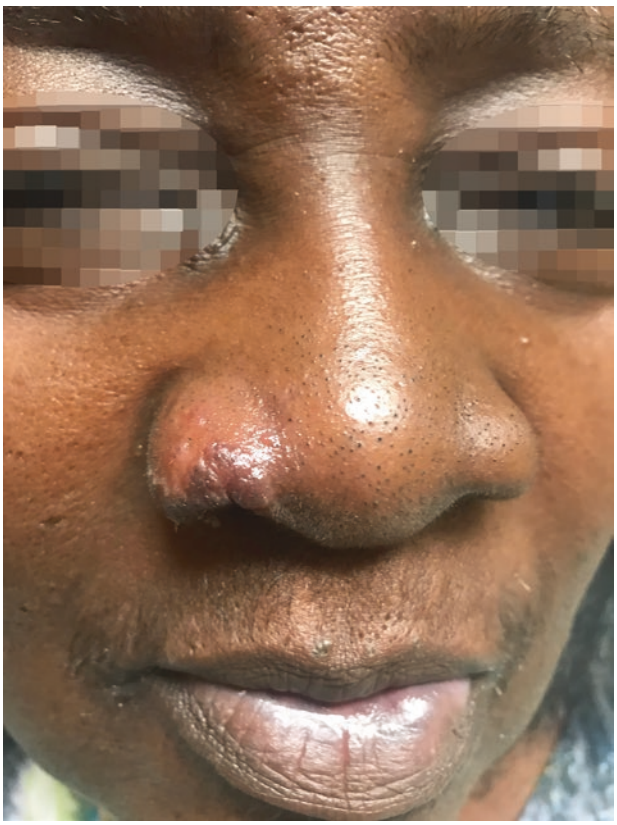

Fig. 4.134 Sarcoidosis

Fig. 4.135 Sarcoidosis, small papules around eyes

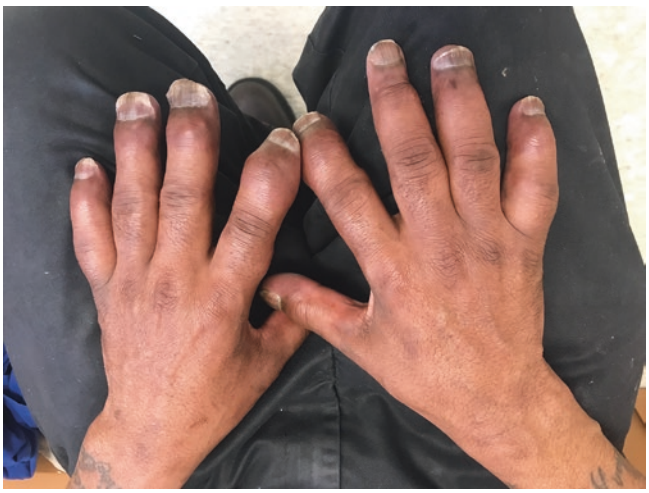

Fig. 4.136 Osseous sarcoidosis

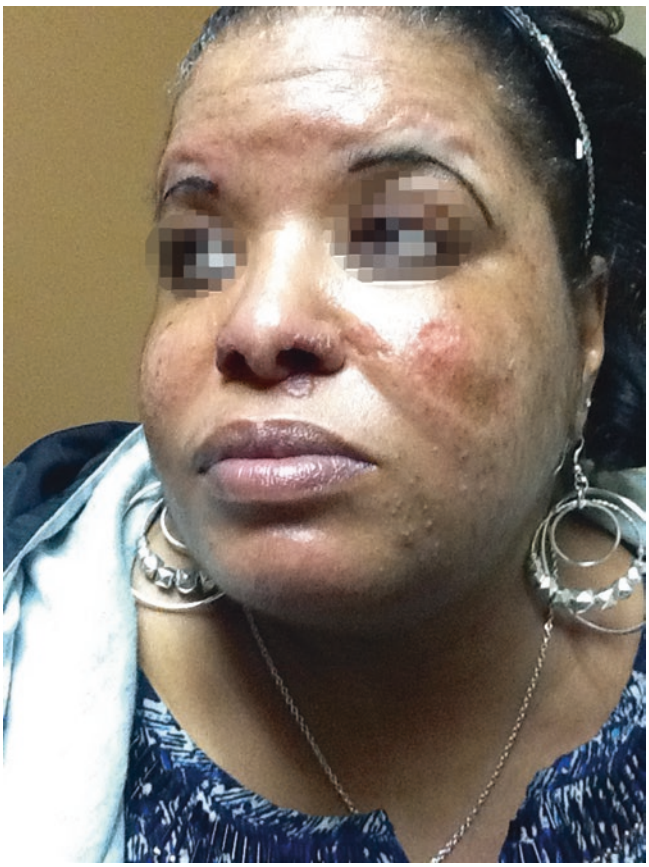

Fig. 4.137 Hypotrophic sarcoidosis

Fig. 4.138 Hypotrophic sarcoidosis

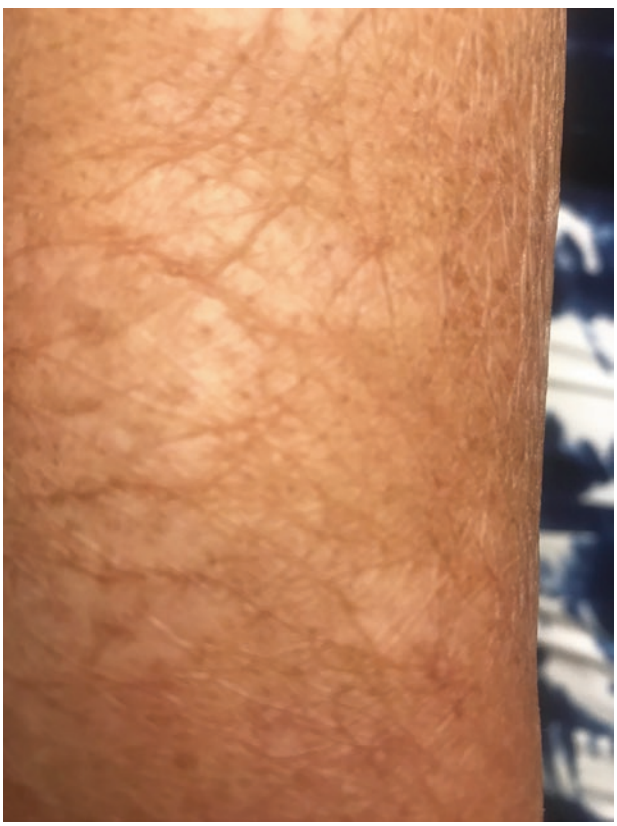

Fig. 4.139 Hypotrophic sarcoidosis

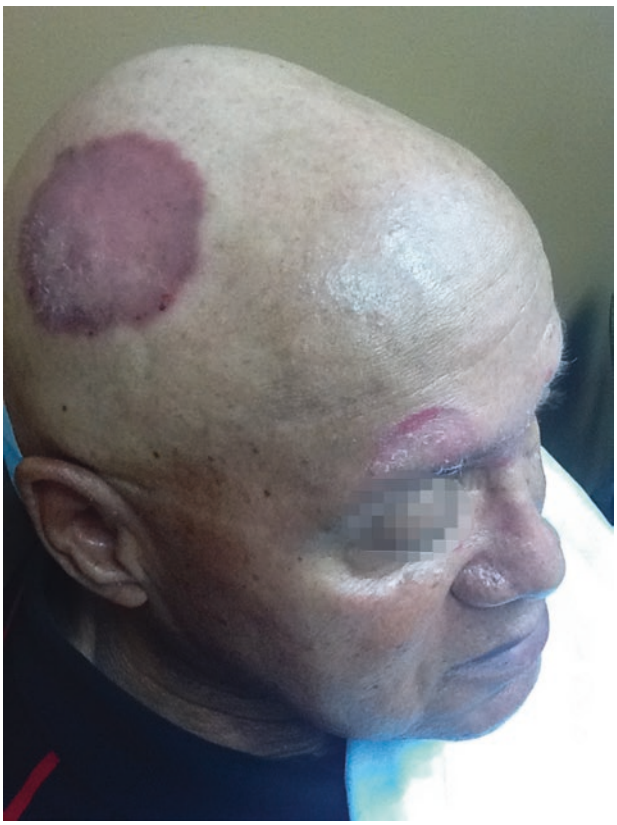

Fig. 4.140 Annular sarcoidosis

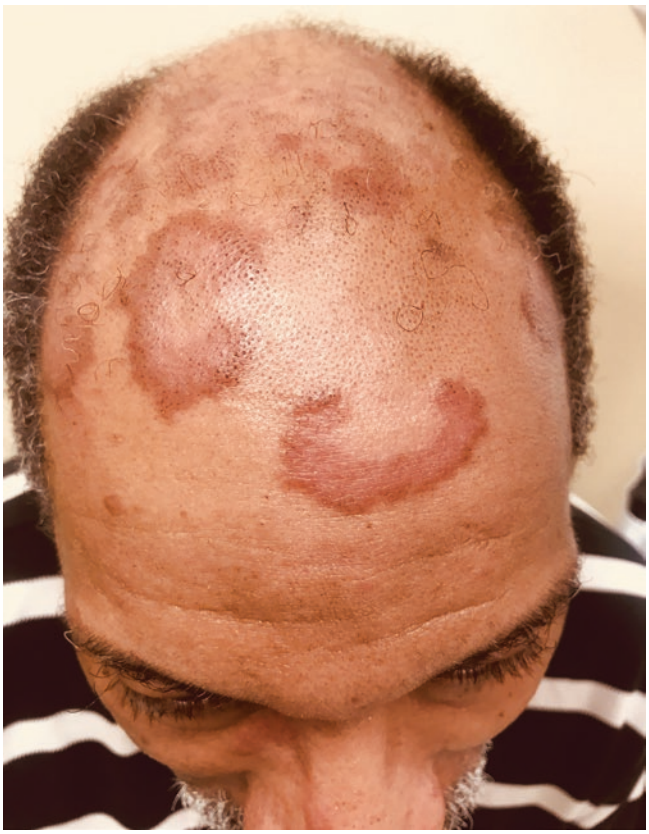

Fig. 4.141 Annular sarcoidosis

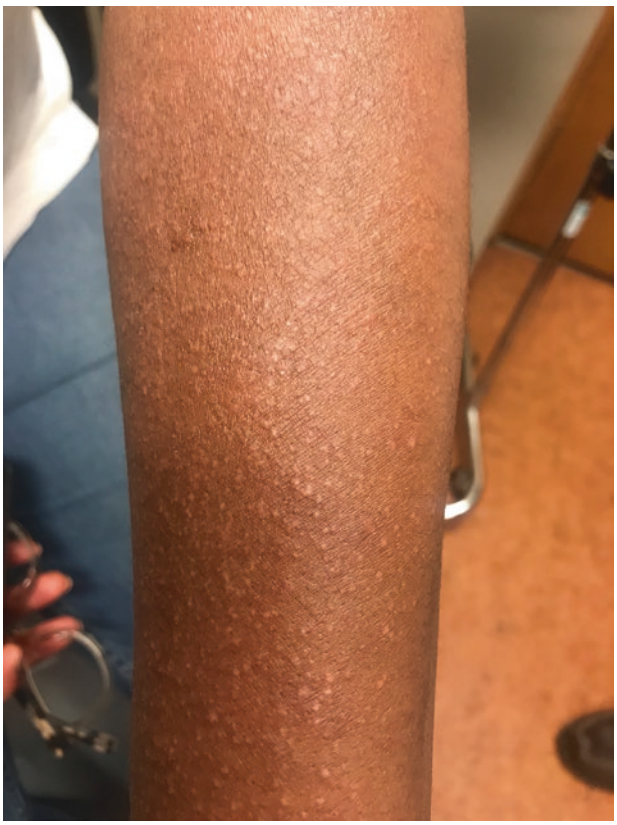

Fig. 4.142 Hypopigmented sarcoidosis

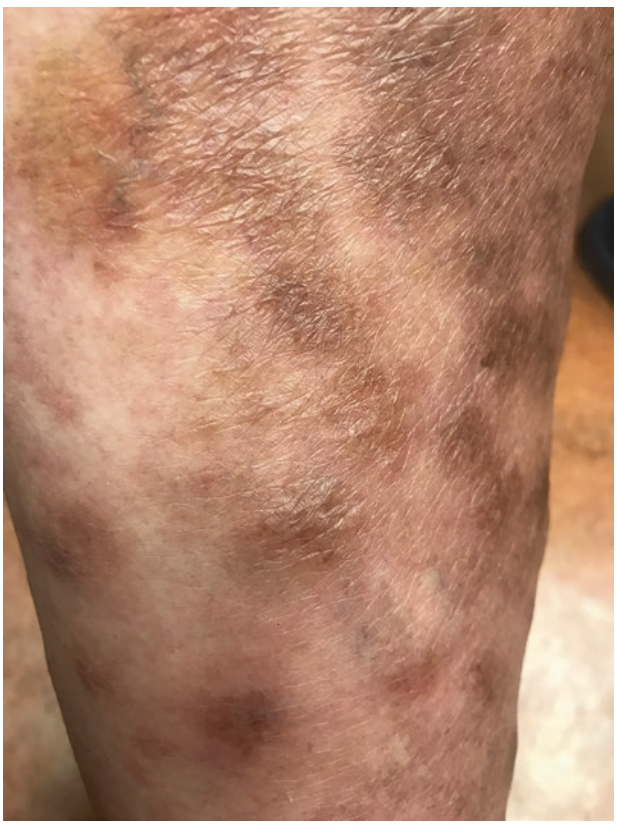

Fig. 4.143 Hypopigmented sarcoidosis

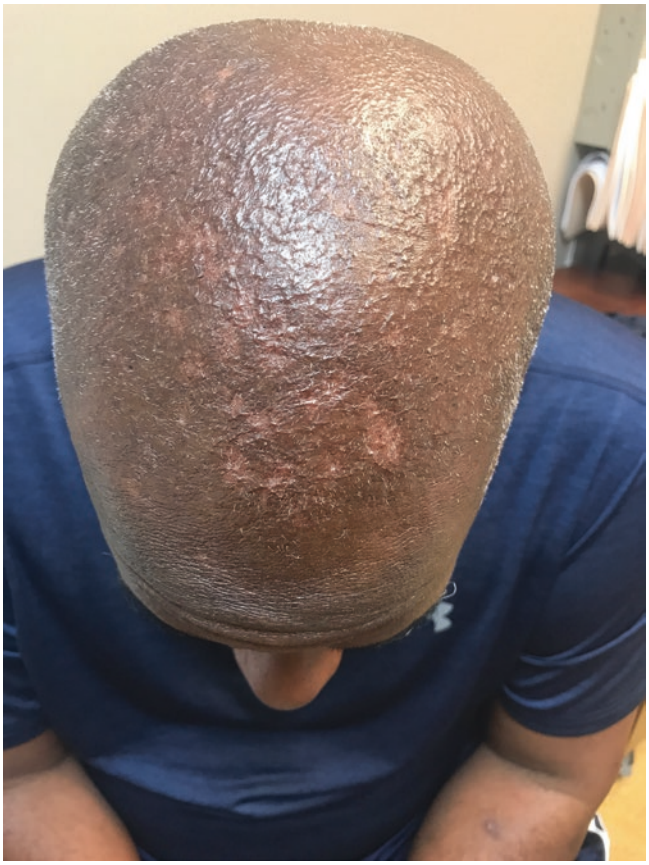

Fig. 4.144 Hypopigmented sarcoidosis

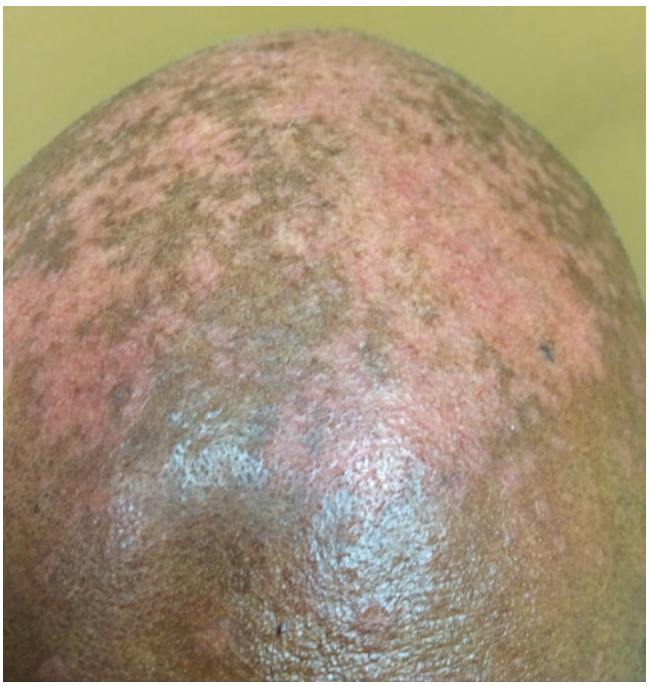

Fig. 4.145 Hypopigmented sarcoidosis

4.6 Lichen Sclerosus et Atrophicus

Lichen sclerosus et atrophicus (LS&A) is a chronic, inflammatory mucocutaneous disease of genital and extragenital skin. Over 90% of disease affects the anogenital region. Females are afflicted more often than males. Autoimmune and genetic factors play a role in the pathogenesis of LS&A. LS&A tends to be a chronic progressive or relapsing and remitting condition that may progress to atrophy, destructive scarring, functional impairment, and malignant evolution.

4.6.1 Presentation in Black Skin

In blacks, extragenital LS&A typically affects the buttocks, thighs, submammary skin, neck, back, chest, shoulders, axillae, and wrists. Extragenital LS&A is more common in blacks. It starts as polygonal, blue-white papules, which coalesce to form atrophic plaques (Figs. 4.146–4.149). It is less symptomatic than genital LS&A.

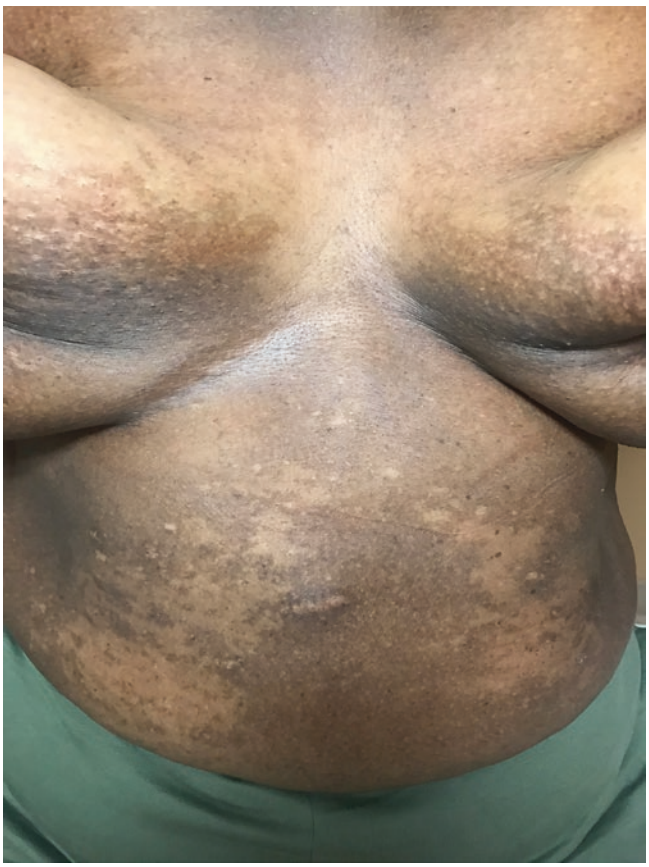

Fig. 4.146 Lichen sclerosus et atrophicus (LS&A)

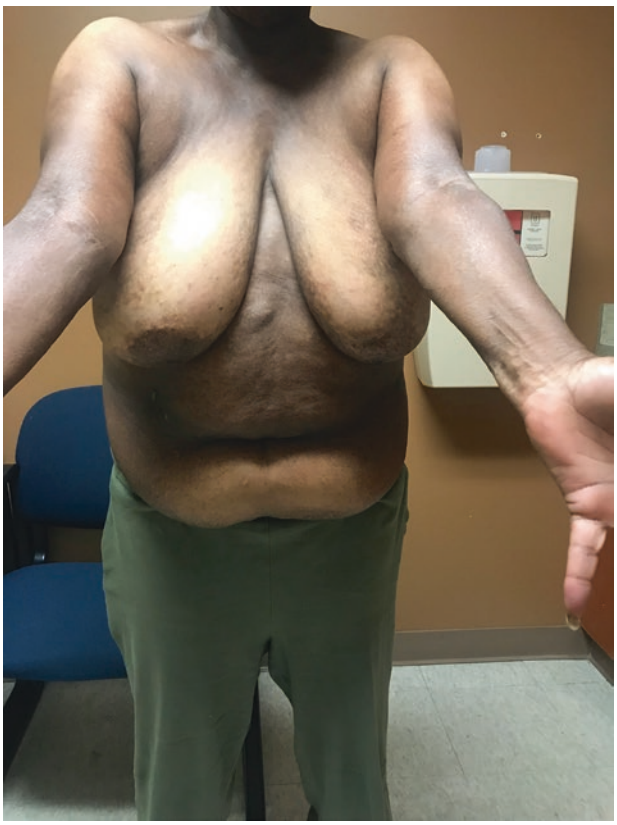

Fig. 4.147 LS&A

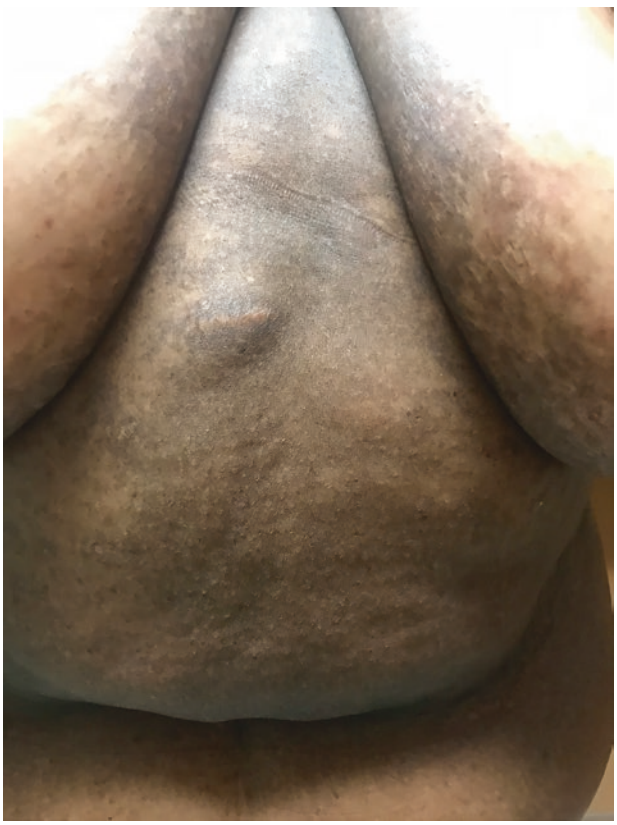

Fig. 4.148 LS&A

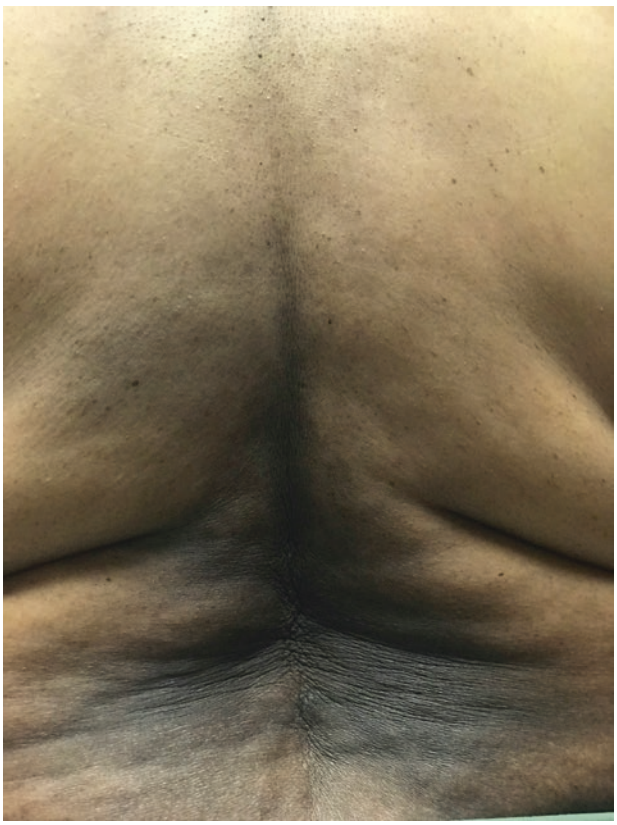

Fig. 4.149 LS&A

4.6.2 Treatment and Management

Potent topical steroids are the treatment of choice for LS&A, with topical calcineurin inhibitors often used in conjunction or as an alternative. There is no cure; control of disease is the goal. Phototherapy may be used in refractory cases. Anogenital LS&A is associated with an increased risk of squamous cell carcinoma, so regular surveillance examinations are recommended.

4.7 Dermatomyositis

Dermatomyositis (DM), an idiopathic inflammatory myopathy, affects individuals of all ages, but it demonstrates a bimodal age distribution, mostly affecting juveniles and adults. In adults, it is associated with an increased risk of malignancy. Dermatomyositis may be amyopathic, but when muscles are involved, proximal muscle weakness is most prominent.

4.7.1 Presentation in Black Skin

Dermatomyositis presents with violaceous poikiloderma, defined by hypopigmentation, hyperpigmentation, telangiectasias, and atrophy. This is usually present on photo-exposed areas such as the upper chest and back (known as the "shawl sign"), the face, and the scalp (Figs. 4.150–4.155). Plaques

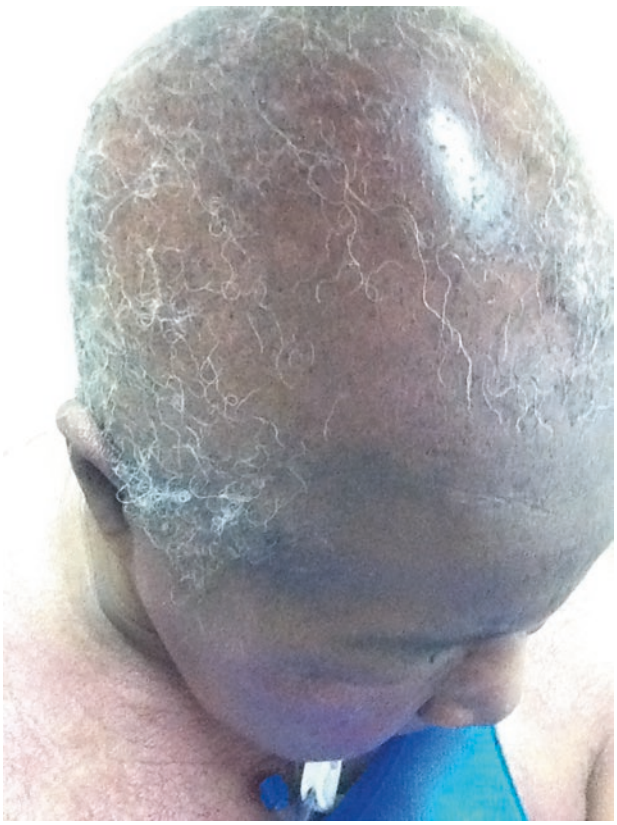

Fig. 4.150 Dermatomyositis

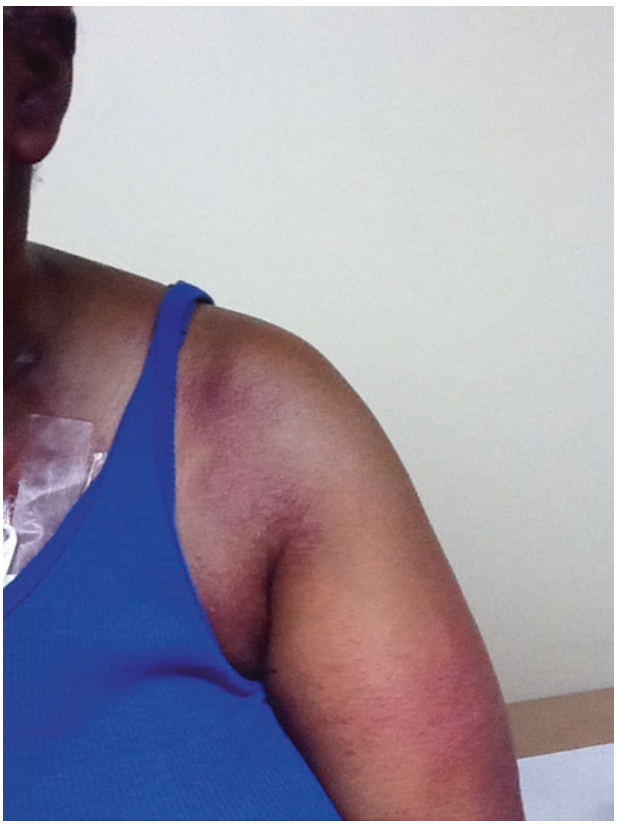

Fig. 4.152 Dermatomyositis

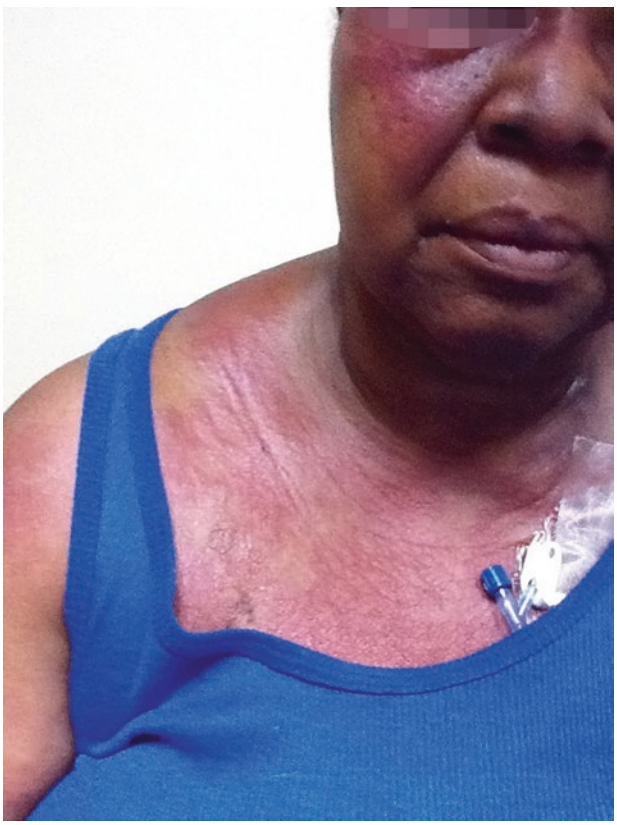

Fig. 4.151 Dermatomyositis

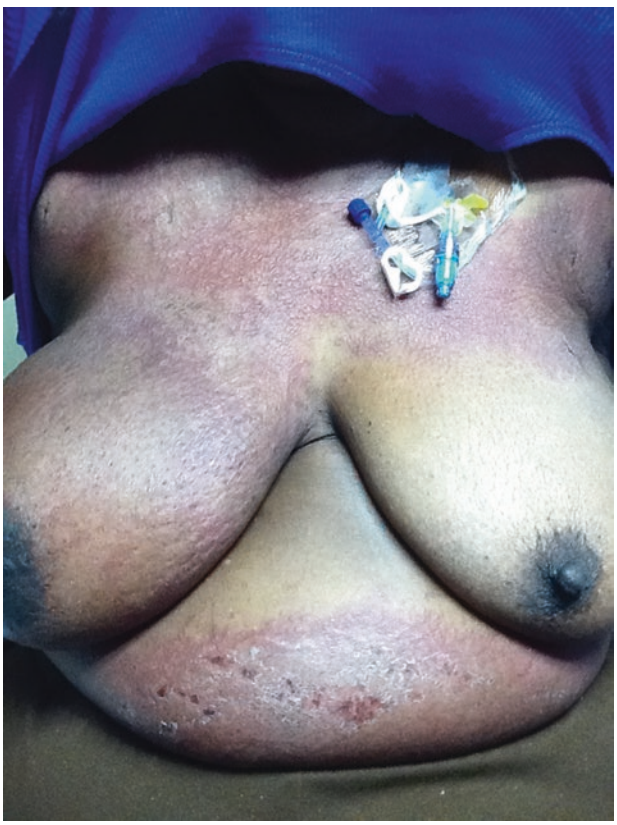

Fig. 4.153 Dermatomyositis

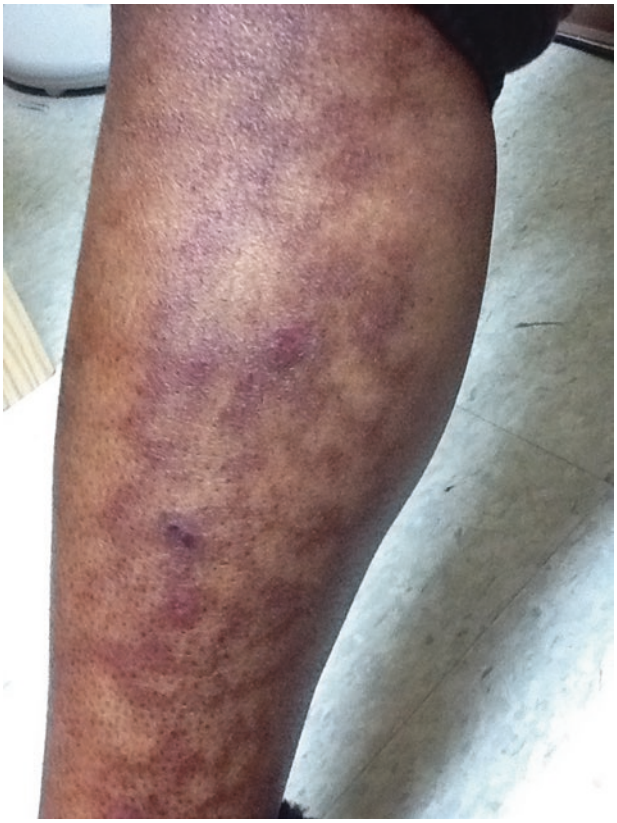

Fig. 4.154 Livedo reticularis in dermatomyositis

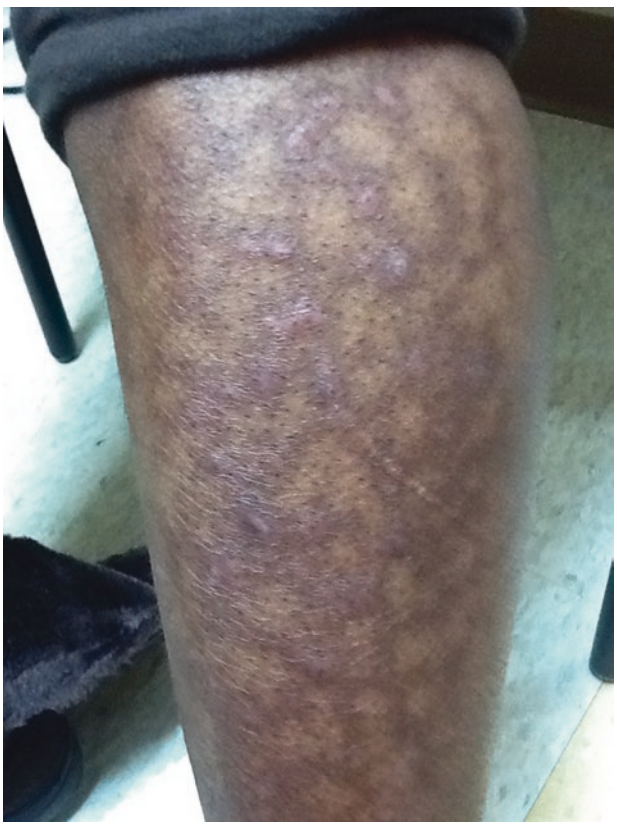

Fig. 4.155 Livedo reticularis in dermatomyositis

also present periorbitally ("heliotrope sign"), and over elbows, knees, and knuckles ("Gottron's papules"). Extensor surfaces are favored. Nailfold telangiectasias may be present. Lesions are often pruritic. Livedo reticularis can be seen in dermatomyositis (Fig. 4.154 and 4.155).

4.7.2 Treatment and Management

Immunosuppression is key in controlling dermatomyositis. Systemic corticosteroids are most often used as first-line treatment. Methotrexate, intravenous immunoglobulin, mycophenolate mofetil, azathioprine, calcineurin inhibitors, and cyclophosphamide, among others, may be used as second-line or adjunctive therapies. Age-appropriate cancer screening should be performed for all adults with dermatomyositis.

4.8 Sweet Syndrome

Sweet syndrome, also known as acute febrile neutrophilic dermatosis, is a neutrophilic dermatosis with tender cutaneous lesions that may be associated with malignancy, infection, inflammatory disorders, and medications. It usually presents in the 5th to 6th decade of life and is more common in women than in men. Classic Sweet syndrome may be idiopathic or associated with infection, vaccination, inflammatory disorders, or pregnancy. Acute myelogenous leukemia is the most commonly associated hematologic malignancy, and genitourinary tumors are the most common solid malignancy. Granulocyte-colony stimulating factor is the most commonly implicated drug. Sweet syndrome is a diagnosis of exclusion.

4.8.1 Presentation in Black Skin

Sweet syndrome presents abruptly with tender erythematous papules, nodules, or plaques. Lesions usually present asymmetrically on the extremities, face, neck, and upper trunk (Figs. 4.156–4.160). Fever, malaise, or arthralgia may precede cutaneous findings. Pustular and bullous variants exist. Extracutaneous involvement is rare, but it may present with ocular, cardiac, pulmonary, gastrointestinal, or neurologic findings.

4.8.2 Treatment and Management

Systemic corticosteroids are the treatment of choice for Sweet syndrome; improvement in response to steroids is a diagnostic criterion. Alternative treatments include dapsone, colchicine, and potassium iodide.

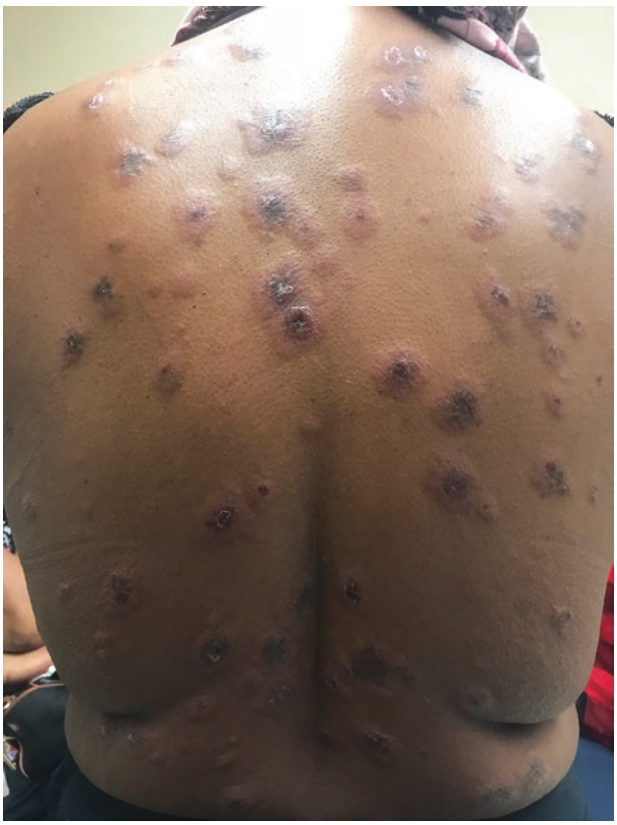

Fig. 4.156 Sweet syndrome

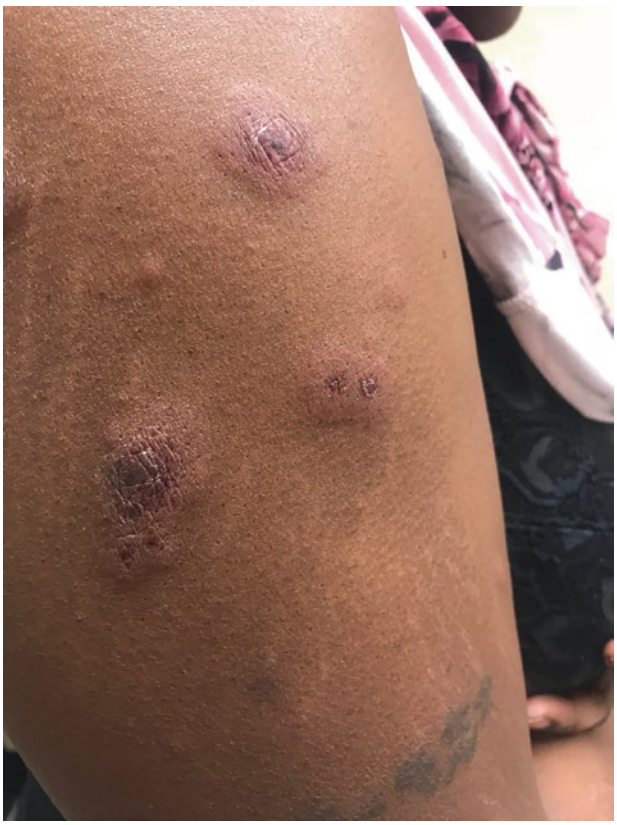

Fig. 4.158 Sweet syndrome

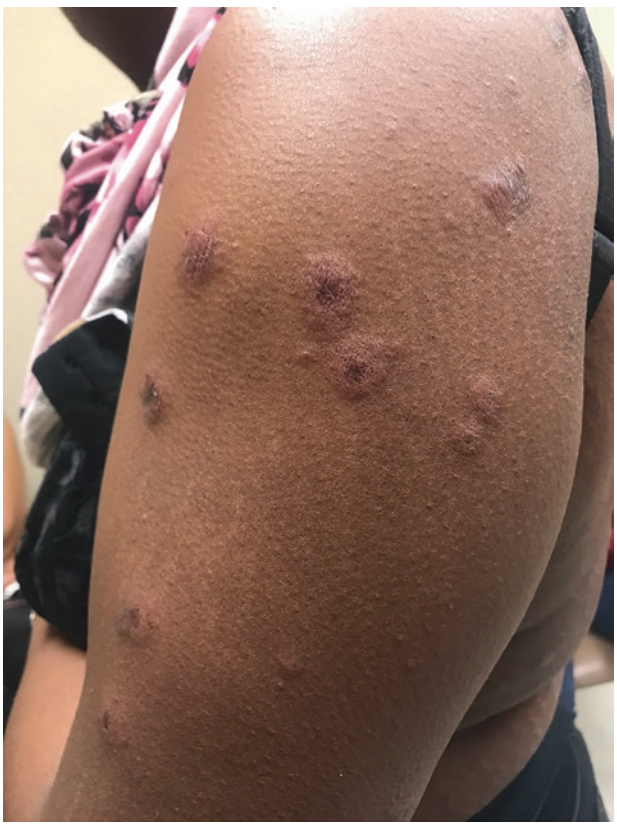

Fig. 4.157 Sweet syndrome

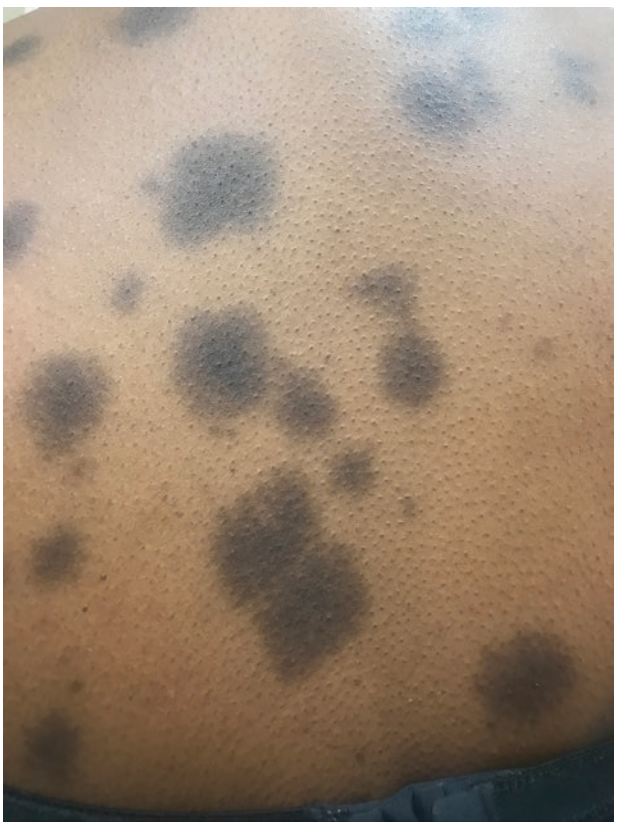

Fig. 4.159 Sweet syndrome post treatment

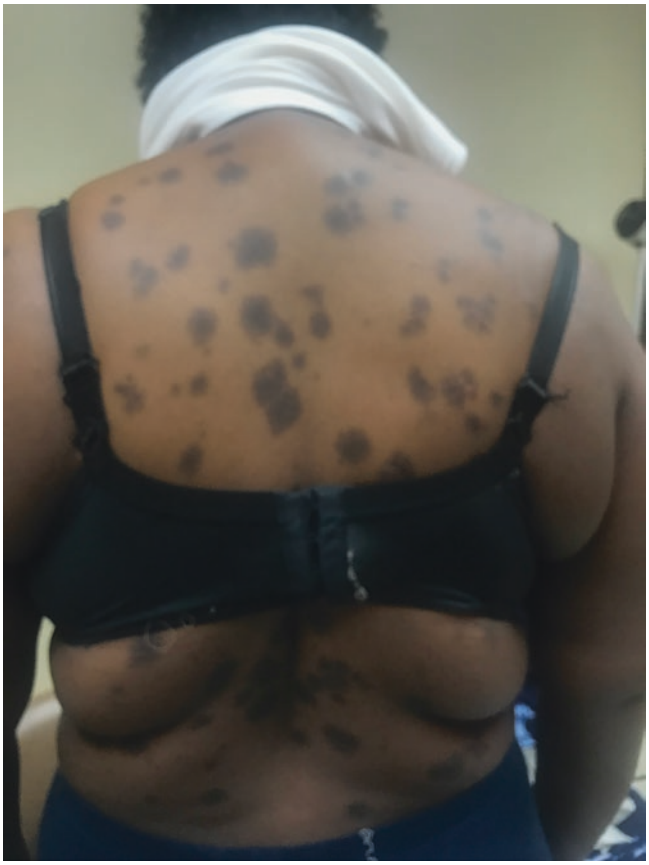

Fig. 4.160 Sweet syndrome post treatment

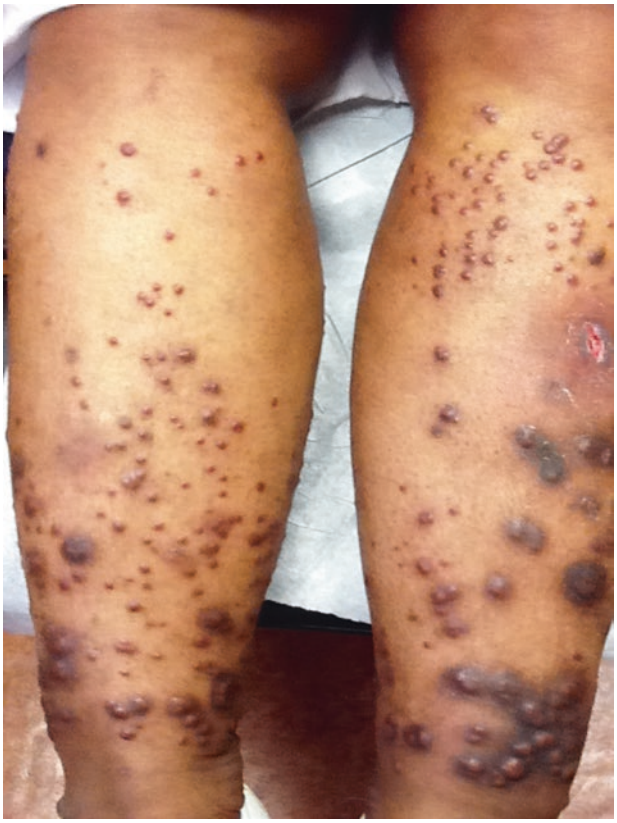

Fig. 4.161 Erythema elevatum diutinum (EED)

4.9 Erythema Elevatum Diutinum

Erythema elevatum diutinum (EED) is a form of leukocytoclastic vasculitis without a known etiology, though it has been associated with immunosuppression and infection. HIV infection has been highly associated with EED. There is no racial predilection, but it is more common in females than in males.

4.9.1 Presentation in Black Skin

In black skin, EED presents with dark or black nodules mostly over the joints and external surfaces, with some pruritis (Figs. 4.161–4.164).

4.9.2 Treatment and Management

Treatment of EED depends on the treatment of the underlying disease. Symptomatic treatment of pruritis with a topical steroid relieves the condition. Dapsone is the first-line treatment, and oral steroids also can be used.

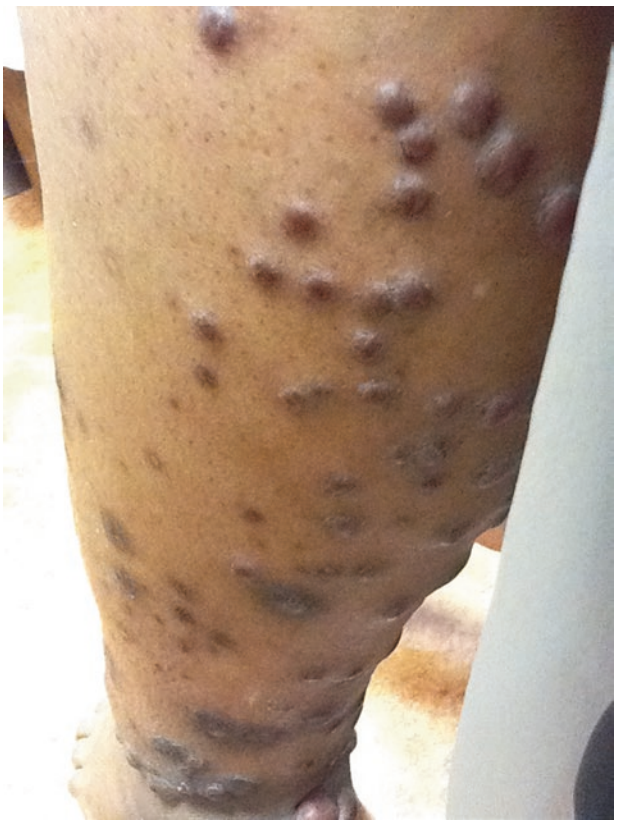

Fig. 4.162 EED

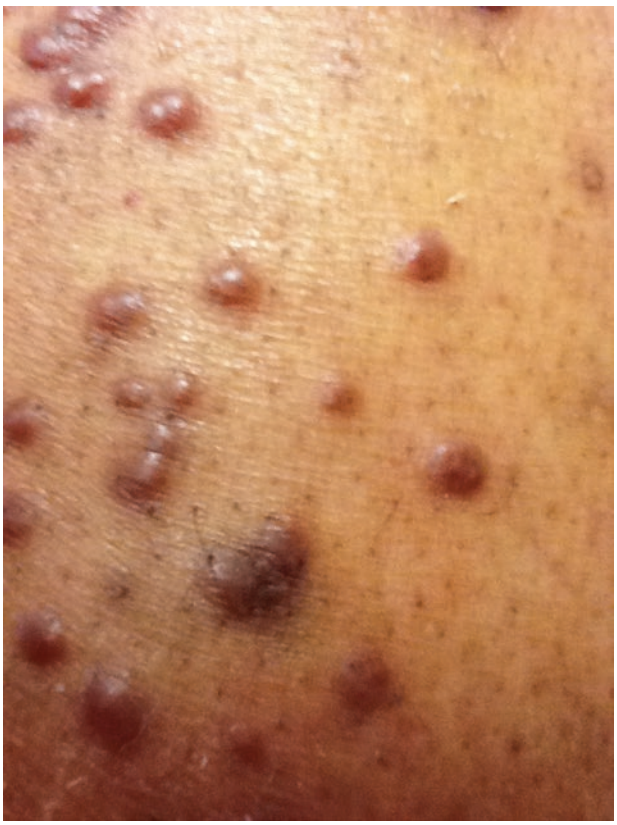

Fig. 4.163 EED

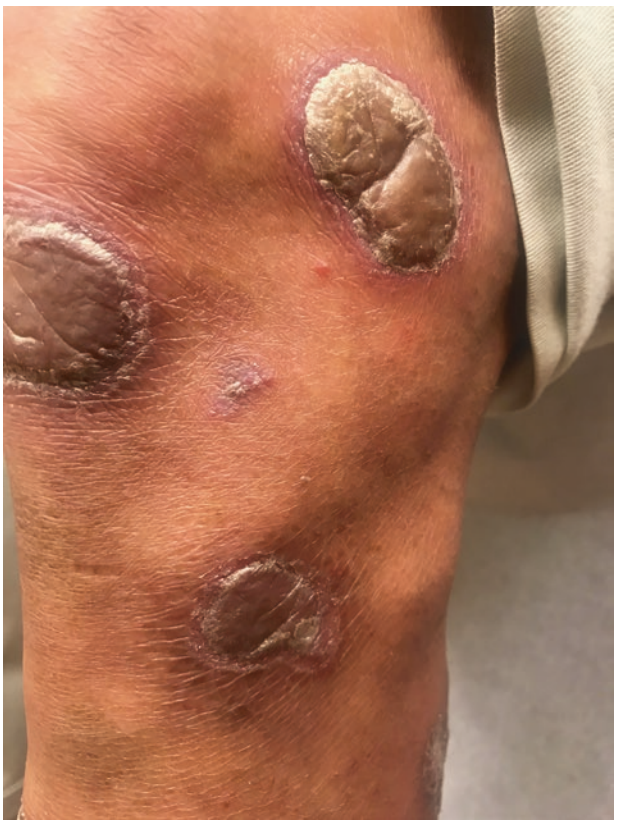

Fig. 4.164 EED

4.10 Granulomatous Reaction to Fillers

Buttock augmentation, either through silicone implants or fat grafting, is used to lift or improve the contour or shape of the buttocks. First popularized in Brazil, buttock augmentation is increasingly common across many cultures, especially in patients of African descent. Injection of liquid silicone or other non–FDA-approved materials to enhance the appearance of the buttocks can lead to the development of tissue-augmentation granulomas.

4.10.1 Presentation in Black Skin

Although cosmetic procedures are not within the scope of this atlas, we present here images of the side effects of buttock augmentation. It is important to mention that none of the patients presented here had their procedures done in a doctor's office or by a licensed physician. The injected substances that led to granuloma formation were silicon and, in one case, cement (Figs. 4.165 and 4.166). The granulomas

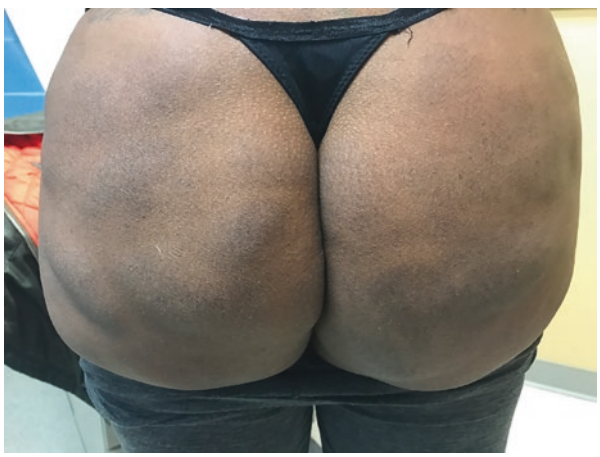

Fig. 4.165 Granuloma due to silicon injection

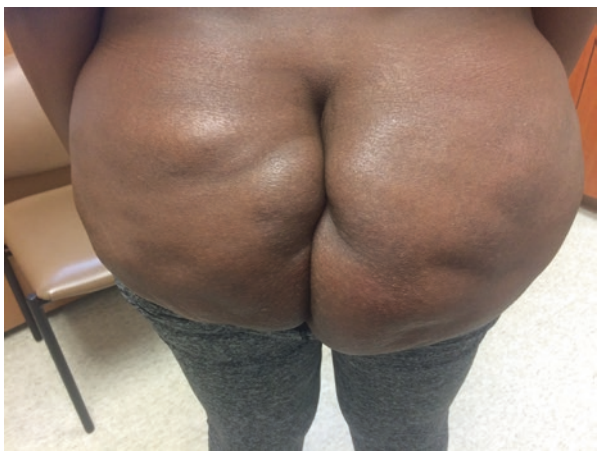

Fig. 4.166 Granuloma due to silicon injection

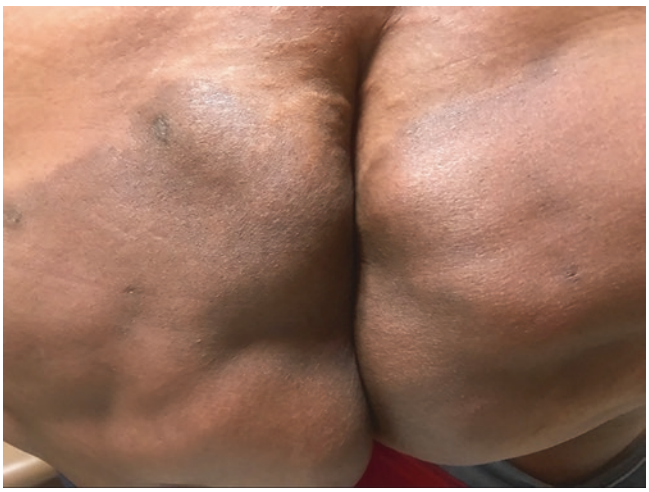

Fig. 4.167 Granuloma due to silicon injection and unknown substance

that formed in the buttocks were painful and disfiguring. Biopsy of the lesion confirmed foreign body granulomas with silicon present in all the cases (Fig. 4.167).

4.10.2 Management of Tissue Augmentation Granulomas

High-potency interlesional steroids helped to decrease the size and symptoms of these granulomas.

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