8 Inflammatory Disorders: Psoriasis, Lichen Planus, Pityriasis Rosea, and Sarcoidosis

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Psoriasis

Psoriasis vulgaris is a chronic, multifactorial, hyperproliferative skin disease. Arthritis may be associated with skin disease in approximately 30% of patients. Psoriasis appears to be most prevalent in northern European populations and is thought to be observed less frequently in patients with skin of color [1]. A population-based study in the United States in 2005 showed that although psoriasis is less common in African Americans than in Caucasians, it is not rare and carries a substantial burden in both groups. In this study, the prevalence of psoriasis was 2.5% in Caucasians and 1.3% in African Americans. African Americans had an approximately 52% reduction in the prevalence of psoriasis compared with Caucasians [2]. In another cross-sectional study using National Health and Nutrition Examination Survey data from 2009 to 2010, the psoriasis prevalence was highest in Caucasians at 3.6%, followed by African Americans (1.9%), Hispanics (1.6%), and others (1.4%) [3]. The psoriasis prevalence is estimated to be approximately 0.3% in Asians (18).

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Pathophysiology

The pathophysiology of psoriasis involves genetic and immune-mediated factors leading to immune dysregulation and hyperproliferation of epidermal keratinocytes with increased epidermal cell turnover. Triggers include infectious episodes (i.e., staphylococcus, streptococcus, HIV), traumatic insult (i.e., surgery), alcohol, or medications (beta-blockers, steroid withdrawal, lithium, antimalarials) [4]. Once triggered, there appears to be substantial leukocyte recruitment to the dermis and epidermis resulting in the characteristic psoriatic plaques [5]. The major inflammatory cells are activated T cells that induce changes in keratinocytes, vascular endothelial cells, and other inflammatory cells. The antigen HLA-Cw6 has the strongest association with psoriasis and correlates with early age at onset and a positive family history [6]. HLA-Cw6 is found in approximately 50–80% of Caucasian psoriatic patients [7]. However, in one study, only 17–18% of Chinese [8] and Taiwanese [9] psoriatic patients, respectively, were found to have the HLA Cw6 allele. Outcome-based studies often suggest that patients with more severe psoriasis have an increased risk of major cardiovascular

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events independent of traditional risk factors [10]. The onset or worsening of psoriasis with weight gain and/or improvement with weight loss has also been observed [11].

Clinical Manifestations

Psoriasis presents similarly across skin types. Psoriasis is characterized by erythematous, well-demarcated plaques with silvery scale (Fig. 8.1a, b). Lesions are most commonly found on the elbows, knees, scalp, umbilicus, and intergluteal folds. The palms and soles may contain sterile pustules and thick scale. External trauma (rubbing, scratching, surgery) may lead to the Koebner phenomenon [5]. In darker skin, the distribution is similar; however, plaques may be violaceous with gray scale, and erythema is sometimes difficult to identify (Fig. 8.1c). Psoriasis has two peak age ranges; early onset occurs in the second decade, and late onset peaks between the ages of 50 and 60 [12].

Guttate psoriasis is characterized by the rapid onset of red, salmon-colored papules and plaques that may be covered with fine silvery scale. In darker skin, violaceous and gray colors predominate. Guttate psoriasis most commonly occurs in

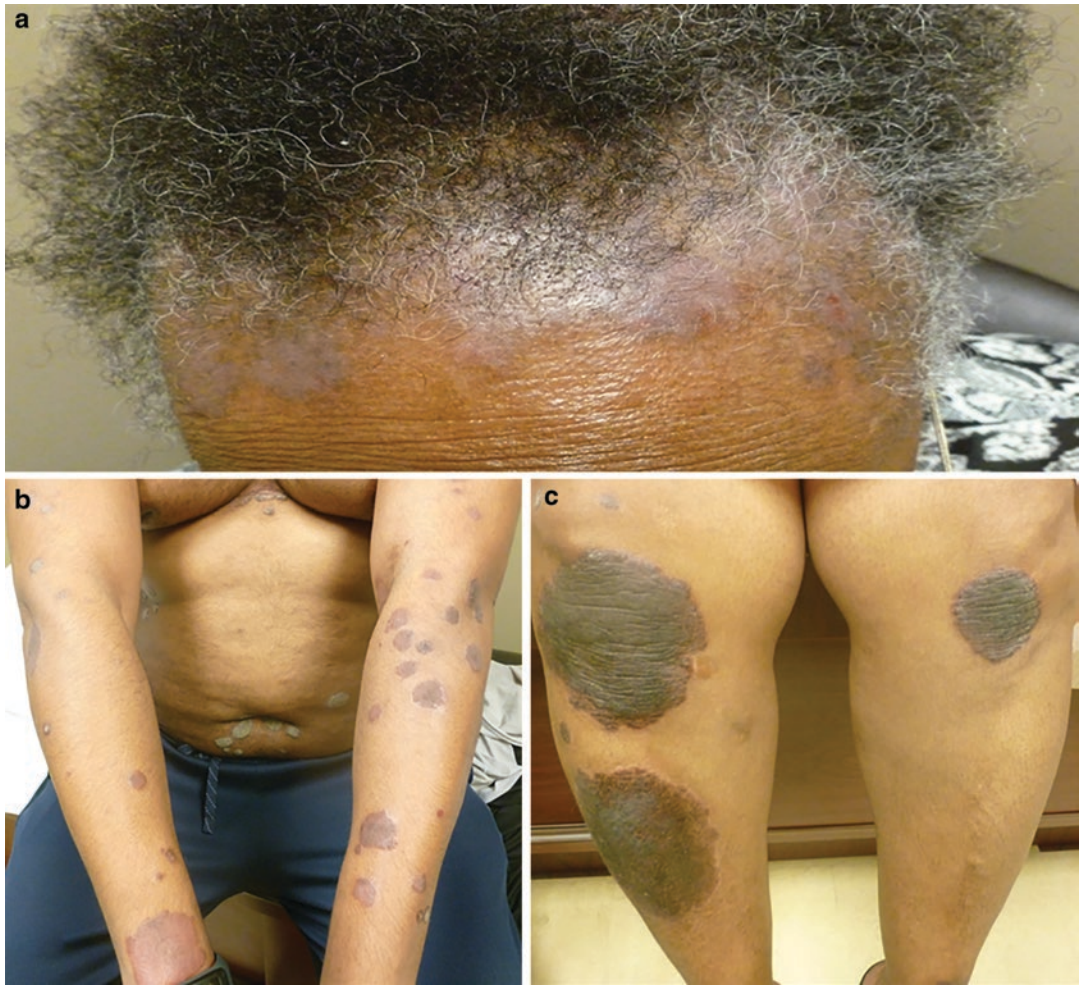

Fig. 8.1 Psoriasis. Erythematous, well-demarcated plaques with silvery scale are noted on the scalp (**a**) and arms (**b**). In darker skin, the distribution is similar; however, plaques are often brown or violaceous, and erythema is sometimes difficult to appreciate (**c**)

young patients and is often associated with viral or streptococcal pharyngitis. Pustular psoriasis is characterized by groups of sterile pustules at the periphery of stable plaques. Pustular psoriasis may occur as a primary manifestation of palmoplantar psoriasis and can be confused with dyshidrotic eczema. Generalized psoriasis, a potentially fatal disorder, is characterized by large sheets of pustules on a fiery red base. It is seen in patients with extensive psoriasis who have been treated with systemic or intensive and prolonged topical corticosteroids. Patients often have systemic symptoms (fever, chills, or peripheral leukocytosis). Erythrodermic psoriasis is characterized by generalized redness, scaling, and warmth of the skin. Body temperature is often erratic, and patients are severely ill, secondary to sudden withdrawal of long-term steroids [12].

Psoriasiform nail findings include nail pitting (most common finding), leukonychia, longitudinal grooves and ridges, the oil drop sign, and subungual hyperkeratosis. Psoriatic arthritis, affecting approximately 10–30% of those with skin disease, produces stiffness, pain, and progressive joint damage, usually in the hands and feet [12].

Treatment

Treatment for psoriasis is similar across ethnicities. Mild to moderate psoriasis is treated with topical corticosteroids, vitamin D derivatives, retinoids, anthralin, and tar-based formulations. For psoriasis that is nonresponsive to topical treatments and for moderate to severe psoriasis, systemic treatment is often needed. Options include systemic retinoids, methotrexate, cyclosporine, and apremilast [13]. Many of the systemic therapies for psoriasis manipulate the function of the immune system and expose the patient to the risk of severe infections while blunting the body's response. In these patients, findings suggestive of minor infections must be taken seriously, and the risk versus the benefit of continuing the drug in the face of the infection must be weighed [13].

Biologic immune-modifying agents have revolutionized psoriasis therapy. Several are now available and block TNF-alpha, IL 12/23, and IL 17, all inflammatory cytokines involved in psoriasis pathogenesis. The risks of these biologic agents include infections, tuberculosis reactivation, and hematologic malignancies [14]. Therefore, the benefit of using these medications must be weighed against the side effects while selecting appropriate patients for treatment. Phototherapy may also be used to treat moderate to severe plaque psoriasis. There is a risk of increased pigmentation (tanning) and post-inflammatory hyperpigmentation in skin of color. Various ultraviolet light treatments are used, with UVB being the most common, although psoralen + UVB (PUVA) is still used [15]. The 308-nm excimer laser is also an effective and safe modality for localized plaques of psoriasis, with good results achieved in a relatively short time [16].

Lichen Planus

Lichen planus is an autoimmune inflammatory mucocutaneous condition that can affect the scalp, oral mucosa, skin, and nails. Lichen planus can be found in approximately 1% of adults [1]. There is no overt racial predisposition, and women develop the condition more than men. Two-thirds develop the disease between 30 and 60 years old; however, lichen planus can occur at any age [17, 18]. Oral lichen planus is found in 50–70% of cutaneous lichen planus, and cutaneous lichen planus is found in 10–20% of oral lichen planus. One-fourth have solely mucosal involvement [18].

Pathogenesis

Lichen planus is a T cell-mediated autoimmune process against basal keratinocytes. Caspase 3 is often elevated in cutaneous and oral lesions, and it is suspected that apoptosis of basal keratinocytes as mediated by cytotoxic T cells is involved [19]. Five percent of hepatitis C patients have lichen planus. Medications that may cause lichen planus include beta-blockers, ACE inhibitors,

NSAIDs, antimalarials, quinidine, hydrochlorothiazide, gold, and penicillamine. Autoimmune liver disease, myasthenia gravis, and ulcerative colitis may also be associated with lichen planus [18]. There is a higher prevalence of serum autoantibodies in Chinese patients with oral lichen planus [20] and a strong correlation between the presence of hepatitis C and lichen planus in the Japanese. In one study, long-standing hepatitis C virus infection, hypoalbuminemia, and smoking were significant risk factors for the presence of oral lichen planus in patients [21]. In oral lichen planus, prolonged exposure to amalgam fillings has also been implicated. Many have regression of disease with removal of the metal [22].

Clinical Features

Cutaneous lichen planus is characterized by small, polygonal, violaceous, flat-topped papules that coalesce into plaques (Fig. 8.2). The surface is often shiny with a network of fine lines, also known as Wickham's striae. The Koebner phenomenon is commonly seen. Lesions often involve the flexor surfaces of the wrists and forearms, the dorsal surfaces of the hands, and the anterior aspect of the lower legs. In skin of color, the classic purple color may be black, gray, brown, or violaceous. If exacerbation occurs, it usually takes 2–16 weeks for maximal spread to occur. Lesions are intensely pruritic, often out of proportion to the amount of disease [18]. There are numerous variants of lichen planus (Table 8.1).

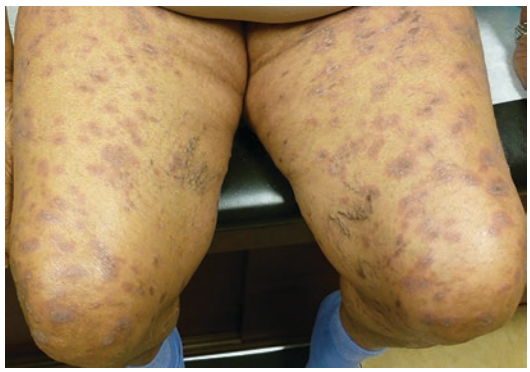

Fig. 8.2 Lichen planus. Polygonal, violaceous, flat-topped papules that coalesce into plaques with Wickham's striae are noted on the thighs

Lichen planus actinicus, also known as actinic lichen planus, is a photodistributed variant of lichen planus more common in darker-skinned individuals from subtropical climates and individuals of Middle Eastern, African, and Asian descent [18, 23]. Sun exposure is a triggering factor. The lateral aspect of the forehead is the most common involved area. It has an earlier age of onset and a longer course. There is a female predominance. Pruritus, scaling, nail involvement, and the Koebner phenomenon are often present [18, 24]. Lichen planus pigmentosus is another variant that is more common in Latin Americans and darker skin. Asymptomatic dark brown macules or patches in sun-exposed areas and flexural folds are observed (Fig. 8.3).

Treatment

Lichen planus is often self-limiting with lesions resolving after 1 year in most patients; however, treatment is often indicated to prevent post-inflammatory hyperpigmentation. Topical corticosteroids are first-line treatment. For lesions refractory to topical treatment or lesions that are more hyperkeratotic, intralesional or systemic corticosteroids may be indicated. Additional therapy for lesions that are refractory to topical treatment and are steroid sparing include acitretin, dapsone, methotrexate, hydroxychloroquine, cyclosporine, thalidomide, low molecular weight heparin, mycophenolate mofetil, and metronidazole. Narrowband UVB phototherapy may also be used [25]. It is important to check for exacerbating factors (e.g., medications and infections). Treatment of oral lichen planus is often more difficult and includes topical, intralesional, or systemic steroids, topical immunomodulators, retinoids, cyclosporine, griseofulvin, antimalarials, and methotrexate [22]. Removal of a contact allergen is also often indicated.

Table 8.1 Variants of lichen planus

Variants	Characteristics	Notes
Acute lichen planus	Eruptive lesions that occur most often on the trunk	
Annular lichen planus	Lesions with central inactivity or involution	Occurs in about 10% of patients
Atrophic lichen planus	Resolving lesions that are classically found on the lower leg	
Bullous lichen planus	Lesions that exhibit blisters within long-standing plaques	
Hypertrophic lichen planus	Lesions that present with thick hyperkeratotic plaques	Risk of squamous cell carcinoma, more common in Blacks
Lichen nitidus	Presents as tiny skin-colored or hypopigmented papules involving the trunk or extremities	Most common in children
Lichen planopilaris	Follicular variant that can result in scarring alopecia of the scalp	
Lichen planus actinicus	Photodistributed variant	More common in darker-skinned individuals from subtropical climates and individuals of Middle Eastern, African, and Asian descent
Lichen planus pemphigoides	Manifests as bullae in previously uninvolved skin of patients with LP	Circulating IgG autoantibodies against BPAG2 (type XVII collagen)
Lichen planus pigmentosus	Asymptomatic dark brown macules or patches in sun-exposed areas and flexural folds are found	More common in Latin Americans and darker skin
Linear lichen planus	Linear lesions that occur spontaneously along the lines of Blaschko	
Lichen planus-lupus erythematosus overlap syndrome	Patients with characteristics of both lichen planus and lupus erythematosus	
Nail lichen planus	Nail thinning, ridging, fissuring, pterygium formation	
Oral lichen planus	White, reticular lacy patches on the buccal mucosa	More common in women; occurs in ~50–75% of patients; risk of squamous cell carcinoma
Ulcerative lichen planus	Consists of bullae and permanent loss of toenails	

Adapted from [18]

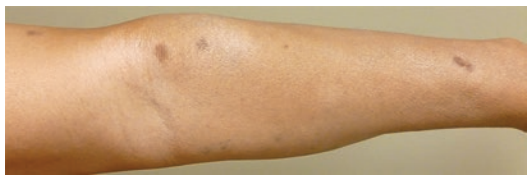

Fig. 8.3 Lichen planus pigmentosus. Dark brown macules and patches found on the arms

Pityriasis Rosea

Pityriasis rosea is a common, self-limiting, papulosquamous eruption that most often occurs in healthy children and young adults. The eruption usually lasts 6–8 weeks. Although there is no racial predilection for pityriasis rosea, patients with skin of color seem to have a more widespread distribution [26]. It has been noted to present atypically in Indian adolescent patients [27].

Pathophysiology

The exact cause of pityriasis rosea remains unknown. The eruption seems to be more prevalent during the spring and fall. A viral etiology has been suspected to induce pityriasis rosea due

to its occasional prodromal symptoms, a low rate of recurrence, and correlation with the changing of seasons. Oxidative stress may also play a role [28]. Human herpes viruses HHV 6 and HHV 7 have also been implicated [29]. A higher incidence of pityriasis rosea is noted among patients with decreased immunity (i.e., pregnant women and bone marrow transplant recipients). Pityriasis rosea during pregnancy may be associated with premature delivery and miscarriage, especially when it develops within the first 15 weeks of gestation [30].

Clinical Presentation

Pityriasis rosea typically presents with a solitary "herald patch" with a well-circumscribed border and collarette of scale on the back (Fig. 8.4) [28]. The herald patch may be absent in 10–15% of cases, especially in drug-induced pityriasis rosea. Within 2 weeks, a generalized truncal exanthem characterized by papules and patches occurs along the Langer lines in a Christmas tree distribution. The eruption most commonly occurs on the chest, abdomen, and back; the palms and soles are spared. Pruritus may occur. Patients with lighter skin tones portray pale pink lesions; the lesions are less noticeable on patients with darker skin. Approximately 5–10% of patients may have a prodrome of fever, chills, fatigue, headache, and lymphadenopathy. The eruption can last as long as 6–8 weeks [31].

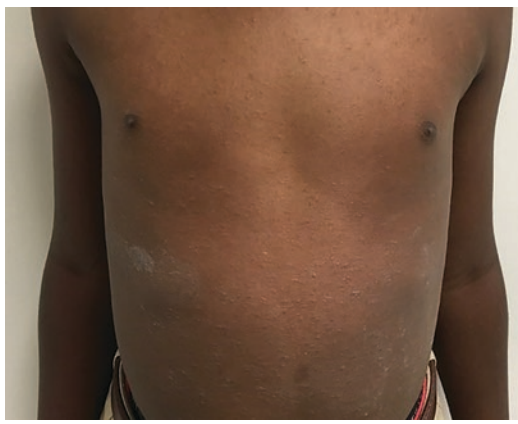

Fig. 8.4 Pityriasis rosea. Generalized papulosquamous eruption on trunk. Note "herald patch" on the right upper abdomen

Several atypical presentations of pityriasis rosea may occur. Papular pityriasis rosea tends to be more common in African Americans. African American patients may experience a more widespread distribution of lesions than Caucasian patients, and there is a higher risk of lymphadenopathy and scalp and face involvement [26, 32]. Patients with skin of color may have post-inflammatory hyperpigmentation that lasts for months. Pityriasis rosea usually will remain on the trunk; however, approximately 10–15% of patients may have oral lesions, such as ulcers, punctate hemorrhages, and petechiae. The majority of patients with oral lesions are patients with skin of color [33]. Pityriasis rosea is often known as the "great mimicker"; tinea corporis, tinea versicolor, atopic dermatitis, psoriasis, and syphilis are often included in the differential diagnosis.

Treatment

Pityriasis rosea is a self-limiting condition; however, topical steroids may help with pruritus and speed up resolution. Informational handouts and reassurance should be provided to the patient and/or their parents [34]. Patients with pityriasis rosea may require antihistamines to help with pruritus. Narrowband UVB phototherapy may also help severe cases of pruritus [35]. Hot water, fragrances, and harsh soaps can cause the eruption to worsen. Systemic steroids are generally not indicated, but they may help with severe disease with pruritus or vesicles. Although acyclovir has been shown to be ineffective against HHV6 and HHV7, some evidence suggests that acyclovir may be useful in the treatment of pityriasis rosea [36]. Patients with skin of color tend to have a high risk of post-inflammatory pigmentation, and topical treatments for dyschromia should be used after the eruption heals.

Sarcoidosis

Sarcoidosis is a multisystem, granulomatous, and inflammatory disease that, depending on the organ involved, has different clinical presentations, with varying degrees severity. The most common organs involved include the lungs, lymph nodes, and skin, with the skin being involved in 20–35% of cases [37]. The most common symptoms of systemic sarcoidosis are low-grade fever, weight loss, cough, dyspnea, chronic fatigue, arthralgia, and lymphadenopathy. Several studies have documented the higher incidence of sarcoidosis in African Americans compared to Caucasians [38]. In a population-based study conducted in the United States, Rybicki et al. found the age-adjusted incidence in African Americans to be 35.5/100,000 compared to 10.9/100,000 in Caucasians [39]. Sarcoidosis has also been found to occur at an earlier age and have a more severe course in African Americans compared to Caucasians [37]. In addition, Black patients are more likely to have cutaneous involvement than Caucasians [40]. The incidence of sarcoidosis may also be higher in other skin of color populations [41]. In a retrospective survey of Caucasian, Black West Indian/African, and Indo-Pakistan Asian patients treated at South London hospitals, the incidence of sarcoidosis was similar in Blacks and Asians, and these two groups had more widespread extrathoracic disease compared to Caucasians [42].

Pathophysiology

No causative agent has been identified for sarcoidosis; however, T cells play a central role in the disease [43], and both tumor necrosis factor (TNF) and receptors [44] are increased in patients with the disease. There are reports that genetically predisposed individuals who are exposed to different mycobacterial, viral, and other environmental antigens are susceptible to developing the disease, and this may initiate the immunologic cascade that produces the noncaseating granulomas most commonly found in the lung, skin, heart, and liver [45]. Recent genetic epidemiology studies from the Black Women's Health Study support the role of the BTNL2 gene and the 5q31 locus in the etiology of sarcoidosis, and also demonstrate that African ancestry is associated with disease risk [46].

Clinical Presentation

Cutaneous sarcoidosis may present as a part of systemic disease but may also only involve the skin. Skin involvement manifests with a wide variety of morphologies. Table 8.2 highlights the different morphologic types of cutaneous sarcoidosis.

Some of the more common lesions presenting in patients with skin of color include the maculopapular (Fig. 8.5), lupus pernio (Fig. 8.6), plaque, nodular ulcerative, and hypopigmented (Fig. 8.7) forms of sarcoidosis. Maculopapular sarcoidosis is the most common lesion seen in cutaneous sarcoidosis, especially in Black women [41]. Lupus pernio is usually more common in Black women with long-standing systemic disease [41]. Sarcoidosis is often called the "great imitator" because it can present with almost any morphology. Scarring alopecia and nail dystrophy may also occur.

The lungs are affected in nearly all cases (90%) of sarcoidosis and are characterized by granulomatous involvement of the interstitium, alveoli, blood vessels, and bronchioles. A third to half of all patients experience dyspnea, dry cough, and chest pain. Bilateral hilar adenopathy is the most common diagnostic radiographic finding. Overall, Blacks tend to have more severe lung disease as compared with Caucasians on presentation, a higher likelihood of progressive pulmonary dysfunction, and a poorer long-term prognosis [39, 41].

Table 8.2 Morphologic subtypes of cutaneous sarcoidosis

Subtype	Clinical features	Characteristics
Maculopapular sarcoidosis	Reddish brown macules and papules involving the cheeks, periorbital area, and nasolabial folds. Lesions may resolve without scarring	Most common manifestation of cutaneous sarcoidosis, especially in Black women. Commonly associated with hilar lymphadenopathy, acute uveitis, and parotid enlargement [40]
Lupus pernio	Red and violaceous, indurated papules, plaques, and nodules that usually affect the nose, lips, cheeks, and ears. Nasal ulceration and septal perforation may occur	More common in Black women [40]. Higher frequency of pulmonary and ocular disease
Plaque sarcoidosis	Annular, erythematous, brown, or violaceous, infiltrated plaques that may be atrophic or scaly. Angiolupoid plaques may have large telangiectasias. Plaques may heal with scarring and alopecia	Patients usually have more chronic and severe systemic involvement [45]
Subcutaneous nodular sarcoidosis (Darier-Roussy)	Nontender, firm, skin-colored, or violaceous mobile subcutaneous nodules commonly found on the trunk or extremities. Usually appear in the early stages of the disease. Nodules may resolve spontaneously	Usually associated with less severe systemic disease [45]
Scar-associated sarcoidosis	Scars from previous trauma, surgery, venipuncture, or tattoo may become infiltrated with sarcoidosis and show a red or violaceous color. These lesions may be tender	May appear early in the disease or parallel chronic systemic findings [45]
Erythema nodosum	Tender, erythematous subcutaneous nodules on the extremities (most commonly anterior tibias)	Associated with a good prognosis and spontaneous resolution of the disease. More common in Scandinavian women [45]
Lofgren syndrome	Triad of erythema nodosum, polyarthritis, and hilar adenopathy. Anterior uveitis, fever, ankle periarthritis, arthralgias, and pulmonary involvement	Acute syndrome with excellent prognosis [45]

Treatment

The goal of therapy is to alleviate symptoms by minimizing the inflammatory process. Treatment is selected based on the type of lesion, the cosmetic disfigurement, and the symptoms [37]. In general, patients presenting with cutaneous disease in the setting of systemic involvement benefit from being treated systemically. In cases where disease is localized to the skin, ultrapotent topical steroids or intralesional steroid injections are first-line treatments. Intralesional injections are most appropriate for papule or plaque sarcoidal lesions and aid in suppressing granuloma formation. Steroid-sparing topical agents such as the topical immunomodulators, topical tacrolimus and pimecrolimus, can be alternated with topical steroids to decrease the risk of steroid-induced skin atrophy or hypopigmentation.

Patients with severely scarring sarcoidosis, lesions refractory to local treatment, or those experiencing cutaneous and systemic involvement may require systemic corticosteroids. Minocycline or doxycycline may also be used as first-line treatment [47]. Widespread, cutaneous disease (especially lupus pernio, mucosal, and nail disease) may require oral corticosteroids or antimalarial agents, such as hydroxychloroquine or chloroquine. Antimalarial agents halt

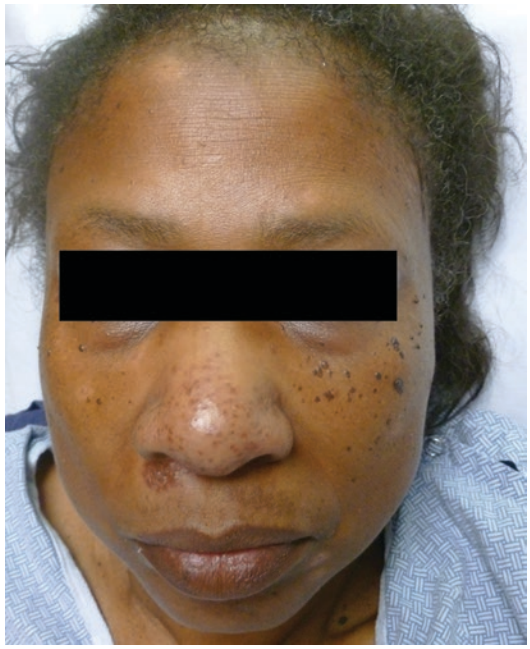

Fig. 8.5 Maculopapular sarcoidosis. Multiple red to violaceous macules are noted on the nose. Scattered violaceous papules coalescing into a thin plaque were noted on the upper cutaneous lip

the body's inflammatory response by preventing the antigen presentation necessary for the process of granuloma formation. Given the potential for ocular toxicity with antimalarial agents, patients should be followed by an ophthalmologist with an eye examination every 6–12 months to monitor for the development of corneal deposits and retinopathy. Patients of African, Mediterranean, or Southeastern Asian descent should be screened for glucose-6-phosphate-dehydrogenase deficiency before prescribing antimalarial medication to avoid precipitating a hemolytic episode [41, 48].

Recalcitrant disease may require the addition of methotrexate, azathioprine, or mycophenolate mofetil. Tumor necrosis factor (TNF) plays an important role in both formation and maintenance of the sarcoidal granulomas, and the TNF-alpha inhibitors, infliximab and adalimumab, have been used successfully in some patients with sarcoidosis [49]. However, it is important to note that there have been cases of TNF-alpha inhibitor-induced sarcoidosis [50].

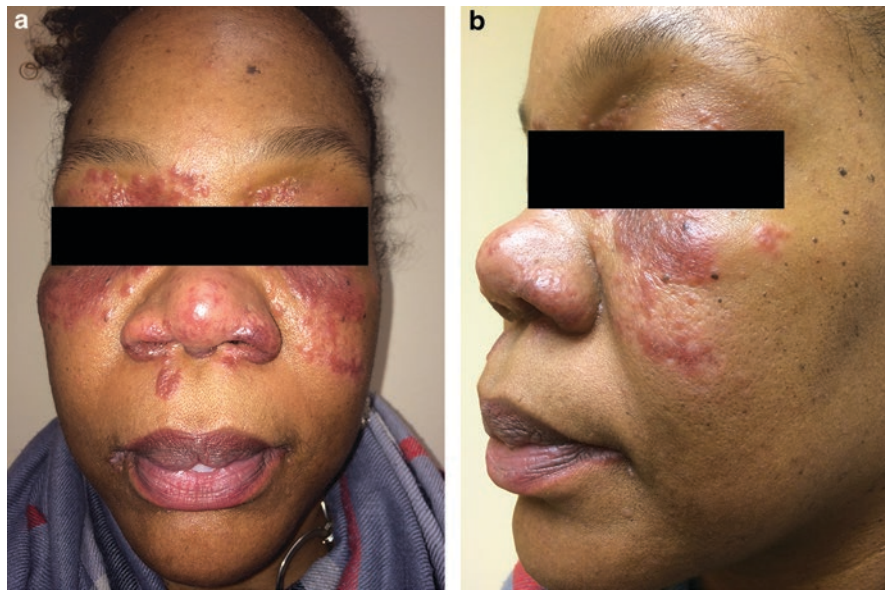

Fig. 8.6 (**a**, **b**) Lupus pernio. Multiple disfiguring violaceous papules and plaques are noted on the periorbital, malar cheeks, upper cutaneous lip, and bilateral nasal rims

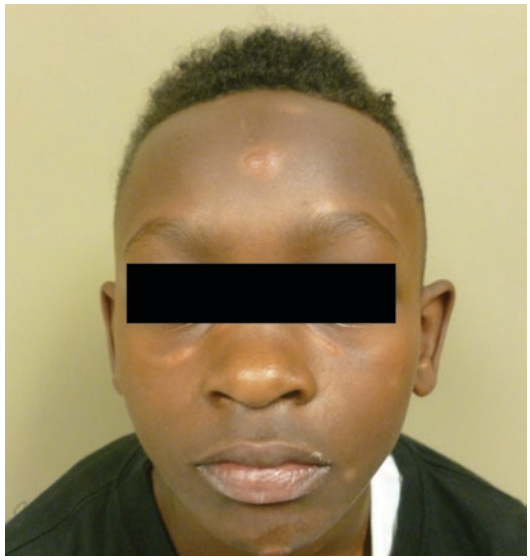

Fig. 8.7 Hypopigmented sarcoid. Scattered hypopigmented subcutaneous nodules are noted on the forehead, cheeks, and chin

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