FEATURE ARTICLE

NCPD 2.0 Contact Hours

Cutaneous Manifestations of Systemic Lupus Erythematosus

Deepa Ragesh Panikkath and Vaneet Kaur Sandhu

ABSTRACT: Inflammation driven by immune-mediated mechanisms can lead to a wide variety of skin manifestations. Cutaneous lupus erythematosus (CLE), for example, is an autoimmune connective tissue disease that may be limited to the skin or be part of the widespread multiorgan involvement seen in systemic lupus erythematosus. Clinical findings are divided into lupus-specific (acute, subacute, and chronic) and nonspecific skin lesions. The diagnosis of CLE requires a thorough physical examination and, in some cases, skin biopsy and laboratory evaluation for any underlying systemic involvement. CLE treatment is dependent on the cutaneous manifestations and severity of disease. In addition to lifestyle measures, particularly avoidance of sunlight, topical and systemic therapies have proven beneficial. Prognosis is varied by disease severity, although chronic CLE notoriously results in cosmetic damage. Early recognition and management of CLE is important as it may also be the presenting manifestation of systemic lupus erythematosus. Patient care includes education and close collaboration with primary care providers, dermatologists, and rheumatologists.

Key words: Cutaneous Lupus, Discoid Lupus, Lupus, SLE

Systemic lupus erythematosus (SLE) is an autoimmune disease with protean manifestations. Skin is the second most commonly involved organ system and may occur as an isolated manifestation or part of multiple manifestations of SLE. Approximately 85% of patients with SLE will show cutaneous manifestations at some point during their disease course, including the presenting symptom of SLE in some (Rothfield et al., 2006). Up to 28% of patients with cutaneous lupus erythematosus (CLE) will progress to systemic disease, and the interval between onset of skin disease and SLE diagnosis ranges from a few months to over 30 years (Cervera et al., 1993; Wieczorek et al., 2014). The current classification criteria for SLE, which are intended for research purposes and weighted by disease manifestation or laboratory criteria, include five cutaneous manifestations: oral ulcers, nonscarring alopecia, subacute cutaneous or discoid lupus, and acute cutaneous lupus. Fourteen points are attributed to these criteria, where a score of 10 or more classifies a patient with SLE (Aringer et al., 2019).

ETIOPATHOGENESIS

The development of CLE is multifactorial, including an interaction between genetic factors, the immune system, and environmental triggers. Major histocompatibility complex Class I and II alleles (HLA B8, DR3, DQA1, and DRB1) may confer susceptibility to CLE (Achtman & Werth, 2015). Complement deficiencies and polymorphisms in inflammatory cytokines such as tumor necrosis factor have been associated with CLE (Agnello et al., 1983, Levy et al., 1979).

Ultraviolet radiation is an important environmental trigger that induces keratinocyte apoptosis, autoantigen presentation, and production of autoantibodies and inflammatory cytokines that leads to tissue damage and scarring (Stannard & Kahlenberg, 2016).

CLASSIFICATION OF CLE

CLE is divided into lupus-specific and nonspecific lesions based on clinical and histologic features. Further classification of lupus-specific cutaneous disease is noted in Table 1 and includes acute CLE (ACLE), subacute CLE (SCLE), and chronic CLE (CCLE; Gilliam & Sontheimer, 1982). Clinical manifestations and prognosis differ between the different categories of CLE.

ACUTE CLE

ACLE is characterized by skin lesions that may be localized or generalized. Localized ACLE is the classic malar

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TABLE 1. Gilliam Classification of Specific Skin Lesions in CLE

- Acute CLE (ACLE)
  1. Localized ACLE
  2. Generalized ACLE
- Subacute CLE (SCLE)
  1. Annular SCLE
  2. Papulosquamous SCLE
- Chronic CLE
  1. Classic discoid lupus erythematosus (DLE)
     a. Localized DLE
     b. Generalized DLE
  2. Hypertrophic/verrucous DLE
  3. Lupus profundus/lupus panniculitis
  4. Mucosal DLE
     a. Oral DLE
     b. Conjunctival DLE
  5. Lupus tumidus
  6. Chilblain LE
  7. Lichenoid DLE

Adapted from Gilliam and Sontheimer (1982). CLE = cutaneous lupus erythematosus; LE = lupus erythematosus.

rash (butterfly rash) seen as symmetrical reddish flat or raised lesions over the cheeks and bridge of the nose. Sparing of the nasolabial fold is a characteristic feature. The rash is often induced by sun exposure and is transient and nonscarring. When present in the setting of systemic disease (up to 52% noted at the time of SLE diagnosis), malar rashes may parallel disease activity (Rothfield et al., 2006). The generalized form of ACLE occurs in a photosensitive distribution and involves the chest, shoulders, and exposed areas of the arms while sparing the knuckles (Figure 1). Lesions often heal without scarring but may be associated with pigmentary changes. A rare subtype of ACLE may manifest as bullous lesions and mucosal involvement, mimicking toxic epidermal necrolysis (Fabbri et al., 2003; Vera-Recabarren et al., 2010). Serum antinuclear antibody (ANA), anti-dsDNA, and anti-Smith antibodies are commonly seen in ACLE (Tebbe et al., 1997).

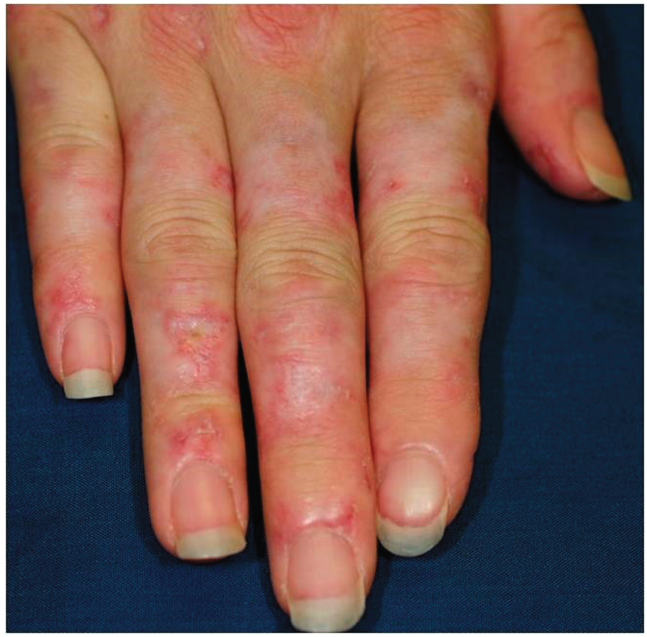

FIGURE 1. Interphalangeal dermatitis because of acute cutaneous lupus erythematosus. Note the sparing of the dorsal metacarpophalangeal and proximal interphalangeal joint regions (ACR Image Bank).

Differential Diagnosis

Localized form may be mistaken for sunburn, rosacea, seborrheic dermatitis, acne, and eczematous dermatitis, whereas the generalized form may present similar to a drug-induced or infectious rash as well as dermatomyositis. In dermatomyositis, however, the sparing of the knuckles is not noted.

SUBACUTE CLE

Skin manifestations of SCLE (Figure 2) are characterized by reddish scaly annular, papular, or plaque lesions that are often seen in sun-exposed areas, including the upper chest, upper back, and extensor aspects of the forearms and arms. The central face and scalp are typically spared (Walling & Sontheimer, 2009). Other less common presentations of SCLE include pityriasiform SCLE, toxic-epidermal-necrolysis-like SCLE, SCLE resembling vitiligo, generalized poikiloderma, and exfoliative erythroderma-like SCLE (Walling & Sontheimer, 2009). Approximately 50% of the patients with SCLE may progress to develop SLE, although systemic symptoms are typically mild with arthritis and myalgias (Cohen & Crosby, 1994; Sontheimer, 1985).

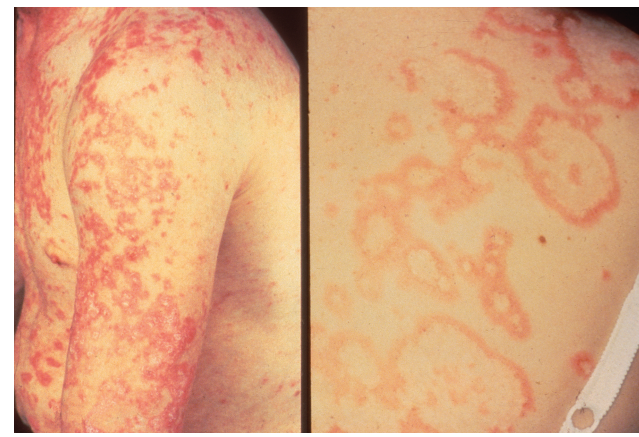

FIGURE 2. Subacute cutaneous lupus erythematosus affecting the back. (Left) Papulosquamous lesions with scaling papules and plaques and that resemble psoriasis. (Right) Annular polycyclic lesions with erythematous, slightly scaling border with central clearing (ACR Image Bank).

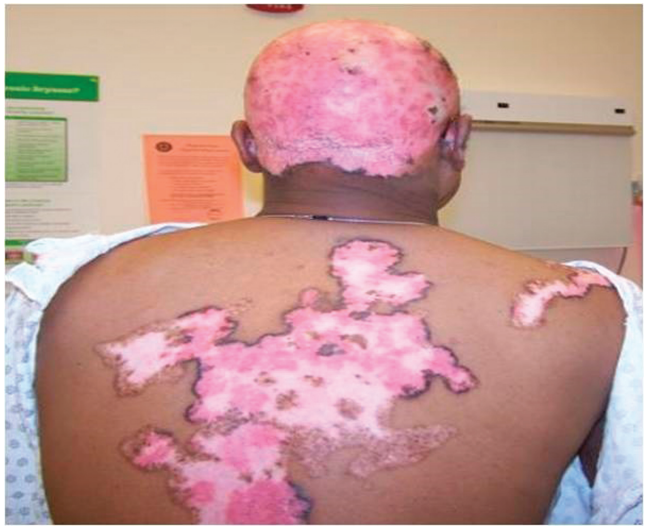

FIGURE 3. Discoid lupus erythematosus involving the scalp and back. Multiple discoid plaques with central scarring and peripheral postinflammatory hyperpigmentation (ACR Image Bank).

Drugs can induce and/or exacerbate SCLE and include antifungals like terbinafine, tumor necrosis factor inhibitors, proton pump inhibitors and nonsteroidal anti-inflammatory drugs, and antihypertensives (hydrochlorothiazide, calcium channel blockers, and angiotensin-converting enzyme inhibitors; Grönhagen et al., 2012). As in ACLE, lesions from SCLE heal without scarring. The anti-Ro (SS-A) antibody is noted in 70% of patients with SCLE and is also associated with an overlap between SCLE and Sjogren's syndrome (Rothfield et al., 2006). The papulosquamous variant of SCLE may resemble eczema, psoriasis, or even pityriasis.

CHRONIC CLE

Discoid lesions (Figure 3) are the most common type of CCLE and can be localized or generalized. Localized discoid lupus erythematosus (DLE) commonly involves the head and neck and particularly the scalp and ears. Generalized DLE occurs both above and below the neck and also involves the extensor forearms and hands. Occasionally, DLE occurs on mucosal surfaces, including lips as well as oral, nasal, and genital mucosa. Lesions are initially erythematous with adherent scaling, crusting, and dilated hair follicles. It is disfiguring at times, healing with permanent atrophic scarring, hypopigmentation or hyperpigmentation, and hair loss. Five to ten percent of patients with DLE develop SLE (Vera-Recabarren et al., 2010). This is more likely in patients with generalized DLE, DLE with arthralgia or arthritis, and positive ANA, elevated erythrocyte sedimentation rate, and blood count abnormalities (Callen, 1982; Insawang et al., 2010).

Hypertrophic or verrucous lupus erythematosus (LE) is a rare form of CCLE, occurring in 2% of cases and typically in combination with discoid lesions (Sontheimer, 1997). Lesions present as erythematous and indurated lesions with overlying scale or crust affecting the extensor upper and lower extremities, upper back, and face. These individuals rarely develop systemic disease, although lesions are often chronic and resistant to therapies. Lupus panniculitis or profundus presents as painful subcutaneous nodules on the arms, face, and buttocks, healing with atrophic scars (Fabbri et al., 2003). Typically, the atrophy is painless but physically deforming (Figure 4), although ulceration may rarely occur.

LE tumidus is characterized by pink to violaceous, raised, smooth skin lesions resembling hives or nodules. Scarring is absent, and the skin generally looks swollen in this region, typically affecting the face (Mascaro et al., 1997). Chilblain lupus is a rare manifestation of CCLE that resembles "frostbite" with painful cold-induced reddish-blue skin lesions in the toes, fingers, nose, or ears in the absence of cold agglutinins and cryoglobulins (Figure 5). Lesions may ulcerate or present with

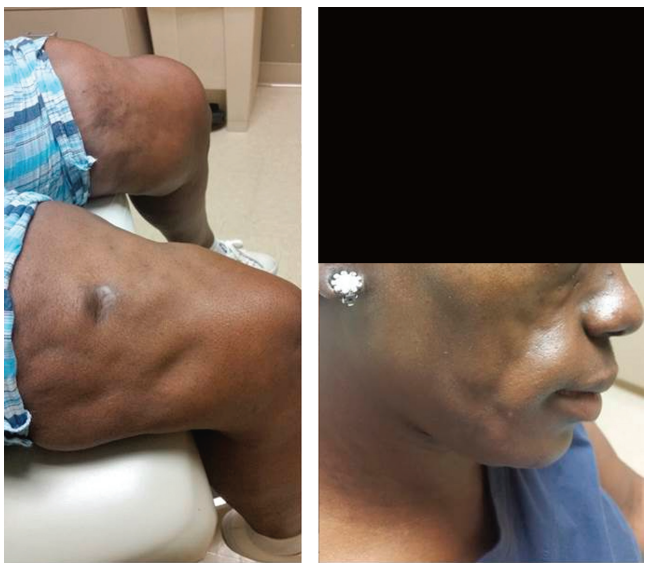

FIGURE 4. Lupus panniculitis affecting the legs and face.

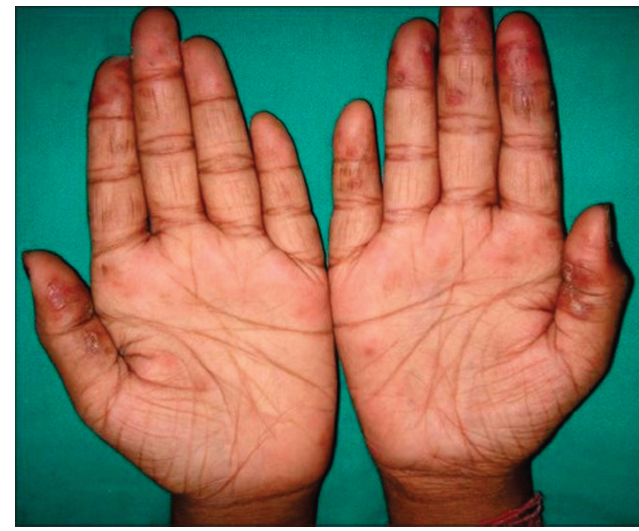

FIGURE 5. Chilblain lupus lesions over the ventral aspect of fingers and palms (reproduced with permission from Dr. Shikha Bansal).

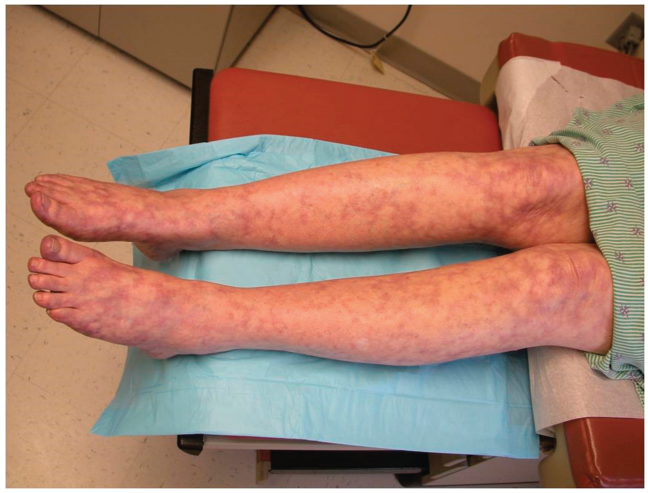

FIGURE 6. Livedo reticularis seen in both legs in a patient with positive lupus anticoagulant (ACR Image Bank).

hyperkeratosis and are often painful (Doutre et al., 1992). LE–lichen planus overlap presents as blue–red to violaceous painful and pruritic plaques or patches mostly on the extremities, particularly the palms and soles, but can also be present on the legs, face, and trunk. It is associated with SS-A antibodies (Schmitz et al., 2018).

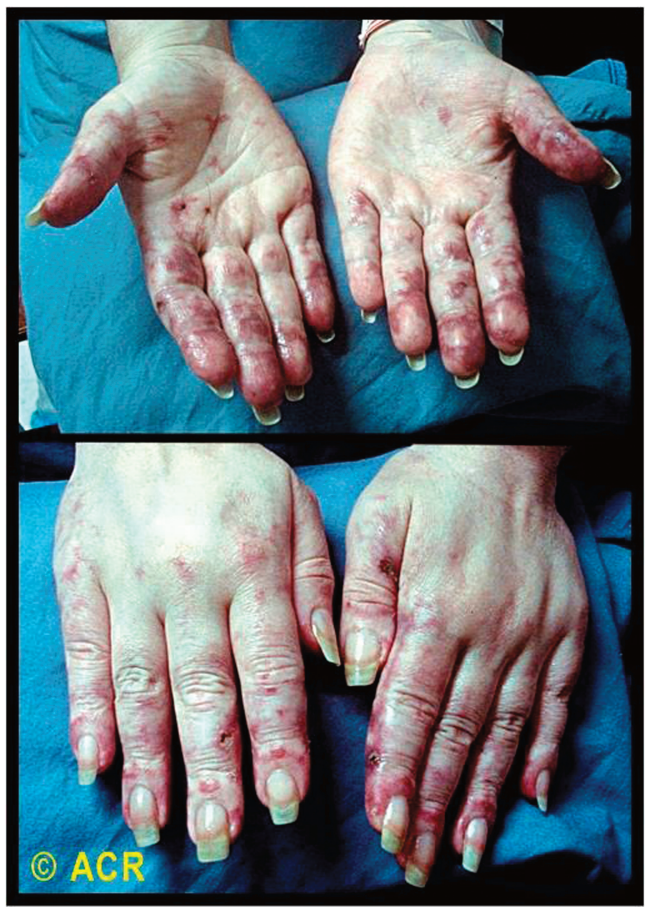

FIGURE 7. (Above) Vasculitis bilateral hands. (Below) After treatment with steroids (ACR Image Bank).

NONSPECIFIC SKIN LESIONS SEEN IN LE

Vascular abnormalities can occur in patients with SLE (Gilliam & Sontheimer, 1982). These include periungual erythema (dilated tortuous loops of capillaries along the base of the nail), livedo reticularis (reddish-cyanotic, reticular pattern on the skin of the arms, legs, and torso, particularly with cold exposure; Figure 6), Raynaud phenomenon (blanching of the nail beds, fingers, and toes with

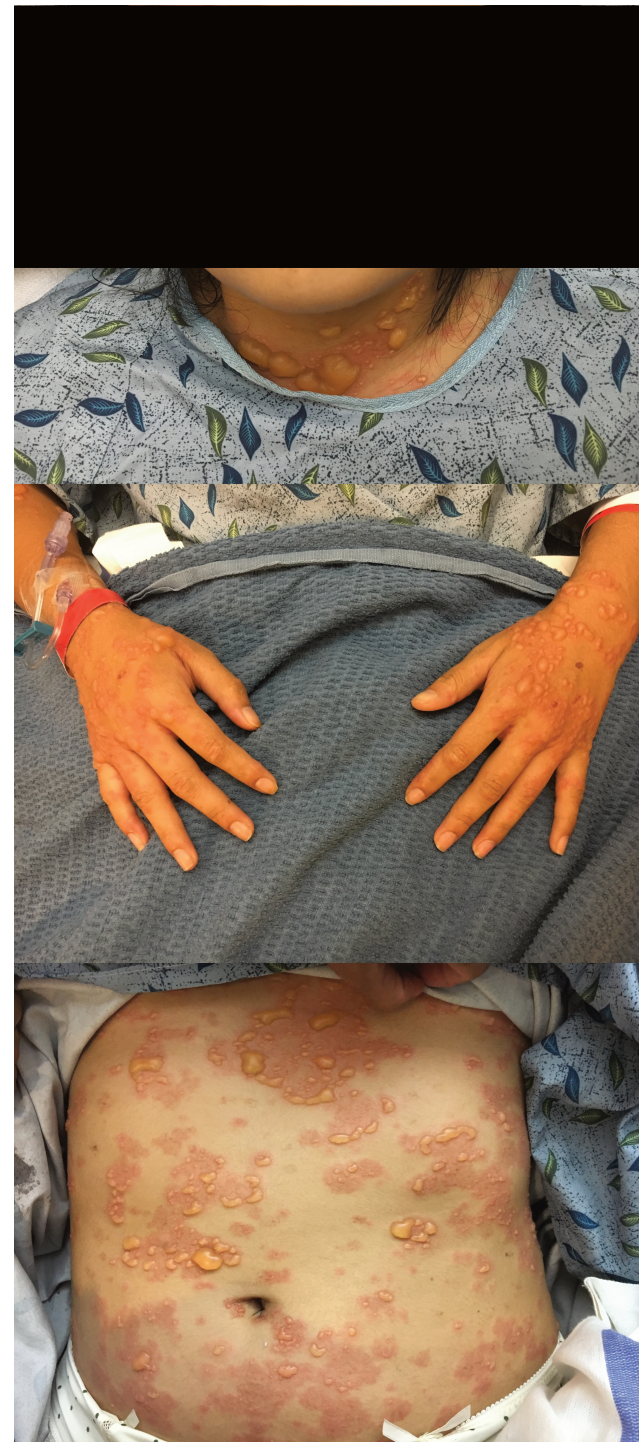

FIGURE 8. Bullous lupus erythematosus (ACR Image Bank).

accompanying pain), and vasculitis (inflammation of blood vessels; Figure 7). The most common type of vasculitis is urticarial vasculitis where urticarial lesions persist for more than 24 hours and frequently evolve into painful petechiae or purpura that may heal with hyperpigmentation. Other vasculitic lesions can include palpable purpura and punched out, painful, nonhealing ulcers.

"Lupus hair" is frequently seen during exacerbations of SLE and is characterized by thin, brittle hair usually occurring along the hairline. The hairs noted in this region look similar to lanugo. Patchy bald spots typical of alopecia are also seen in CLE as well as SLE. Painless oral and nasal lesions characterized by whitish plaques, erosions, and ulcerations may be seen in patients with CLE and SLE. Although not common, fluid-filled lesions (bullous lesions; Figure 8) can be seen anywhere in the body including the oral mucosa. Additional cutaneous lesions described in patients with LE include rheumatoid nodules, calcinosis cutis, erythema multiforme, acanthosis nigricans, thrombophlebitis, facial edema, and cheilitis (Rothfield et al., 2006).

DIAGNOSIS

Detailed patient history and clinical examination are required to determine the extent of skin and systemic involvement. Typical clinical findings are often enough to make a diagnosis. Skin biopsy can be helpful in cases when diagnosis is uncertain or when there is overlap with other connective tissue disorders. Antibodies toward extractable nuclear antigens can be positive in 60%–80% patients with CLE, generally of the Ro/SSA and La/SSB subtypes (Gilliam & Sontheimer, 1982). Additional antibody positivity includes ANA, dsDNA, and histone antibodies, the latter of which can be seen in drug-induced LE (Vera-Recabarren et al., 2010). Continuous monitoring for systemic disease is of utmost importance, particularly in patients with ACLE. In addition to ANA, dsDNA, Ro/SSA, and La/SSB, patients with systemic symptoms (pleural/pericardial symptoms, arthritis, and neuropsychiatric symptoms, to name a few) may present with positive antibodies to Smith, ribonucleoprotein, and low complement proteins, C3 and/or C4. In addition, some patients with blood count abnormalities may present with positive Coombs testing or evidence of hemolysis on further bloodwork.

TREATMENT

The treatment of CLE includes preventive measures and a combination of topical and systemic therapies based on type, extent of involvement, and disease activity.

Lifestyle Measures

The most important preventive measure includes educating patients on the role of ultraviolet A and B irradiation in causing flares of CLE, thereby encouraging routine sun avoidance and sunscreen usage. Patients should apply sunscreen with a sun protection factor of 50 and above at least 30 minutes before exposure (Kuhn et al., 2011). These measures, in addition to protective clothing (long sleeves, long pants, and a broad-brimmed hat), have shown a reduction in flares and improved outcomes in patients with LE (Winkelmann et al., 2013).

Topical Therapies

Topical Corticosteroids

The anti-inflammatory action of topical steroids is useful in treating CLE. Typically, potency of steroids varies with the region of involvement. Common side effects of these therapies include skin thinning, striae, and telangiectasia. In refractory cases of DLE, intralesional steroids may also be useful.

- Intralesional corticosteroid injections are used in some cases of CLE, particularly where an inflammatory component within a lesion may be addressed by local injection.
- Shampoos and foams have shown benefit in scalp disease (typically DLE and alopecia).

Calcineurin Inhibitors

Calcineurin inhibitors act on T cells and inhibit release of various inflammatory cytokines. These medications include tacrolimus and pimecrolimus. The anti-inflammatory benefit of these therapies lacks those side effects seen with corticosteroids, although they may cause temporary redness, itching, and irritation (Sárdy et al., 2009).

Systemic Therapies

Antimalarials

Oral antimalarial medications are commonly used anti-inflammatory systemic agents for cutaneous lupus. Hydroxychloroquine, chloroquine, and quinacrine are the most commonly used antimalarials. It takes at least 6–8 weeks to achieve peak response to these medications. Potential side effects include gastrointestinal discomfort, pigmentary changes, dry skin, and retinal toxicity (generally after long-term use). Because of the retinal toxicity risk, routine monitoring with ophthalmology is recommended (Ritschel et al., 1978).

Systemic steroids

Short-term steroids for 2–4 weeks are preferred in acute phases of CLE, with avoidance of long-term steroids because of various systemic side effects seen with prolonged therapy.

Retinoids

Vitamin A derivatives like acitretin and isotretinoin are useful especially in hyperkeratotic lesions and often used as second-line agents for CLE (Ruzicka et al., 1992).

Second- or third-line agents

For severe and aggressive diseases, immunomodulatory treatment may be indicated and mycophenolate mofetil, methotrexate, azathioprine, leflunomide, and thalidomide are used. Dapsone may also be considered, particularly in

bullous disease (Sebaratnam & Murrell, 2011). Biologic agents such as belimumab and rituximab are additional potential treatments for CLE (Vashisht et al., 2017; Vital et al., 2015).

CONCLUSION

Cutaneous manifestations of lupus may occur independently or in association with SLE. Early recognition of CLE allows for timely intervention and potential prevention of disabling disease. Obtaining a skin biopsy may be warranted for diagnostic confirmation, although much of CLE is diagnosed by clinical evaluation alone. Laboratory testing may also add value to identify patients progressing to systemic disease. Finally, a collaborative approach to managing CLE between the primary care provider, dermatologist, and rheumatologist fosters the provision of high-quality healthcare with improved outcomes.

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