

Subacute Cutaneous Lupus Erythematosus

Subacute cutaneous lupus erythematosus (SCLE) is a distinct form of cutaneous lupus erythematosus characterized by erythematous, scaly papular eruptions with a photosensitive distribution. While it is often associated with systemic lupus erythematosus (SLE), it may also occur in individuals with other autoimmune disorders such as Sjögren's syndrome. This condition can be triggered by genetic factors, ultraviolet (UV) light exposure, and certain medications, making it a multifactorial disease. SCLE lesions are typically non-scarring and non-atrophic but may leave some residual pigmentation. Management primarily involves photoprotection and anti-inflammatory treatments, along with careful monitoring of disease activity.

Clinical Features

SCLE typically presents as small, erythematous, scaly papules in sun-exposed areas, including the shoulders, forearms, neck, and upper torso. These lesions may evolve into psoriasiform or annular forms, often with a characteristic pattern of clearing in the center and a raised border. The disease is non-scarring, and lesions usually resolve without atrophy, though they may leave behind dyspigmentation. Approximately 50% of patients with SCLE also experience joint involvement, particularly arthralgias that affect small joints like those of the wrists and hands, and tend to be symmetrical in nature.

SCLE occurs more frequently in individuals with genetic predispositions. These include the presence of anti-Ro (SS-A) autoantibodies, positive antinuclear antibodies (ANA), and certain human leukocyte antigens (HLAs). Genetic susceptibility is a critical factor in the development of SCLE, and it is more common in females, especially during their reproductive years.

Pathogenesis and Triggers

The pathogenesis of SCLE is multifactorial and involves both genetic and environmental factors. Exposure to UV light is a well-established trigger, leading to an exacerbation of disease in genetically susceptible individuals. Furthermore, certain medications have been implicated in drug-induced SCLE, including hydrochlorothiazide, calcium channel blockers, angiotensin-converting enzyme (ACE) inhibitors, terbinafine, procainamide, antihistamines, and tumor necrosis factor (TNF) antagonists. A comprehensive drug history is essential in the diagnostic workup, and discontinuation of the offending medication is often necessary to alleviate symptoms.

Additional immunologic factors play a role in the pathogenesis of SCLE. Elevated erythrocyte sedimentation rate and positive rheumatoid factor may be present, alongside decreased complement levels. Hematologic abnormalities such as anemia, leukopenia, and thrombocytopenia are also common findings. Routine urinalysis should be conducted at baseline and periodically during the patient's clinical course to monitor for potential renal involvement.

Diagnosis

The diagnosis of SCLE is largely clinical but can be confirmed through a combination of laboratory testing and histopathology. A biopsy from sun-exposed or unaffected skin can be useful, with the lupus band test (LBT) being a specific diagnostic tool. Histological examination of the affected skin reveals characteristic vacuolar alteration of the basal cell layer, accompanied by a lymphocytic infiltrate around vessels and appendiceal structures. Additionally, there may be inflammation in the subepidermal layer. The LBT helps confirm the presence of immune deposits at the dermoepidermal junction in affected skin, which is indicative of lupus erythematosus.

Treatment Strategies

The mainstay of treatment for SCLE involves strict sun protection to prevent exacerbation of symptoms. Patients should be advised to use broad-spectrum sunscreen with a high SPF and wear protective clothing, including hats and long sleeves, when exposed to sunlight. In addition to sun protection, several therapeutic options are available to manage SCLE lesions and associated systemic symptoms.

- **Topical and Intralesional Steroids:** Topical corticosteroids, especially low to mid-potency formulations, are commonly used to treat localized lesions. In cases of more extensive or resistant lesions, intralesional corticosteroids can provide more targeted relief. However, systemic corticosteroids should be avoided unless there is evidence of concurrent systemic involvement or the need for short-term treatment.
- **Hydroxychloroquine:** Hydroxychloroquine, an antimalarial drug, is a cornerstone of therapy for SCLE and systemic lupus erythematosus (SLE). It has been shown to reduce inflammation and prevent flare-ups. However, its efficacy may be reduced in smokers, who experience less favorable outcomes due to impaired drug metabolism.
- **Immunosuppressive Agents:** In cases of refractory SCLE or those with systemic involvement, immunosuppressive agents such as dapsone, thalidomide, and methotrexate may be considered. These drugs can help modulate the immune response, but they should be used cautiously due to potential side effects.
- **Systemic Retinoids and Interferon:** For severe or persistent cases, systemic retinoids such as acitretin may be considered, especially in patients with extensive disease. Interferon therapy has also been explored as an option in treating SCLE, with some studies showing promising results in terms of disease control.

- **Vitamin D and Calcium Supplementation:** Some patients with SCLE, particularly those with reduced sun exposure due to photosensitivity, may experience vitamin D deficiency. Supplementation with vitamin D and calcium is recommended to prevent bone loss and improve overall health, particularly in patients with longstanding disease.

Conclusion

SCLE is a distinct clinical entity that often occurs in the context of systemic lupus erythematosus or other autoimmune disorders, with genetic and environmental factors playing critical roles in its development. Early recognition and treatment are essential to managing the disease and preventing complications. Photoprotection remains the cornerstone of management, with topical therapies, antimalarials, and immunosuppressive agents used to control disease activity. A personalized approach, including the consideration of drug-induced SCLE and potential vitamin D deficiency, is crucial in optimizing patient outcomes.

References

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