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Lupus: Open Access

Short Communication

Percutaneous Manifestations with Lupus Erythematosus

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ABOUT THE STUDY

The word "lupus," which comes from the Latin for "wolf," is used to refer to a variety of dermatological disorders, some of which are connected to the underlying systemic lupus erythematosus and others which are separate disease processes. Numerous visible skin signs are part of cutaneous lupus erythematosus, which in certain instances can lead to systemic lupus erythematosus. Acute cutaneous lupus erythematosus, subacute cutaneous lupus erythematosus, and chronic cutaneous lupus erythematosus are the three primary subtypes of cutaneous lupus. Differentiation of cutaneous lupus subgroups is made possible by physical examination, laboratory research, and histology. This distinction is crucial because the subtype of cutaneous lupus affects prognosis, surveillance, and treatment. This review describes the various cutaneous lupus erythematosus presentations and offers an update on both topical and systemic therapy options for affected individuals [1].

Systemic Lupus Erythematosus (SLE), a multiorgan, chronic autoimmune illness that can cause disability and death, frequently has substantial skin involvement. With a female-to-male incidence ratio of 7 to 15:1 in adults and 3 to 4:1 in children, gender is the biggest risk factor for SLE. Patients with isolated cutaneous lesions exhibit less pronounced gender preponderance, but the female-to-male ratio is still 3 to 1. It should be highlighted that SLE is among the top 20 terminators of females between the ages of 5 and 64. Patients of African origin tend to acquire the disease earlier and have a higher incidence of SLE, which affects black women four times more often than white women [2].

Although cutaneous signs of SLE are prevalent, cutaneous lupus can develop in the absence of systemic lupus erythematous. The three most prevalent cutaneous lupus erythematosus presentations are Acute Cutaneous Lupus Erythematosus (ACLE), Subacute Cutaneous Lupus Erythematosus (SCLE), Discoid Lupus Erythematosus (DLE), and Cutaneous Lupus Erythematosus (CLE). The toxic epidermal necrolysis variant of lupus, chilblain lupus, hypertrophic or verrucous discoid lupus, mucosal discoid lupus, and lichenoid cutaneous lupus-lichen planus overlap syndrome are a few less frequent cutaneous signs of lupus [3]. Lupus vulgaris, lupus miliaris disseminates faciei, and lupus pernio are three additional skin disorders that utilize the term "lupus" but are independent from lupus erythematosus. Each of these skin conditions has a connection to sarcoidosis, granulomatous rosacea, or tuberculosis. Within three to five years of the initial CLE diagnosis, there is a 5% chance that the condition would advance to SLE. SLE has already been diagnosed in one-third of CLE patients or will be in the near future. Comparing those with SCLE, DLE, lupus tumidus, lupus panniculitis, or chilblain lupus to those with ACLE, bullous lupus, and non-specific cutaneous lesions of lupus (such as vasculopathic lesions), it is clear that those with these conditions have a higher probability of acquiring systemic lupus [4].

The majority of CLE variations are characterised molecularly by a lichenoid tissue reaction brought on by activation of keratinocytes, endothelial cells, and dendritic cells. at the day a complicated interaction of genetic and environmental factors causes CLE. A cascade of inflammatory reactions involving skin cells and enlisted inflammatory cells can be brought on by ultraviolet radiation, specific drugs, smoking, viral infection, and other factors. The considerable diversity in clinical presentation of cutaneous LE is largely due to genetic variance depending on parentage and gene mutations. The three main CLE types are not mutually exclusive, and several cutaneous lesion types can coexist in a single individual. There are both localized and generalized types of Acute Cutaneous Lupus Erythematosus (ACLE), which is usually linked to systemic lupus erythematosus. Antinuclear antibodies are present in 95% of ACLE patients Anti Nuclear Antibody (ANA) [5]. Though there may be exceptions, rash flare-ups generally coincide with systemic disease activity in both subtypes of ACLE. ACLE lesions normally heal without leaving scars, but post inflammatory dyschromia can happen, particularly in people with darker skin. The cheeks and nasal bridge are typically covered with a malar or "butterfly" rash that is a common description of the localized form of ACLE. The nasolabial folds are spared, although the forehead and the front of the neck may be included. It is typical to have a confluent, reddish-purple discoloration along with minor edema and/or papules. Typically, the rash lasts a few days to a few weeks and is brought on by sun exposure. In 40% of SLE patients at the time of diagnosis, the rash is frequently confused for seborrheic dermatitis,

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which affects the nasolabial folds, or rosacea, which manifests with papules and pustules.

The presence of telangiectasias, erosions, depigmentation, and poikiloderma are all indicators of ACLE. Dermatomyositis can also cause a malar rash, which can be challenging to distinguish from the traditional malar rash of ACLE. However, the nasolabial folds are typically not spared by the malar rash of dermatomyositis [6].

CONCLUSION

The term "cutaneous lupus erythematosus" refers to a broad range of rashes with various clinical presentations, histopathologies, and therapeutic choices. For rheumatologists and dermatologists alike, the use of the name "lupus" to describe a few additional dermatological disorders that are actually unrelated to lupus erythematosus sometimes leads to confusion. The care of all types of CLE centers on sun protection and quitting smoking. For cutaneous lupus, topical steroids are frequently used as a first step; however, when systemic therapy is necessary, antimalarial medications should be used first. If these methods are unsuccessful, a range of additional immunosuppressive drugs may allow steroid sparing while keeping cutaneous lupus under control.

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