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REVIEW ARTICLE

Interaction between hidradenitis suppurativa and systemic lupus erythematosus: inflammatory and autoimmune mechanisms

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ABSTRACT

Introduction: systemic lupus erythematosus (SLE) and hidradenitis suppurativa (HS) are chronic inflammatory conditions that could have an immunological interrelationship. HS presents with painful abscesses and nodules in intertriginous areas, whereas SLE is a multisystem disease characterized by skin eruptions, joint involvement, and physical alterations.
Objective: to describe the immunological mechanisms, predisposing factors, the clinical manifestations of HS and SLE, and the possible intersections that could guide future research and therapies.
Methods: aAn integrative bibliographic analysis of publications from 2015 to 2024 was performed, selected from scientific databases such as PubMed, BVS, SciELO, and Elsevier. Studies addressing pathophysiology, immunological interaction, and potential relationships between HS and SLE were incorporated.
Results: findings indicate that proinflammatory cytokines such as TNF-α, IL-17, and IL-23 play a crucial role in both conditions, promoting chronic inflammation and aberrant immune activation. These similarities in the inflammatory cascade could justify the co-occurrence of both diseases in certain patients and propose new avenues of study regarding their pathophysiological link.
Conclusion: contemporary research indicates that hidradenitis suppurativa and systemic lupus erythematosus share inflammatory mechanisms that could be involved in their etiology. This underscores the importance of combined diagnostic and therapeutic strategies to optimize the management of patients with both conditions. Additional investigations are advised to corroborate these links and develop more effective therapeutic approaches focused on common immunological pathways.

Keywords: Hidradenitis Suppurativa; Lupus Erythematosus, Systemic; Autoimmunity.

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INTRODUCTION

Hidradenitis suppurativa (HS) and systemic lupus erythematosus (SLE) are two chronic inflammatory diseases that, despite their clinical and pathogenic differences, share underlying immunological mechanisms. HS is characterized by the emergence of painful inflammatory lesions in areas rich in apocrine glands, while SLE is a multisystem disease with manifestations ranging from skin eruptions to renal and neurological involvement. Both disorders significantly impact patients' quality of life, both physically and psychologically.

Despite advances in understanding these mechanisms, there are significant gaps in knowledge about predisposing factors and the interrelation between HS and SLE. Genetic and environmental factors play crucial roles in susceptibility and progression of both diseases, although their specific impact varies. For example, mutations in γ-secretase subunit genes have been identified in HS, while in SLE, susceptibility loci are mainly associated with genes of the HLA system and complement.

The present work aims to describe the immunological mechanisms, predisposing factors, the clinical manifestations of HS and SLE, and the possible intersections that could guide future research and therapies.

This critical literature analysis seeks to contribute to a deeper understanding of the relationship between inflammation and autoimmunity in these disorders, highlighting the importance of an integrated approach to their study and management.

METHODS

This integrative bibliographic review was conducted through several recognized databases, including PubMed, BVS, SciELO, and Elsevier. The search covered the period from 2015 to 2024. A retrospective approach was adopted, selecting articles written in English and Spanish. These articles were exhaustively reviewed to extract and analyze data pertinent to the relationship between hidradenitis suppurativa and systemic lupus erythematosus, focusing on the underlying immunological mechanisms and predisposing factors, clinical manifestations, and treatment of both diseases.

DEVELOPMENT

Hidradenitis suppurativa (HS)

Hidradenitis suppurativa can be defined as an inflammatory disease, which is chronic, recurrent, and debilitating, typically appearing after puberty with deep, inflammatory, and painful lesions affecting areas of the body where apocrine glands are present, with the axillary, inguinal, and anogenital regions being the most frequent. Throughout history, this entity has been known as Vernouil disease; subsequently, its pathogenesis was attributed to occlusion of the pilosebaceous follicle, and it has been related to other diseases such as acne conglobata, dissecting cellulitis of the scalp, or pilonidal sinus. It forms part of the follicular occlusion tetrad. By 1989, a new term was introduced: inverted acne.[1]

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Being a chronic inflammatory disorder, it can impact interpersonal relationships, physical appearance, self-esteem, and body image; while it is true that it can play a significant role in the patient's psychological aspect, it can also cause physical limitations due to scarring, resulting in reduced mobility of the limbs.

Incidence and prevalence of hidradenitis suppurativa.
Hidradenitis suppurativa has an estimated prevalence of 1 % to 4 %; however, the number of diagnosed individuals is lower than the number of people who may suffer from the disease, thus most cases occur in the young population.[2] Most cases are observed in the second and third decades of life with an average age of onset around 20 years.[3] Other studies determine a prevalence of 0.08%. Regarding age distribution, the disease usually begins after puberty, typically at the start of the third decade and tends to remain active until the fourth decade of life; it has also been observed that women are affected more frequently than men, in a 3:1 ratio.[1] In terms of racial distribution, hidradenitis suppurativa is higher in Black individuals.

Clinical manifestations
The primary lesions are usually preceded by itching and general malaise, especially in areas rich in apocrine glands. These lesions may appear as subcutaneous erythematous nodules, described as firm and typically measuring approximately 0.5 to 1.5 cm in size, and can be clinically difficult to differentiate from inflamed sebaceous cysts. At this stage, there may be clues that can guide the diagnosis, such as triple comedones in the surrounding area. Primary nodules may remain inactive for days or months before progressing to abscesses that eventually penetrate the skin, draining the nasal sinuses with purulent or seropurulent discharge.[3] Inflammation is also frequently detected in internal organs, as in the case of metabolic syndrome, inflammatory bowel disease, etc. This entire constellation of manifestations profoundly influences patients' quality of life, thereby reducing life expectancy.[4]

Systemic lupus erythematosus (SLE)
Systemic lupus erythematosus (SLE) is a chronic multisystem inflammatory disease of unknown cause in which tissue damage is produced by deposits of antibodies and immune complexes. It shows a predilection for females; however, age plays an important role in reducing sex differences. In Black individuals, it is three times more frequent and more aggressive.[5]

Incidence and prevalence of lupus
For the general population, the worldwide incidence of SLE and new diagnoses is estimated at 5.14 per 100,000 person-years and 0.4 million people annually. In women, the values are 8.82 per 100,000 person-years and 0.34 million people annually; in men, these values correspond to 1.53 per 100,000 and 0.06 million people annually. In Ecuador, the incidence is 6.5 with a total of 1,146.64 cases; of this value, in men it is 1.89 with a number of patients 166.76. On the other hand, in women in Ecuador it is 10.97 corresponding to 967.27 patients.[6]

The global prevalence of lupus is SLE and the affected population was estimated at 43.7 per 100,000 people and 3.04 million people. In women, the estimated values correspond to 78.73 per 100,000 and 3.04 million people, while in men the estimates were 9.26 per 100,000 people and 0.36 million people. In Ecuador, the estimated prevalence is 68.1, corresponding to a total of 12,011.7 cases. In men the estimated prevalence is 14.34, a total of 1,266.45 cases. On the other hand, the estimated prevalence of women is 121.9 with a total of 10,750.76 cases.

Clinical manifestations
SLE can affect the skin, joints, kidneys, lungs, the nervous system, serosa, the digestive tract, and the circulatory system. Patients with lupus can develop a multitude of immunological alterations, among which antinuclear antibodies formation stands out.[5]

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Pathogenesis of HS and SLE

Common immunological mechanisms

Role of proinflammatory cytokines

In HS, predisposing factors will induce the predisposition factors for peripheral follicular immune activation, leading to blockage, sebaceous stagnation, and dilation of the pilosebaceous unit. With a blockage, bacteria can grow inside them; the bacteria that grow within release molecules that are associated with tissue damage, stimulating local immune cells, especially macrophages, producing inflammatory cytokines such as TNF-α and IL-1β. In the case of TNF-α, it induces inflammation by activating various immune system cells and stimulates the production of other inflammatory cytokines; it also increases the expression of adhesion molecules on endothelial cells, facilitating infiltration of immune cells to the site of inflammation. Its impact on HS is that it promotes chronic inflammation and contributes to abscess formation.

Th1 and Th17 cells and their mediators are abundant in HS skin; by contrast, Th22 cells and IL-22 are not abundant, leading to insufficient production of epidermal antimicrobial proteins, inadequate to limit bacterial growth.[4] IL-17 is produced by Th-17 cells; in HS, it contributes to chronic inflammation and tissue damage, with high production associated with neutrophil infiltration. IL-23, produced by dendritic cells and macrophages, helps to potentiate the inflammatory pathway of Th-17 cells.

In the case of lupus, there is dysregulation of both innate and adaptive immunity. In the innate arm, type I interferons, especially IFN-α, are involved and are associated with SLE; they activate natural killer cells, altering immune tolerance. By generating inadequate apoptosis, immune complexes may form, thereby creating more IFN-α, generating a vicious circle. In the case of Th17, IL-17 is produced contributing to inflammation and autoantibody formation. IL-17 promotes inflammation by inducing the production of other inflammatory cytokines; TNF-α promotes activation of T cells, B cells, and macrophages, involved in autoantibody production, thus leading to loss of immune tolerance.[7]

Predisposing factors

Genetic factors in SLE

There is not enough evidence to determine whether there are specific genetic factors that increase the risk of lupus. Early genetic-type studies, based on observations of SLE, have implicated HLA.[8] There appears to be an association with HLA-B8, HLA-DR3, and HLA-DR2 ,[5] and some genes of the complement system C1, C2, and C4. These are related to nucleic acids due to mutations in some pro-apoptotic genes, an example of monogenic lupus. Studies have identified at least 70 susceptibility loci for lupus. In addition, sexual hormones and possibly play roles. [8]

Genetic factors in HS

In hidradenitis suppurativa, a limited number of patients with predominantly severe disease and associated severe acne present mutations in genes encoding the γ-secretase (G-secretase) subunit, most of them in the gene for the presenilin subunit. In addition to γ-secretase mutations, variations in other genes have been identified in HS patients. One of these genes (MEFV) encodes pyrin, a pattern recognition receptor (PRR) component of the inflammasome. Activation of the inflammasome leads to production of IL-1β, a cytokine with an important role in HS pathogenesis.[4] The most frequently observed inheritance pattern is autosomal dominant. The implicated genes are located at the locus 1p21.1–1q25.3; inactivating mutations in the presenilin genes (PSEN1), and presenilin enhancer (PSENEN) have also been described.

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Environmental factors

Environmental factors in SLE

Among the factors frequently associated with SLE are smoking, where it has been shown that current smokers with more than 10 pack-years have a higher risk of anti-dsDNA–positive SLE in the range of 1.60–1.86, whereas former smokers do not. These findings resemble the known association between smoking and anti-cyclic citrullinated peptide antibodies in rheumatoid arthritis. Endometriosis confirmed by laparoscopy has been associated with an SLE diagnosis. Several studies have also demonstrated an inverse association between alcohol consumption and the risk of SLE in women (about 5 g or half a drink per day). Likewise, although with unresolved questions, an association between SLE and silica has been demonstrated.

Some factors that are weakly related to SLE are obesity, diet, infections, pesticides, and pollution. While no study has established a strong link between obesity and a marked risk of developing SLE, obesity has recently been related to autoimmune diseases due to increased adipokines expressed in adipose tissue and the increased resident macrophages. The apoptosis-inhibiting protein secreted by macrophages (AIM) is attributed the function of promoting cell survival against pro-apoptotic stimuli. AIM induces lipolysis, which favors the release of fatty acids; this process is associated with an increase in macrophage infiltration into adipose tissue. AIM forms immune complexes and autoreactive autoantigens of the IgM and IgG type.[10]

Environmental factors in HS

There are two important lifestyle factors that, although not present in all patients, have an accepted role in the development of the disease: obesity and smoking.[4] It has been observed that the prevalence of obesity in patients with HS is up to 18,1 %. Moreover, the disease improves after weight loss; therefore, the notion that obesity plays a role in development may be supported by the prevalence of associated diseases.[11] It is considered more of an exacerbating factor rather than a trigger. It also influences mechanical irritation, occlusion, and maceration.[1]

Central obesity has been found in approximately 60 % of patients. It is one of the factors that define metabolic syndrome. Obesity preceded the HS diagnosis by an average of five years. It is also noteworthy that there was a 4,5-fold higher risk of recurrence of skin alterations preceding surgery. It is proposed that obesity favors skin alterations in HS in two ways: firstly, by enlarging the body's skin folds and, consequently, increasing mechanical stress; secondly, by inducing a lower level of systemic inflammation and metabolic changes in the individuals, producing pro-inflammatory cytokines and inducing a dysregulated adipokine pattern that results in a negative effect on the skin.

Clinical manifestations and cutaneous characteristics

Skin lesions in HS

As noted previously, hidradenitis suppurativa (HS) occurs predominantly in intertriginous areas. It can affect the inguinal region, the inner thighs, the perianal and perineal areas, the breast regions, buttocks, pubic region, scrotum, vulva, trunk, and, occasionally, the scalp and reticular areas. Primary locations in women are the groin, chest, and axilla; in men, the primary localization areas are the groin or thigh, axilla, perineal or perianal regions, and buttocks. Other non-intertriginous sites commonly affected in both sexes include areas of friction and rubbing: beltline areas due to belt friction, abdominal folds, shoulder and chest areas from bra straps or bands.

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Most often, the first visible lesion is a solitary inflammatory, painful, deeply rooted nodule (0.5 to 2 cm) that can last days to months. After a period, the nodule usually progresses to form an abscess that may spontaneously rupture, producing purulent or sanguineous drainage. Pain improves after drainage. Sometimes multiple recurrent nodules develop after a delineated area, leading to interconnected fistulas; open comedones may also form. The appearance of scarred areas ranges from acneiform scars due to resolution of small nodules to dense fibrotic bands or indurated, thick, and marked plaques affecting the entire involved area. Keloids or atrophic scars can also occur, and in the buttocks, it sometimes presents as multiple scars.[12] See Figure 1

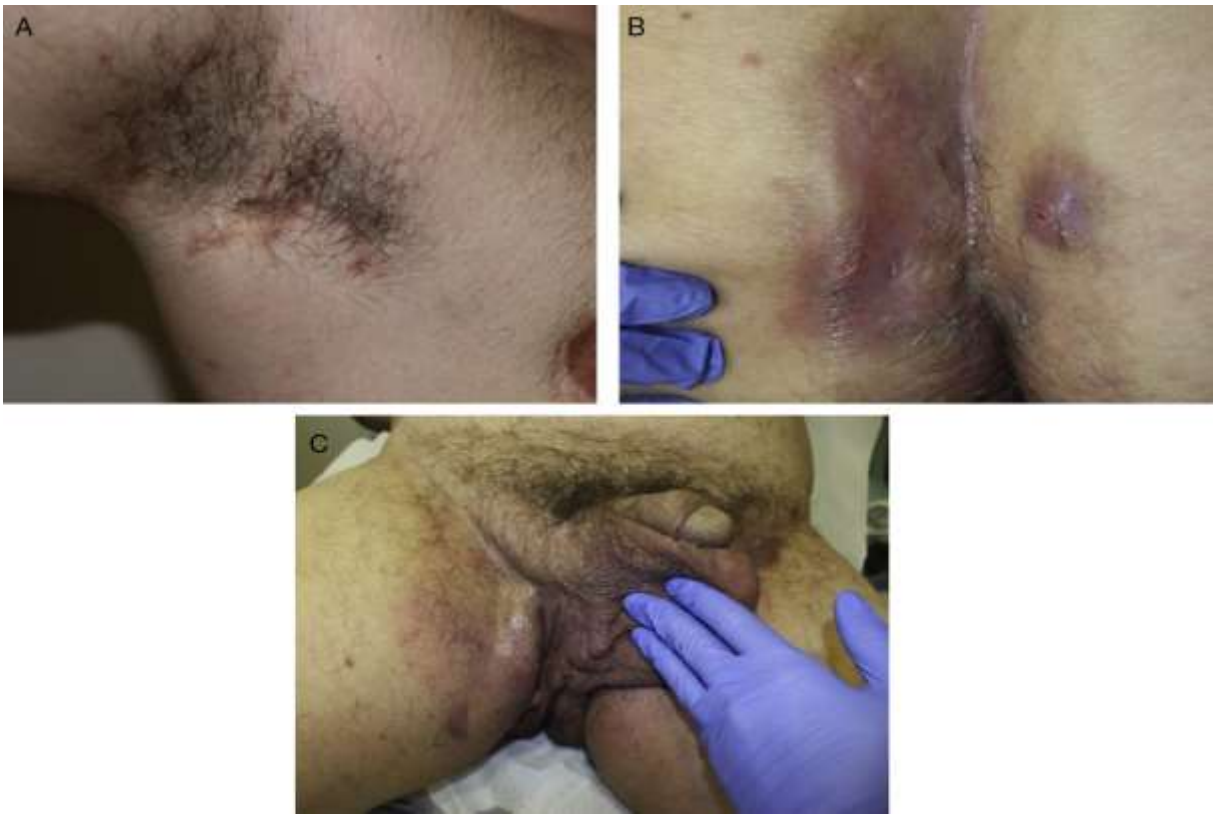

Fig. 1 Illustration 1: 36-year-old male with HS grade III. A. Linear axillary infiltrated and painful lesions. B. Presence of inflammatory nodules and suppurative abscesses on the buttocks. C. Abscesses coalescing in the right groin.[1]

Skin lesions in SLE

Cutaneous lesions appear in about 80 % of patients with SLE. They are part of the disease classification criteria and can be either specific or non-specific. In acute cutaneous lupus erythematosus, edema and erythema localize to the facial area, sparing the nasolabial folds; it is usually acutely painful or pruritic, precipitated by sun exposure, and distributes across photoexposed regions. It leaves no scarring and generally indicates disease activity. In subacute cutaneous lupus erythematosus, it occurs in approximately 10 % of patients with SLE. Lesions may appear in photoexposed as well as photoprotected areas. They can present as an annular form with rounded lesions of an active border and a clear center, with peripheral growth and central clearing, or as a psoriasiform or papulosquamous form with uniform growth and no central clearing; when it remits, it may leave a hypopigmented area. These patients frequently also have mucosal lesions with leukoplakic plaques or ulcers, transient diffuse alopecia, periungual telangiectasias, Raynaud phenomenon, and livedo reticularis.

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Finally, in chronic cutaneous lupus erythematosus, it is subdivided into discoid lupus lesions, hypertrophic lupus, and lupus panniculitis, with discoid lupus being the most frequent form. It consists of papules or erythematous and scaly plaques or hyperkeratosis, of variable size, well defined, with a tendency toward chronicity and peripheral growth, leaving atrophic scars and pigmentation changes.[13]

Complications of HS

Prolonged, untreated disease can lead to physical and psychological complications. HS lesions can become secondarily infected. In these rare cases, erysipelas and sepsis following soft tissue infection have been described. Movement impairment due to extensive fibrosis and scarring, especially in the axillary region, can occur. Lymphedema and squamous cell carcinoma can develop in areas of chronic inflammation. Other complications from prolonged systemic inflammation include arthritis, anemia, hypoalbuminemia, and AA amyloidosis leading to renal failure. [14] Among other complications, hypoproteinemia, sacral osteomyelitis, and malaise have been reported.[12] Psychologically, depression and suicide are noted complications, with issues such as embarrassment due to symptoms and pain contributing to a sense of helplessness regarding one's own body.[15]

Complications of SLE

SLE, being a complex and multifactorial disease, can lead to grave complications, including muscular complications. Pain is one of the most frequent features and is localized to the shoulder–glenohumeral girdle, with symptoms such as myalgia and muscle weakness often appearing. There are also osteoarticular complications, such as arthritis in SLE, which resembles rheumatoid arthritis and affects symmetrically the small joints of the hands; however, in SLE, the arthritis is non-erosive, a special form being Jaccoud arthropathy, which is erosive when termed rhupus. Osteoporosis, pericarditis, endocarditis, pleural effusion, and renal involvement with lupus nephritis may occur, among other clinical manifestations.[16]

Therapeutic approach

Treatment used in HS

In stages I and II, systemic antibiotics such as clindamycin, rifampin, azithromycin, moxifloxacin, ertapenem, alone or with non-steroidal anti-inflammatory drugs (NSAIDs) can be used. Cyclosporine and oral retinoids, intralesional glucocorticoids, and surgical treatment are also employed.[17]

Treatment used in SLE

In the case of SLE, the therapeutic approach depends on whether SLE affects any organ in a way that justifies the use of potent but aggressive therapies; also, whether the disease plays a reversible role and how to balance disease treatment with prevention of lupus- and drug-related complications. Mild therapy consists of NSAIDs and antimalarials. Methotrexate and leflunomide may be used for articular manifestations. For severe disease, high-dose steroids are required, given intravenously or orally, accompanied by immunosuppressive or cytotoxic agents such as intravenous or oral cyclophosphamide. Exposure to ultraviolet light should be avoided in patients with photosensitivity.[5]

Comparison between hidradenitis suppurativa (HS) and systemic lupus erythematosus (SLE).The comparison reveals both significant differences and shared immunological mechanisms. Both diseases are chronic and inflammatory in nature, but their clinical manifestations, pathogenesis, and predisposition factors vary considerably.

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Incidence and prevalence
HS incidence and prevalence vary significantly across studies, with prevalence estimates ranging from 1 % to 4, and higher prevalence observed in young women and people of African descent. Worldwide, SLE incidence is approximately 5.14 per 100,000 person-years, with a global prevalence estimated at 43.7 per 100,000 persons. In Ecuador, data show notably higher incidence and prevalence in women compared with men. The discrepancy in HS prevalence data may be due to underdiagnosis, while the higher prevalence of SLE in women may be related to hormonal and genetic factors.

Pathogenesis
Both diseases share involvement of proinflammatory cytokines such as TNF-α and IL-17 in their pathogenesis. In HS, perifollicular inflammation induced by obstruction of the pilosebaceous follicle leads to bacterial proliferation and release of inflammatory cytokines, perpetuating a cycle of chronic inflammation. In contrast, in SLE, dysregulation of innate and adaptive immunity, including type I interferon production and T- and B-cell activation, results in autoantibody and immune complex formation, contributing to multisystem inflammation.

Genetic and Environmental Factors
Genetic factors play a crucial role in both diseases. In SLE, at least 70 susceptibility loci have been identified, including HLA genes and components of the complement system. In HS, mutations have been identified in genes encoding γ-secretase subunits and in the MEFV gene, which encodes pyrin. Environmental factors also influence both diseases significantly. Smoking and obesity are strongly associated with HS, while in SLE, smoking, endometriosis, and silica exposure have been identified as risk factors.

Clinical Manifestations
The clinical manifestations of HS and SLE are distinct. HS is characterized by painful inflammatory nodules in areas rich in apocrine glands, such as the axillae and groin, which can progress to abscesses and fistulas, substantially impacting patients' quality of life. In contrast, SLE can affect multiple systems, including the skin, joints, kidneys, and nervous system. Cutaneous manifestations of SLE, such as malar erythema and discoid lesions, are disease-specific and form part of the classification criteria.

Complications
HS complications include secondary infections, extensive fibrosis, and an increased risk of squamous cell carcinoma in areas of chronic inflammation. Additionally, HS patients may suffer from depression and anxiety due to the physical and psychological impact of the disease. Conversely, SLE complications are more systemic and include lupus nephritis, pericarditis, arthritis, and neurological complications, reflecting the multisystemic nature of the disease.

Treatment
The therapeutic approach for HS and SLE differs as well. HS is managed with antibiotics, retinoids, immunosuppressants, and, in severe cases, surgical interventions. In SLE, treatment depends on severity and the organs affected, including NSAIDs, antimalarials, immunosuppressants, and steroids. Sun exposure should be avoided in photosensitive SLE patients. A notable point is that in both conditions, immunosuppressants are indicated.

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CONCLUSION

The relationship between hidradenitis suppurativa and systemic lupus erythematosus: The integrative literature review provides evidence of a significant correlation between hidradenitis suppurativa (HS) and systemic lupus erythematosus (SLE). Shared immunological mechanisms, such as dysregulation of proinflammatory cytokines (TNF-α, IL-17, IL-23) and aberrant activation of T and B cells, are critical factors contributing to the manifestations of both diseases. Both conditions involve an inflammatory process that can lead to impaired quality of life for affected patients. Impact of proinflammatory cytokines on pathogenesis: The results underscore the central role of proinflammatory cytokines in the pathogenesis of HS and SLE. The overexpression of TNF-α, IL-17, and IL-23 is closely linked to chronic inflammation and the altered immune response observed in these diseases.Therapeutic potential of cytokine inhibitors: Cytokine inhibitors could be effective in the treatment of both diseases, given their central role in inflammation and autoimmunity.

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