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REVIEW ARTICLE

Systemic Lupus Erythematosus: Clinical, Diagnostic, and Therapeutic Perspectives

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Abstract

Background: Systemic Lupus Erythematosus is a chronic autoimmune disease with systemic involvement, predominantly affecting women of reproductive age. Its development is related to genetic, hormonal, and environmental factors that lead to immune dysregulation and loss of tolerance to self-antigens. Cutaneous manifestations are common, especially photosensitivity and acute cutaneous lesions, such as the malar rash. Due to its heterogeneous clinical presentation, diagnosis and management remain challenging.

Methods: This narrative review synthesizes current evidence from books and scientific articles retrieved from the Scientific Electronic Library Online, PubMed, and MEDLINE databases. The search was conducted using standardized terminology related to Systemic Lupus Erythematosus, focusing on pathophysiology, clinical manifestations, diagnostic criteria, and therapeutic approaches.

Results: The analysis highlights that Systemic Lupus Erythematosus presents a wide range of clinical manifestations, affecting the skin, joints, kidneys, and other organs. Diagnosis relies on the integration of clinical findings and laboratory biomarkers that reflect immunological and inflammatory activity. Current treatment strategies require a multidisciplinary approach aimed at controlling disease activity, preventing organ damage, and improving quality of life. Advances in research have contributed to better disease understanding, although limitations in early diagnosis and long-term management persist.

Conclusion: Systemic Lupus Erythematosus is a complex disease with significant physical, psychological, and social impact on affected individuals. Early recognition of symptoms and accurate diagnosis are essential to reduce morbidity and improve patient outcomes. Continuous research and comprehensive clinical care are fundamental to optimize disease management and enhance the quality of life of patients living with Systemic Lupus Erythematosus.

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Abbreviations

SLE: Systemic Lupus Erythematosus; ANA: Antinuclear Antibody; ACR: American College of Rheumatology; EULAR: European League against Rheumatism; ESR: Erythrocyte Sedimentation Rate; IFN: Interferon; BAFF: B-cell Activating Factor; BLyS: B Lymphocyte Stimulator; CAR-T: Chimeric Antigen Receptor T cells; JAK: Janus kinase; IL: Interleukin; TNF: Tumor Necrosis Factor.

Introduction

Systemic Lupus Erythematosus (SLE) occurs worldwide. Epidemiological data from the United States indicate that its prevalence ranges from 14.6 to 50.8 cases per 100,000 inhabitants. Although SLE can affect individuals of all races, studies conducted in the U.S. show that Black women have a prevalence three to four times higher than that of White women. SLE predominantly affects females, with a ratio of 10 to 12 women for every diagnosed man. This female predominance is observed at all ages, being more pronounced between 15 and 64 years and less frequent in children and the elderly [1].

The skin is constantly exposed to environmental factors; however, in patients with SLE, its defense mechanisms are impaired, leading to the accumulation of auto-proteins and nucleic acids due to susceptibility to UV radiation and DNA damage. These structural changes overload the cellular repair system, resulting in activation of the immune system, which recognizes auto-proteins and produces cytokines through Toll-like receptors, such as type I IFN, type III IFN, IL-1, and TNF. This activation facilitates the recruitment of T and B lymphocytes, resulting in the production of autoantibodies directed against Ro and La proteins and DNA, which form antigen–antibody complexes deposited in the basement membranes of cells. The process is intensified by phagocytosis by dendritic cells and monocytes, as well as by neutrophil activation [2].

The diagnostic criteria of the American College of Rheumatology (ACR) for SLE have been revised several times over the years. In 2019, a collaboration between the ACR and the European League Against Rheumatism (EULAR) led to the development of new classification criteria aimed at improving sensitivity and specificity in the diagnosis of SLE. These criteria, known as the 2019 ACR/EULAR criteria, were designed to facilitate disease identification through a structured approach based on scores assigned to clinical and laboratory signs and symptoms. To initiate diagnosis, the patient must have a positive Antinuclear Antibody (ANA) titer of at least 1:80. This requirement is considered mandatory and serves as an entry criterion for the evaluation of the remaining criteria. Subsequently, SLE signs and symptoms are classified into different clinical domains, including constitutional symptoms, hematologic abnormalities, neuropsychiatric manifestations, mucocutaneous conditions, renal involvement, among others. Each manifestation is assigned a specific score, and diagnosis is suggested when the total score reaches 10 points or more [3].

Pharmacological treatment for SLE includes the use of antimalarials (hydroxychloroquine and chloroquine), glucocorticoids (betamethasone, dexamethasone, methylprednisolone, and prednisone), immunosuppressants, monoclonal antibodies, and inhibitory proteins. These therapeutic options aim to improve disease management by enhancing disease control, reducing flare frequency, and preventing organ damage. For non-pharmacological treatment, a multidisciplinary approach is essential. It is important to encourage regular aerobic exercise and to protect patients from sunlight and exposure to fluorescent or halogen lamps. Counseling, emotional support, and guidance are also crucial, ensuring that patients receive comprehensive support throughout their treatment journey [4].

Individuals with SLE undergo various

changes in their daily lives. In addition to physical and psychological symptoms, they commonly experience reduced muscle strength, changes in productivity and work capacity, as well as limitations imposed by treatment and changes in body perception and self-esteem. In this context, many patients view the disease as a challenge that affects their future perspectives, highlighting the importance of prevention through early diagnosis, regular follow-up, and awareness of symptoms [5].

Unlike traditional narrative reviews, this manuscript bridges well-established clinical features of systemic lupus erythematosus with recent advances in immunopathogenesis and the development of targeted therapies, including biologic agents and cell-based strategies. In addition, it highlights psychosocial and preventive aspects that are often underrepresented in the literature, offering a broader and more clinically relevant perspective for healthcare professionals engaged in the multidisciplinary care of patients with SLE.

Methods

A literature review on SLE was conducted based on books and scientific articles addressing the pathology, as well as its clinical, diagnostic, and therapeutic aspects. The sources were identified in the databases SciELO (Scientific Electronic Library Online), PubMed (National Center for Biotechnology Information – NCBI, U.S. National Library of Medicine), and MEDLINE, using the following keywords: Systemic Lupus Erythematosus, SLE, autoimmunity, lupus symptoms, and lupus treatment.

The literature search was carried out between January 2014 and December 2024. Original research articles, narrative and systematic reviews, as well as clinical guidelines published in English, Portuguese, or Spanish were considered. Studies were eligible if they addressed at least one key aspect of SLE, including epidemiology, pathophysiology, clinical manifestations, diagnostic criteria, therapeutic approaches, or preventive strategies.

Case reports, conference abstracts, editorials, and publications not directly related to SLE were excluded. Final study selection was guided by relevance to the review objectives, methodological quality, and publication recency, resulting in a total of 36 references included in the narrative synthesis.

Systemic lupus erythematosus – an epidemiological analysis

The etiology of SLE involves hormonal, genetic, and environmental factors that promote dysregulation of the immune system and loss of tolerance to self-antigens. Due to the interaction of these factors, autoantibodies are produced and, consequently, systemic inflammation occurs, ultimately leading to organ failure [6].

The incidence of individuals diagnosed with SLE has increased over the past four decades, largely due to advances in early disease detection. The ratio of this pathology between women and men can reach up to 13:1, while among children and the elderly this ratio is approximately 2:1 [7]. Regarding sex, there is a higher prevalence among women of childbearing and reproductive age, due to the influence of sex hormones that are capable of regulating the immune response. Another relevant difference between males (XY chromosome) and females (XX chromosome) is related to the presence of two functional X chromosomes, whether due to sex determination, duplication, or translocation, which confers a higher risk for the development of SLE compared to having only one X chromosome. Consequently, mortality follows this same prevalence pattern [6].

Socioeconomic level and differing climatic conditions generally and distinctly influence the pathology under discussion [8]. Ethnic-racial and cultural diversity also contributes;

although SLE is present in all ethnic groups, it predominates among non-Caucasian individuals and presents greater severity in Latin American patients and those of African descent. In addition, the Black population also tends to receive an earlier diagnosis [7].

Regarding regions of Brazil, a higher number of deaths was observed in the Central-West, Northeast, and North, with a predominance of individuals of mixed race. In contrast, a higher proportion of White individuals was observed in the Southeast and South. Furthermore, in the Northeast region, conditions such as undernutrition, exposure to ultraviolet radiation, and difficulties in accessing adequate treatment and essential medications may contribute to increased SLE lethality [8].

Most deaths occurring at all stages of SLE are due to infections associated with this pathology. In addition, recurrent complications of the circulatory and respiratory systems are notable, along with other comorbidities that may cause damage to the body, such as diabetes and hypertension [8]. Conditions associated with SLE, such as diseases of the genitourinary system, pose a greater risk for the development of different comorbidities, which can negatively impact long-term prognosis and patient mortality [6].

From an epidemiological perspective, SLE is not a compulsory notification disease, as it is not included in the list of quarantine or contagious diseases, according to Law No. 6,259 of 1975. As a result, there are few epidemiological studies available, which is explained by the limited amount of data recorded in official computerized systems [8].

Nevertheless, it is extremely important to conduct future research on epidemiology, as well as to analyze the affected population and the main conditions related to the pathology. Such studies are relevant for the implementation of strategies and public policies, with an emphasis on early diagnosis and improving the quality of life of individuals with SLE [8].

Pathophysiological events of SLE

SLE is a chronic, inflammatory autoimmune disease whose causes remain unknown [9]. However, genetic, hormonal, and environmental factors contribute to its development. Thus, the most widely accepted theory today is that external factors are involved in disease development, especially in the presence of genetic predisposition and the use of certain medications [4].

To update references on the immunological mechanisms of SLE, recent studies highlight that an imbalance in autoantibody production remains central to the pathogenesis of the disease, as it leads to the formation of immune complexes that deposit in blood vessels and trigger inflammation. These processes affect multiple organs, such as the skin, kidneys, heart, lungs, and joints, with an exacerbated inflammatory response due to the involvement of antibodies that attack the body's own proteins. As a result, the pathology is characterized by the presence of autoantibodies directed against antigens of nuclear origin [10].

In addition, new research shows that hyperactivation of the immune system, especially pathways involving type I interferon, plays a significant role in the continuous activation of immune cells, exacerbating tissue damage. This phenomenon, known as the type I interferon signature, is one of the recent discoveries that helps clarify how SLE causes widespread tissue injury, severely affecting organs such as the nervous and cardiovascular systems [11].

More recent studies confirm that B lymphocytes play a central role in the pathogenesis of SLE, as they produce autoantibodies and form immune complexes that deposit in tissues, triggering inflammation. These lymphocytes,

activated by various stimuli such as Toll-like receptors and type I interferons, migrate and proliferate in target tissues, forming aggregates that contribute to organ damage. In addition, activated helper T lymphocytes promote the autoimmune response, while the regulatory function of suppressor T lymphocytes (Tregs) is impaired, contributing to the loss of immune tolerance [12].

Tissue inflammation in SLE is further aggravated by activation of the complement system, which intensifies the immune response and immune complex formation. This mechanism, combined with inefficient clearance of apoptotic cells, leads to recurrent presentation of autoantigens, perpetuating the inflammatory process and chronic tissue damage associated with SLE. These findings highlight the crucial role of B and T lymphocytes in the autoimmune response in SLE and provide the basis for more recent therapeutic approaches, such as the use of Chimeric Antigen Receptor T Cells (CAR-T), which may better direct the immune system to restore balance and reduce SLE-related damage [13].

Described clinical manifestations of SLE

The clinical and laboratory manifestations of SLE are diverse and vary widely among individuals [14]. This represents a major challenge for achieving early diagnosis and effective management, as this characteristic of the disease allows it to be confused with other pathologies [15]. However, according to Garcés [16], some symptoms are described as general and more frequent, namely fever, fatigue, myalgia, and weight loss. Although less common, other manifestations are also described that may affect additional organ systems of the human body.

Current literature clearly shows that mucocutaneous manifestations of SLE affect the majority of patients. The most common lesion is characterized as acute cutaneous lupus erythematosus erythema (popularly known as the "butterfly rash"), which appears after sun exposure and affects the cheek and nasal areas while sparing the nasolabial folds. Some patients may also present discoid lesions, which favor the development of cicatricial alopecia. The presence of painless oral and nasal ulcers also serves as a marker of these manifestations [17]. In addition, a large proportion of patients with SLE present altered renal function, which acts as an important factor of morbidity and mortality [18]. Therefore, frequent investigation of the renal system is necessary in order to detect the presence of lupus nephritis, which may progress to more severe conditions causing loss of renal function [17].

Furthermore, there are osteoarticular manifestations, especially inflammatory arthritis, which tends to be migratory and symmetrical, mainly affecting the joints of the wrists, hands, and knees. Pleuropulmonary and cardiovascular manifestations are also described, with pleuritis and pericarditis being the most frequent, respectively [16]. However, patients with SLE who present with dyspnea or chest pain should be considered at risk for coronary artery disease, as they have a higher likelihood of myocardial infarction [19].

Diagnostic approaches for this pathology

The diagnosis and classification of SLE are based on clinical symptoms, physical signs, and laboratory biomarkers that indicate immune activity and inflammation in different organs of the patient. Clinical examination reveals a wide range of symptoms, including swollen and painful joints, skin and oral cavity lesions, fever, muscle wasting, and arrhythmias. Laboratory findings commonly include hematological abnormalities such as thrombocytopenia, hemolytic anemia, and lymphopenia. With regard to immune system alterations, the presence of anti-DNA antibodies directed against the body itself is observed, in addition to abnormal

levels of IgG or IgM and a positive ANA test [20]. Tests such as Erythrocyte Sedimentation Rate (ESR) and C-reactive protein may help identify inflammation caused by SLE, with ESR being more commonly used to monitor treatment response [21].

For diagnosis and clinical research, it is essential to develop consistent classification criteria for SLE. The most widely adopted classification criteria are those established by the American College of Rheumatology (ACR), which include laboratory biomarkers such as proteinuria, presence of urinary casts, hemolytic anemia with reticulocytosis, leukocyte, lymphocyte, and platelet counts, as well as specific antibodies such as anti-Smith (Sm), ANA, and anti-DNA antibodies. Other parameters include total complement activity, complement components (C2, C3, and C4), and lupus erythematosus cells [22].

In 2019, Aringer and colleagues [3] introduced the EULAR/ACR criteria, which emphasized specificity—raising it to the high level of the revised ACR criteria—while simultaneously increasing sensitivity. ANA became a mandatory entry criterion, despite not being specific to SLE, and individual weights were assigned to all items within the criteria. Ten domains were defined (seven clinical and three immunological), encompassing a total of 22 criteria with distinct weights, requiring a minimum score of 10 for a patient to meet the classification criteria for SLE. The clinical domains include constitutional, hematologic, neuropsychiatric, mucocutaneous, serositis, musculoskeletal, and renal. The immunological domains assess antiphospholipid antibodies, complement system proteins, and specific antibodies such as anti-dsDNA or anti-Smith. The most extensively studied antibodies in lupus are anti–double-stranded DNA antibodies, a subgroup of antinuclear antibodies (usually of the IgM or IgG classes) that can bind to both single-stranded and double-stranded DNA [20].

The clinical form remains the most commonly used approach for diagnosis, as approximately 75% of patients with SLE present with cutaneous manifestations during the course of the disease, which represent the first sign in about 25% of cases. Cutaneous lesions are divided into lupus-specific lesions, such as acute cutaneous lupus erythematosus, which manifests as a "butterfly-wing" rash or generalized exanthema, and discoid lupus erythematosus, characterized by inflammatory plaques that may cause irreversible alopecia. Non-specific lesions include vascular changes such as telangiectasia and Raynaud's phenomenon [23].

Among musculoskeletal manifestations, arthralgia and synovitis are frequently observed, affecting approximately 90% of patients. The most common presentation is symmetric polyarthritis involving joints such as the metacarpophalangeal and proximal interphalangeal joints, as well as the knees. Renal involvement occurs in approximately 50% of patients with SLE. Early detection and treatment are essential, as lupus nephritis is one of the main causes of morbidity and mortality in SLE [24]. Lupus nephritis is a glomerulonephritis characterized by proteinuria and hematuria, especially with the presence of dysmorphic erythrocytes, in addition to red blood cell casts in urinary sediment [23].

Neurological and psychiatric involvement in SLE is highly diverse, presenting a broad spectrum of manifestations ranging from mild cognitive dysfunction to acute confusion, psychosis, and stroke [25].

In summary, the diagnosis and classification of SLE are complex processes that involve a combination of clinical symptoms, physical signs, and laboratory biomarkers. Disease manifestations are varied and may affect multiple systems. Furthermore, early detection of complications such as lupus nephritis and neurological involvement is essential to reduce

the morbidity and mortality associated with SLE. Therefore, an integrated and systematic approach is crucial for the effective management of this autoimmune disease, enabling improved quality of life for affected patients [26].

Treatment according to current literature

The treatment of SLE includes antimalarials, corticosteroids, and immunosuppressants. However, there remains a significant unmet need for new therapies, given the high risk of mortality and the progression of organ damage associated with the disease [27].

Hydroxychloroquine, for example, is an antimalarial drug that inhibits B-cell activation and has demonstrated relevant benefits. However, it is important to note that its use may be associated with an increased frequency of seizures [27].

Corticosteroids, such as prednisone and methylprednisolone, are frequently used in disease management [28]. These drugs aim to reduce the signs and symptoms of the condition, as well as to prevent associated complications. They act by modulating the inflammatory response, regulating the expression of transcription factors, and reducing the synthesis of prostaglandins, plasminogen, and histamine. Nevertheless, chronic use of this class of medications may result in the development of significant side effects. Thus, although corticosteroids can induce disease remission, administration at high doses for prolonged periods may lead to serious complications [29].

Immunosuppressants, including cyclophosphamide and methotrexate, play a crucial role in disease control, especially in more severe cases. Rituximab, a chimeric anti-CD20 monoclonal antibody, is also widely used in the treatment of SLE, aiming at B-cell depletion. On the other hand, belimumab, a fully human IgG1λ monoclonal antibody that specifically binds to BLyS, reduces the number of circulating B cells and is particularly useful in patients with persistent disease activity despite standard therapies [27].

Recent studies comparing belimumab and rituximab demonstrate specific benefits of each agent, with belimumab standing out in terms of long-term disease activity control, especially when used after rituximab. Belimumab inhibits the B-cell Activating Factor (BAFF) and has shown potential to reduce SLE relapses after rituximab therapy. This effect is particularly relevant because BAFF levels may increase after rituximab, triggering new inflammatory flares. Studies such as BLISS-BELIEVE, which evaluate the combination and sequencing of these therapies, indicate that adding rituximab to belimumab did not significantly increase short-term disease control or remission, but may have additional effects in reducing anti-DNA antibodies in specific patients. Belimumab, being a fully human antibody, has less potential for immunogenicity compared to the chimeric antibody rituximab [30].

Other promising therapeutic agents are also under investigation, such as baricitinib, a Janus kinase inhibitor that blocks the JAK1 and JAK2 subtypes, and ustekinumab, a human monoclonal antibody that targets IL-12 and IL-23 [27].

Furthermore, it is essential that treatment not be limited to pharmacological interventions alone. A multidisciplinary approach is fundamental and should include psychological support, dietary guidance, and regular physical exercise. Patients should be educated about the disease and its potential complications, and receive guidance regarding the need for preventive examinations [28]. Recent studies indicate that cognitive and behavioral interventions are particularly effective in reducing symptoms of stress, anxiety, and depression, which are common among patients with SLE. This support not only promotes

emotional well-being but also facilitates treatment adherence and may help mitigate the impact of medication side effects. Stress management therapies, such as biofeedback and cognitive-behavioral therapy, have demonstrated significant benefits in clinical trials, improving self-perception and emotional coping skills, which may lead to a more positive adaptation to life with SLE [31].

Patient diversity and differing responses to treatments highlight the need for personalized therapeutic approaches. In addition, the toxicity of immunosuppressive therapies and the complexity of managing chronic disease symptoms must also be considered [27].

In conclusion, the treatment of SLE requires an integrated and multidisciplinary approach that takes into account both pharmacological and non-pharmacological interventions. Continuous advances in research and the development of new therapies are crucial to addressing the challenges posed by this condition, always with the aim of improving patients' quality of life [27].

Despite major progress in targeted and biologic therapies, several challenges persist. Agents such as belimumab and rituximab show variable responses across patients, underscoring the marked heterogeneity of SLE and the ongoing need for reliable biomarkers to predict treatment response. Moreover, high costs, concerns about long-term safety, and limited availability — particularly in low- and middle-income countries — may restrict broader use of these therapies. Emerging treatments, including JAK inhibitors and CAR-T cell–based strategies, offer promising immunomodulatory effects; however, their long-term efficacy, safety profile, and applicability in real-world clinical settings still need confirmation through large, well-designed clinical trials.

Forms of prevention

Recent literature indicates that, although there is still no established way to prevent SLE, certain lifestyle practices may help mitigate complications and improve patients' quality of life. Recommendations include regular physical exercise, a balanced diet, and adequate sun protection. In addition, emotional and psychological support is essential for coping with the stress and anxiety associated with the disease, promoting improvements in overall well-being. These guidelines emphasize the importance of medical follow-up and adjusting the frequency and intensity of physical activities according to symptom fluctuations, so that patients can engage safely and effectively in an active lifestyle. Initially, SLE does not present specific preventive measures; however, it has been shown that individuals with this condition are more likely to have an unsatisfactory perception of their health-related quality of life [1]. Thus, the importance of adopting healthy lifestyle practices, such as physical activity, is evident [32].

Additionally, SLE is a chronic autoimmune disease that affects multiple systems of the human body, resulting in high morbidity and mortality rates. Cardiovascular, infectious, and renal complications are commonly observed. In this context, it is well established that physical activity provides benefits in chronic diseases. In particular, patients with SLE show improvements in cardiovascular health with regular exercise, as it increases muscle strength, reduces the likelihood of inflammatory processes, and improves mental health—an important factor in ensuring better quality of life for patients with SLE. Consequently, physical activity acts as a preventive factor by reducing cardiovascular risk [33].

Moreover, it is important to highlight the frequent occurrence of fibromyalgia, a non-articular and non-inflammatory condition that causes intense body pain, leading to fatigue, mental confusion, difficulty with mobility, among other symptoms, in patients with SLE.

This condition, in addition to causing disability and difficulty in performing activities of daily living, is a major contributor to symptom recurrence and is closely associated with symptom severity. Therefore, proper care and treatment of fibromyalgia represent a preventive approach to disease worsening and to improving the health–disease relationship of patients with SLE [34].

In this context, the vicious cycle of inflammation caused by the disease, combined with the social context in which patients live, interferes with the worsening of SLE. This occurs because symptoms associated with the pathology impair psychosocial and interpersonal relationships due to the disease process, particularly among women, who often experience low body self-esteem and decreased self-acceptance. Thus, it is evident that care for the mental and social health of patients with SLE is extremely important, especially as a preventive measure. As previously mentioned, this is particularly relevant in females, in whom the disease is more prevalent and who may be more affected by societal beauty standards and by how they perceive themselves in relation to these norms [35,36].

Conclusion

SLE is a complex autoimmune disease with significant implications for patients' lives. Special attention to the most affected demographic groups is essential, requiring an inclusive public health approach. Understanding the immunological mechanisms involved in the disease, as well as the diagnostic criteria, is fundamental for effective SLE management.

Notably, patients face daily challenges due to the chronic nature of the disease, which may lead to social stigma, often resulting in isolation and difficulties in interpersonal relationships. Patients' mental health should also be a major focus of care, as the variable and unpredictable symptoms of the disease can interfere with work capacity and daily activities.

Treatment, both pharmacological and non-pharmacological, should be individualized and include strategies to promote patients' physical and emotional health through a comprehensive multidisciplinary approach that considers not only clinical aspects, but also the psychological and social impact of the disease. Awareness of symptoms and the importance of early diagnosis can help reduce the adverse effects of SLE on patients' quality of life. Therefore, continuous research and the implementation of evidence-based multidisciplinary strategies are essential to optimize early detection, treatment outcomes, and quality of life in patients with SLE.

By bringing together current insights into immunology, modern therapeutic approaches, and patient-centered preventive perspectives, this review aims to connect advances in basic science with everyday clinical practice. This integrated view may help guide more informed clinical decisions and point to key directions for future research in SLE. Future research should prioritize the identification of reliable biomarkers to support early diagnosis, monitor disease activity, and guide individualized treatment decisions. In parallel, expanding access to emerging therapies and reinforcing multidisciplinary and psychosocial care strategies will be essential to improving long-term outcomes for patients with SLE.

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