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UPDATE ON THE DIAGNOSIS AND MANAGEMENT OF SYSTEMIC LUPUS ERYTHEMATOSUS

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Abstract:

Background: *systemic lupus erythematosus (SLE) is a complex autoimmune disease affecting multiple organ systems, characterized by immune dysregulation and the production of autoantibodies. Pathogenesis involves aberrant activation of programmed cell death (PCD) signaling and accelerated cell death, leading to self-antigens release. SLE manifests with diverse symptoms, ranging from musculoskeletal and gastrointestinal symptoms to neuropsychiatric and cardiovascular complications.*

Objective: *The present study aims to review the update on the diagnosis and management of systemic lupus erythematosus*

Methods: *Comprehensive research of the diagnosis and management of SLE. PUBMED and Google scholar search engines were the database used for the search process, and articles were collected from 2013 to 2023. The term used in the search were: Systemic lupus erythematosus (SLE) – manifestation – Diagnostic criteria – treatment.*

Conclusion: *Accurate diagnosis relies on established criteria like ACR-1997, SLICC-2012, and EULAR/ACR-2019, incorporating clinical and immunological features. Various diagnostic methods, such as immunoassays and multi-omics analysis, contribute to precise diagnosis, with ongoing evaluation of criteria in juvenile SLE. SLE management involves a multidisciplinary approach, combining conventional therapies, targeted biological agents, and adjuvant treatments. The focus remains on improving treatment response, enhancing quality of life, and minimizing side effects. Ongoing research endeavors seek to deepen our understanding of SLE and develop novel therapeutic strategies for this challenging autoimmune disorder.*

Keywords: *Systemic lupus erythematosus (SLE) – manifestation – Diagnostic criteria – treatment.*

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INTRODUCTION:

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by an aberrant host immune response and chronic inflammation [1]. It affects multiple organ systems, including the cardiovascular, gastrointestinal, hematologic, integumentary, musculoskeletal, neuropsychiatric, pulmonary, renal, and reproductive systems that can cause recurrent flare-ups without adequate treatment [2-4]. SLE is characterized by immune dysregulation and the production of various autoantibodies. The pathogenesis of SLE involves aberrant activation of programmed cell death (PCD) signaling and accelerated cell death, leading to the release of self-antigens. These self-antigens, particularly nucleic acids and nucleic acids-protein complexes, activate auto-reactive B Lymphocyte Stimulator (BLyS) and promote interferon-I responses, contributing to the development of SLE [5]. Defective apoptosis is believed to play a critical role in SLE development.

SLE manifestation:

Systemic lupus erythematosus (SLE) is characterized by various symptoms that can affect multiple organs and systems in the body and result from autoantibodies, immune complexes, and cytokines with autoantibody specificities[6]. These symptoms include inflammation and disease activity (type 1 symptoms) as well as fatigue, anxiety-depression, pain, and respiratory manifestations (type 2 symptoms) [7]. The inflammatory activity and type 1 symptoms of SLE, such as joint pain and swelling, are often poorly correlated with type 2 symptoms and health-related quality of life (HRQoL) [8]. Musculoskeletal (MSK) symptoms, such as joint pain, swelling, and stiffness, are common in SLE flares and negatively impact functioning and HRQoL [9]. Lupus enteritis, characterized by abdominal pain, nausea, vomiting, and diarrhea, can also occur during an SLE flare [10].

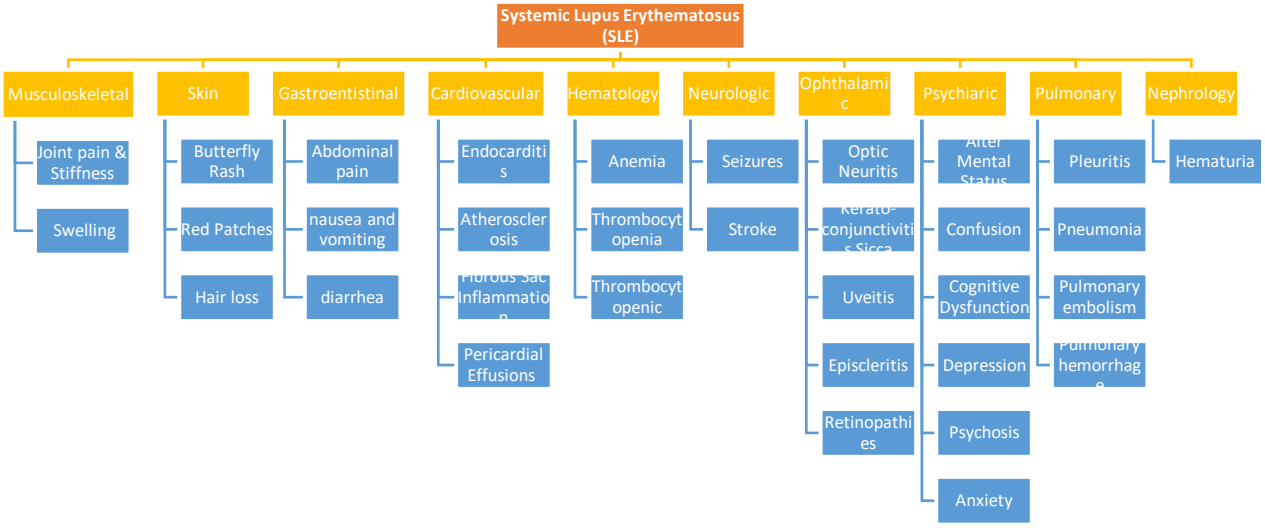

Figure (1): Systemic lupus erythematosus manifestations.

In addition to these symptoms, SLE can also manifest with neuropsychiatric symptoms such as movement disorders and cognitive disturbances [11]. Other neurological manifestations can include seizures and stroke [12]. Cutaneous manifestations are common in SLE and can take various forms, ranging from mild skin rashes to more severe lesions that can cause functional and aesthetic disabilities [13]. Ocular manifestations of SLE can include keratoconjunctivitis sicca, uveitis, episcleritis, scleritis, optic neuritis, and retinopathies [4]. Hematological manifestations include anemia, thrombocytopenia, and thrombotic thrombocytopenic purpura [14]. Other possible signs of SLE can involve the cardiovascular system, such as pericardial and pleural effusions [15]. Psychiatric complications such as altered mental status, acute confusional state, cognitive dysfunction, mood disorder (depression), psychosis, and anxiety disorder are common in SLE patients [9].

Diagnosis of SLE:

The diagnostic criteria for systemic lupus erythematosus (SLE) include the American College of Rheumatology (ACR)-1997 criteria, the Systemic Lupus International Collaborating Clinics (SLICC)-2012 criteria, and the European League Against Rheumatism (EULAR/ACR)-2019 criteria. These criteria have been developed to diagnose SLE effectively and have been validated in various populations. The ACR-1997 criteria include a combination of clinical and laboratory features, while the SLICC-2012 criteria have a broader range of clinical and immunological manifestations. The EULAR/ACR-2019 criteria include positive antinuclear antibody (ANA) as an obligatory entry criterion and weighted criteria grouped in clinical and immunological domains. These criteria have shown good sensitivity and specificity for diagnosing SLE in different populations, including juvenile SLE. However, the applicability of the EULAR/ACR-2019 criteria in juvenile SLE is still being evaluated [16-18].

American College of Rheumatology (ACR)-1997 criteria	Malar rash Discoid rash Photosensitivity Oral ulcer Arthritis Serositis Renal disease Neurologic disorder	
	Hemolytic anemia with reticulosis Leukopenia < 4000/mm^3 Lymphopenia < 1500/mm^3 Thrombocytopenia <10x10^3/mm^3	
	Anti-dsDNA positive Anti-Sm positive Anti-Phospholipid positive ANA test Positive	
Systemic Lupus International Collaborating Clinics (SLICC)-2012 criteria	Acute cutaneous lupus Chronic cutaneous lupus Oral or nasal ulcer Non-scarring alopecia Arthritis Serositis Renal disease Neurologic disorder Hemolytic anemia Leukopenia Thrombocytopenia <10x103/mm3	
	ANA test Positive Anti-dsDNA positive Anti-Phospholipid positive Low complement (C3, C4, CH50) Direct Coombs' test [not count if case of hemolytic anemia]	
European League Against Rheumatism (EULAR/ACR)-2019*	Fever Leukopenia Thrombocytopenia Autoimmune hemolysis Delirium Psychosis Seizure Oral or nasal ulcer Non-scarring alopecia Discoid rash Acute cutaneous lupus Pleural or pericardia effusion Acute pericarditis Proteinuria >0.5g/24h Biopsy of II or V lupus nephritis Biopsy of III or IV lupus nephritis	2 3 4 4 2 3 5 2 2 4 6 5 6 4 8 10
	Anti-Phospholipid positive Low complement C3 OR C4 Low complement C3 and C4 Anti-dsDNA or Anti-Sm positive	2 3 4 6
	* Classify as SLE with a score of 10 or more.	

Table (1): Systemic lupus erythematosus diagnostic criteria.

Systemic lupus erythematosus (SLE) can be diagnosed using various methods. Commonly used techniques include Line immunoassay (LIA), enzyme-linked immunosorbent assay (ELISA), and Crithidia luciliae indirect immunofluorescence (CLIF) assay [19]. Another approach is multi-omics analysis, which involves evaluating serum metabolic spectrum and protein levels using gas chromatography-mass spectrometry (GC-MS) and Tandem Mass Tag (TMT) quantitative detection technology [20]. The enzyme immunoassay and immunofluorescence assay (IFA) are also commonly used for detecting antinuclear antibodies (ANAs) [21]. Additionally, different methods such as fluorescence enzyme immunoassay, microdot array, chemiluminescent immunoassay, multiplex flow immunoassay, and particle multi-analyte technology immunoassay can be used to measure anti-double-stranded DNA (anti-dsDNA) antibody levels [22].

Treatment of SLE:

The goal of treatment is to decrease symptom severity and achieve remission without organ damage. Treatment options for systemic lupus erythematosus (SLE) include antimalarials, glucocorticoids (GCs), immunosuppressants (ISs), and biological agents. Hydroxychloroquine is commonly used as an antimalarial treatment for SLE [23]. GCs are often used, but their dosages are minimized or discontinued due to adverse reactions [24]. ISs, such as cyclophosphamide, minimize GCs and prevent flares [3].

Antimalarials:

hey are considered a mainstay in managing SLE and have been widely used since the 1950s[25]. Hydroxychloroquine, a type of antimalarial, is the most commonly prescribed antimalarial for SLE and is recommended for all patients with the disease which have been shown to decrease symptom severity and suppress disease activity, particularly in skin manifestations and fatigue, also have steroid-sparing properties, allowing for the reduction or discontinuation of glucocorticoids (GCs) [26,27].

Glucocorticoids (GCs):

Glucocorticoids are commonly used in the treatment of systemic lupus erythematosus (SLE), considered the gold standard treatment, and have potent and fast actions that can quickly relieve symptoms and lower mortality in life-threatening conditions and are used mainly to reduce immune activation and inflammation [25,28]. Glucocorticoids are used to induce remission or treat acute situations and as maintenance therapy in SLE [29]. However, the use of glucocorticoids is associated with side effects that limit the duration and dose of treatment. To minimize the adverse reactions of glucocorticoids, immunosuppressants are used as steroid-sparing agents [30]. Despite the side effects, glucocorticoids remain an important component of therapy for controlling acute disease-related inflammation in SLE.

Immunosuppressants:

Immunosuppressants are used in the treatment of systemic lupus erythematosus (SLE) as steroid-sparing agents to minimize the use of glucocorticoids (GCs) and to reduce disease activity and prevent flares [25,31]. Some commonly used immunosuppressants in SLE include azathioprine, methotrexate, mycophenolate mofetil, cyclophosphamide, tacrolimus, and cyclosporin A [32]. These medications are used to prevent the accumulation of damage to the main organs affected by SLE and to minimize the side effects of long-term GC use [3]. In cases of lupus nephritis, pulse methylprednisolone, mycophenolate mofetil, and cyclophosphamide are used as induction treatments [33]. In non-renal SLE, rituximab, an anti-CD20 biologic agent, may be used. Belimumab, another biologic agent, can be used in non-renal SLE cases that are not controlled with other medications. The development of new immunosuppressants and targeted therapies is an area of ongoing research in the treatment of SLE.

Biological agents:

Biological agents, including belimumab and anifrolumab, are recommended when other treatments fail to target various pathogenetic pathways and improve treatment response and quality of life [25]. B cell targeting agents, such as belimumab, a B cell targeting agent, and rituximab, an anti-CD20 targeting agent, have been successfully used in SLE management [24,34]. Anifrolumab, an interferon I receptor-targeting agent, has shown beneficial effects on SLE. Biologic therapies, including monoclonal antibodies, have reduced the risk of flares and improved serological activity in SLE patients [35].

Janus kinase inhibitors:

Tofacitinib, a JAK1/3 inhibitor, has been shown to reduce cholesterol levels, improve vascular function, and decrease the type I interferon signature in SLE patients . Baricitinib, a JAK1/2 inhibitor, demonstrated significant improvements in lupus rashes and arthritis in phase 2 and phase 3 trials . Deucravacitinib, a selective TYK2 inhibitor, has shown promise in a phase 2 trial and will be investigated further in larger phase 3 trials . JAK inhibitors have the potential to modulate various immune networks and regulate the complex immunopathogenesis in SLE . However, the efficacy of JAK inhibitors in the treatment of SLE is still being

determined, and further studies are needed to assess their risk-benefit ratio and identify the most appropriate patients for treatment [23,36,37]

Adjuvant Treatments:

Intravenous immunoglobulins (IVIG) have been used as a monotherapy for the management of systemic lupus erythematosus (SLE) which found to be effective in the treatment of lupus nephritis (LN), with overall responses in 60-70% of patients [38]. IVIG prepared using purified human plasma and has immunomodulatory effects on autoimmune diseases, including severe SLE. in addition, Therapeutic plasma exchange (TPE) which is extracorporeal blood purification technique used to remove immunological active substances from plasma and provide essential factors when supplementing with plasma [39]. TPE has been used successfully in the treatment of SLE ocular involvement, particularly in cases where the patient's condition rapidly deteriorates. In one case study, a 21-year-old female with SLE presented with bilateral decreased vision and severe ocular manifestations. Despite high-dose steroid therapy, the patient's condition worsened, but TPE was successful in treating the ocular involvement [40].

CONCLUSION:

Accurate diagnosis relies on established criteria like ACR-1997, SLICC-2012, and EULAR/ACR-2019, incorporating clinical and immunological features. Various diagnostic methods, such as immunoassays and multi-omics analysis, contribute to precise diagnosis, with ongoing evaluation of criteria in juvenile SLE. SLE management involves a multidisciplinary approach, combining conventional therapies, targeted biological agents, and adjuvant treatments. The focus remains on improving treatment response, enhancing quality of life, and minimizing side effects. Ongoing research endeavors seek to deepen our understanding of SLE and develop novel therapeutic strategies for this challenging autoimmune disorder.

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