OPEN ACCESS Freely available online

ISSN: 0974-8369
Biology and Medicine

Short Communication

Systemic Lupus Erythematosus: Exploring its Pathogenesis, Clinical Features and Therapeutic Approaches

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DESCRIPTION

Systemic Lupus Erythematosus (SLE) is an autoimmune disorder, a multisystem autoimmune disease includes immunological complex deposition, persistent inflammation in the traditional target organs the skin, joints and kidneys. However, in people with SLE the immune system produces autoantibodies that target and destroy healthy cells and tissues. This immune response leads to chronic inflammation and damage to various organs [1,2].

The effects of SLE remains high even with significant advancements in diagnosis and treatment. To enable early patient referral and diagnosis, it is important to understand typical presentations and the diagnostic process. The majority of patients appear with constitutional, mucocutaneous, and musculoskeletal symptoms initially; these symptoms may include myalgia, exhaustion, mouth ulcers, alopecia, joint pain and rash specific to lupus. The most recent classification criteria, a diagnostic strategy for symptoms of SLE includes an organised assessment of clinical manifestations (weighted within each domain) and autoantibody profiles (such as anti-Sm, anti-double-stranded DNA, antiphospholipid, or hypocomplementaemia antibodies) [3]. Individualised lifestyle interventions and patient education are used in non-pharmacotherapy management to enhance quality of life and medication adherence (e.g., hydroxychloroquine or immunosuppressant). A few significant advancements in lupus nephritis and SLE treatments have been made in the past ten years, including belimumab, anifrolumab and voclosporin. The disease progression is still unpredictable and the death rate is too high. Different parts of the world have imposed restrictions on access to these pricey treatments. Vigilant prevention and management measures are required for comorbidities resulting from both disease activity and medication adverse effects, particularly infections, osteoporosis and cardiovascular disease. Priority areas of managing SLE include balancing treatment-related comorbidities and customising therapy options to achieve remission.

The pathogenesis of SLE is complex and involves multiple factors. Genetic predisposition plays a significant role, as several genes associated with the immune system have been linked to the disease. Environmental factors, such as Ultraviolet (UV) light exposure, infections and certain medications, can trigger the onset or exacerbation of SLE symptoms. Hormonal influences, particularly estrogen, are also thought to contribute to the higher prevalence of SLE in women [4,5].

The clinical presentation of SLE is highly variable, with symptoms ranging from mild to severe. The disease can affect virtually any organ system, leading to a wide array of symptoms. Common manifestations include fatigue, joint pain and swelling, skin rashes (especially the characteristic "butterfly" rash on the face), fever, and photosensitivity. Some patients may experience more severe complications, such as kidney inflammation (lupus nephritis), neurological symptoms (seizures, psychosis), and cardiovascular issues (pericarditis, myocarditis) [6]. The chronic nature of SLE, combined with its unpredictable course, can significantly impact a patient's quality of life. Flares, periods when symptoms worsen are common and can be triggered by various factors, including stress, infections and exposure to sunlight.

Diagnosing SLE can be challenging due to its diverse and nonspecific symptoms. There is no single test to confirm the disease, so diagnosis often involves a combination of clinical evaluation, laboratory tests and imaging studies. Laboratory tests play an important role in diagnosing SLE and assessing disease activity. ANA is the most common autoantibody found in SLE patients, present in more than 95% of cases [7,8]. Other specific autoantibodies, such as anti-double-stranded DNA (anti-dsDNA) and anti-Smith (anti-Sm) antibodies are also associated with the disease and can help confirm the diagnosis. Additionally, blood

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Received: 24-Jun-2024, Manuscript No. BLM-24-26735; **Editor assigned:** 26-Jun-2024, PreQC No. BLM-24-26735 (PQ); **Reviewed:** 10-Jul-2024, QC No. BLM-24-26735; **Revised:** 17-Jul-2024, Manuscript No. BLM-24-26735 (R); **Published:** 24-Jul-2024, DOI: 10.35248/0974-8369.24.16.703

Citation: Samo UB (2024). Systemic Lupus Erythematosus: Exploring its Pathogenesis, Clinical Features and Therapeutic Approaches. Bio Med. 16:703.

tests may reveal anemia, leukopenia, thrombocytopenia, and elevated markers of inflammation, such as Erythrocyte Sedimentation Rate (ESR) and C-reactive protein (CRP). There is no cure for SLE, but with appropriate treatment many patients can manage their symptoms and prevent serious complications. The treatment approach for SLE is individualized, depending on the severity of the disease and the specific organs involved. The primary goals of treatment are to control inflammation, prevent flares and minimize organ damage [9,10].

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