RESEARCH PAPER

The Value of PLR, and NLR in Assessment of Systemic Lupus Erythematosus Patients

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Received: 20th Feb, 2026 | **Revised:** 4th Mar, 2026 | **Accepted:** 25th Mar, 2026 | **Available Online:** 10th Apr, 2026

ABSTRACT

Systemic lupus erythematosus (SLE) is a systemic autoimmune disorder that involves skin, joints, renal, cardiac, and/or neurologic pathology. Classification of SLE as predominantly an immune complex-mediated autoimmune disease was detected after isolation of complexes of antibody with DNA, RNA, or DNA- or RNA-binding proteins. No organ is immune from involvement in disease process in SLE. The disease process can show periods of remissions and exacerbations. Mucocutaneous exacerbation can induce systemic exacerbation. While serositis, arthralgia and arthritis represent the mild to moderate form of disease severity in SLE, cytopenias, renal involvement and neuropsychiatric lupus (NPSLE) can be presented with different grades of severity. Renal affection is common in SLE and has a significant impact on morbidity and mortality. Disease activity in SLE can be measured by tests like dsDNA, C3 and C4 or scores like SLEDAI. This score evaluates SLE patients clinically and laboratory giving them different weights according to health effects.

How to cite this article: Elmohsen AA, Abdelkareem MI, Nafady A, Mohammed AM. The Value of Plr, and Nlr in Assessment of Systemic Lupus Erythematosus Patients. Int J Drug Deliv Technol. 2026;16(29s):471-475. DOI: 10.25258/ijddt.16.29s.59

Source of support: Nil.

Conflict of interest: The authors declare no conflict of interest.

Definition

Systemic lupus erythematosus (SLE) is a systemic autoimmune disorder that typically involves skin and joints, with renal, cardiac, and/or neurologic pathology often documented with histologic or imaging studies. Underlying immunopathogenic mechanisms have been investigated along many decades and showed a great progress. Early studies told us that alterations of blood vessels in many organs represent an essential component of disease process. Onion skinning" of splenic arterioles, vasculopathy in skin and brain and cellular infiltration and damage to renal glomeruli. [1] In 1948, lupus erythematosus (LE) cell was described by Hargraves. Engulfment of cellular debris is still an important concept in understudying of lupus pathogenesis. Role of immune system was indicated by recognition that antibodies directed at cellular components. Classification of SLE as predominantly an immune complex-mediated autoimmune disease was detected after isolation of complexes of antibody with DNA, RNA, or DNA- or RNA-binding proteins, such as histones or components of the spliceosome from patient sera. Pathologic studies of kidneys from patients with lupus nephritis showing glomerular deposition of immunoglobulin and complement components, combined with the elution of anti-DNA autoantibodies from lupus kidneys, contributed to the concept that autoantibodies, particularly anti-DNA antibodies, were pathogenic.

Occurrence of several individuals with SLE within a family, along with a high frequency of concordance of SLE in identical twins are good clues for role of genetic background in disease progress. Also, multiple autoimmune diseases are associated with common genetic susceptibility factors.[2] Autoantibodies represent the corner stone in pathogenesis of SLE. TLR-dependent and -independent innate immune signaling, and lymphocyte activation results in production of autoantibodies and their immune complexes, cytokines, and complement components, along with pro-inflammatory mediators and reactive oxygen products.[3] Almost anti-dsDNA and anti-Sm Autoantibodies are traditionally viewed as essential mediators of pathology in SLE, particularly when they are in the form of immune complexes.[4,5] Anti-Ro, anti-La, and anti-RNP antibodies are characteristic of SLE but are also seen in other systemic autoimmune diseases. The characteristic autoantibodies in SLE can be categorized in relation to their targets, including DNA and DNAbinding proteins, typically aggregated in nucleosomes that contain histones; RNA and RNA-associated proteins, typically aggregated in cytoplasmic or nuclear ribonucleoprotein (RNP) particles; phospholipids exposed in plasma membranes and associated proteins such as β2-glycoprotein I; and cell membrane proteins, typically those expressed on blood cells. Some of these self-antigens typically targeted by lupus autoantibodies are most likely accessed by antigen-presenting cells in the form of cell membrane–enclosed blebs derived from apoptotic cells or microparticles, small aggregates of cellular material derived from cells and generated after flipping of phosphatidylserine to the outer aspect of the cell membrane in the setting of cell activation or apoptosis. 2 questions have arised; what about the pathogenic antibodies in SLE and timing of its generation. Preplasma cells or plasma cells that have undergone immunoglobulin class switching are responsible for production of pathogenic antibodies in SLE.[6] it is probable that the generation of autoimmunity can begin in any number of ways. Presentation of self-antigen by an excessively activated antigen-presenting cell, stochastic activation of low-avidity selfreactive T cells that are present in healthy individuals but have the potential to expand and drive differentiation of B cells with broad specificity for self-antigens, or direct activation of selfreactive B cells in the presence of activating TLR ligands, such as those provided by demethylated CpG-rich DNA from bacteria or viruses or self-derived nuclear debris, might all be starting points for development of pathogenic autoimmunity. As noted earlier, microbial epitopes present on endogenous bacteria might also represent an initiating point for generation of autoimmunity.[7]

Clinical manifestation

Mucocutaneous Involvement

Mucocutaneous involvement is very common in SLE and can be manifested with rash, rashes, ulcerations or alopecia. Cutaneous manifestations have been categorized into lupus specific" or "lupus nonspecific'.[8,9] Lupus-specific lesions are further subdivided into acute cutaneous lupus erythematosus (ACLE), subacute cutaneous lupus erythematosus (SCLE), and chronic cutaneous lupus erythematosus (CCLE) lesions. Discoid lupus is the most common subtype of CCLE. Many patients displayed more than one type of cutaneous lesion.[10] ACLE lesions can be localized or generalized. The main feature of these lesions are observed in the malar region and is characterized by confluent, macular, or papular erythema lasting days to weeks, appearing symmetrically on the cheeks and bridge of the nose.SCLE lesions are presented withf nonscarring, photosensitive lesions of two types; papulosquamous lesions and annular-polycyclic lesions. Annular-polycyclic lesions have peripheral scale and central clearing . These two forms can occur concurrently in the same patient. SCLE has a predilection for the back, neck, shoulders, and extensor surfaces of the arms and usually spares the central face. The lesions typically last for weeks to months and heal without scarring.[11] CCLE lesions are characterized by skin atrophy and scarring. These lesions may persist for several months or years. DLE is subdivided into localized discoid lupus (limited to head and neck) and generalized discoid (occurring above and below the neck). Left untreated, DLE can result in permanent alopecia and disfigurement. Squamous cell carcinoma has been described as a sequela of long-standing DLE; thus active surveillance of known lesions and evaluation of changing lesions are critical.[12]

Musculoskeletal Involvement

Arthritis and arthralgias are classic manifestations of SLE. They vary from mild joint pain to deforming arthritis. Although any joint can be involved, lupus arthritis is typically characterized by a symmetric, inflammatory arthritis that predominantly affects the knees, wrists, and small joints of the hands.[13]

Renal Involvement

Renal affection is common in SLE and is a significant leading cause of morbidity and mortality. glomerulonephritis may be asymptomatic or presented by frank nephritis.proteinuia is major symptom.[14]

Pleuropulmonary Involvement

One of the common forms of pulmonary affection is pleurisy which develops in about 50% of patients. patient complain of chest pain or it may be asymptomatic and discovered accidentally. Aspiration of massive effusion is indicated . [15]

Cardiovascular involvement

It is frequent complication of SLE.it can involve pericardium, valves and myocardium.[16] pericarditis is the most common clinical manifestation of cardiac affection in SLE.

Gastrointestinal Involvement

SLE may affect any region of the gastrointestinal system. Dysphagia occurs in about 13% of patients and can be caused by heartburn, esophageal disturbed motility, and changed saliva production in the setting of secondary Sjögren's syndrome. [17]

Investigations

No clinical manifestation or laboratory test can serve as a definitive diagnostic test.

(1) Complete blood count

Essential as baseline and for hematological evaluation. cytopenia is common finding either monocytopenia, bicytopenia or pancytopenia. Anemia of chronic disease is considered the most common anemia in SLE. It is a normocytic, normochromic anemia characterized by low serum iron. Autoimmune hemolytic anemia (AIHA) may be suspected in case of elevated serum unconjugated bilirubin, elevated lactate dehydrogenase (LDH). The direct Coombs test is positive.[18, 19]

Leukopenia may occur in patients with SLE and may be secondary to lymphopenia and/or neutropenia.Lymphopenia may be a side effect of treatment with glucocorticoids or other immunosuppressive medications. Neutropenia results from marrow suppression.

thrombocytopenia occurs in 50% of patients with SLE either mild or severe .Thrombocytopenia can be the result of immune-mediated platelet destruction. Thrombocytopenia may be caused by a consumptive process such as splenomegaly. Anti-thrombopoietin antibodies have been found in the sera of some patients with SLE and have been correlated with lower platelet counts.[20, 21]

(2) Complete urine analysis

Microscopical examination important as baseline data in addition to screening and monitoring of glomerulonephritis.Hematuria, dysmorphic, , pyuria red blood cells, red blood cell casts, and white blood cell casts may all be present [22, 23]. Red blood cell casts are important and specific . RBCs & WBCs cast are specific for tubulointerstitial affection. hematuria without proteinuria non specific . proteinuria need accurate measurement. [24]

(3) Renal biopsy

Indicated in a patient with nephritis to determine degree of inflammation ,activity and course of treatment.[25]

(4) Serologic Tests

Serologic tests play an important role in the diagnosis of SLE. SLE is the prototypic systemic humoral autoimmune disease. As such, it is characterized by production of a wide variety of autoantibodies, which often provide important diagnostic information.[26]

Treatment of systemic lupus erythematosus

(a) Glucocorticoids

GCs have general effects on immune responses mediated by T and B cells. On the basis of these effects and their rapid onset of action, GCs are very effective in controlling acute SLE manifestations. Low-dose GC therapy (≤10 mg prednisone equivalent per day) is generally used when initial therapies (mainly anti-malarials) are inadequate to control activity. In moderate to severe disease, GCs are used preferably in combination with immunosuppressive agents, at doses of 0.5 to 0.7 mg/kg in a single dose, usually in the morning. When combined with immunosuppressive agents, the GC dose should rarely exceed 0.5 to 0.6 mg/kg to avoid infections and other toxicities. Gradual withdrawal of GC dose should be started after 4 weeks of therapy, until reaching a maximum daily dose of 7.5 mg after 6 months. Concomitant use of immunosuppressives.[27]

(b) Methotrexate

Methotrexate (MTX) is a steroid-sparing drug.it can be used in mild to moderate SLE, especially for skin & joint manifestations.MTX is administered once weekly orally or parenterally. MTX reflect good result in control of disease activity.Concomitant prescription of folic acid (1 to 2 mg/day) is recommended to minimize toxicity.

(c) Azathioprine

AZA (2 to 2.5 mg/kg/day) is safe in the long term without increased the risk for infections. Gastrointestinal complaints are frequent and mild liver

enzyme elevation may occur, but severe liver injury is rare. bone marrow suppression is common but it is dose related and reversible .leukopenia is encountered in 4.5% and thrombocytopenia in 2% of patients. AZA toxicity has been associated with genetic polymorphisms resulting in decreased thiopurine methyltransferase activity.

(d) Mycophenolate Mofetil

It is used as induction and maintenance therapy. induction therapy for 6 monthes.

Biologic Therapies

(e) Belimumab

Belimumab is a human monoclonal antibody act against the B lymphocyte stimulator protein. [28, 29]

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