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SYSTEMIC LUPUS ERYTHEMATOUS: CLINICAL MANIFESTATIONS, PATHOPHYSIOLOGY, AND DIAGNOSTIC CRITERIA

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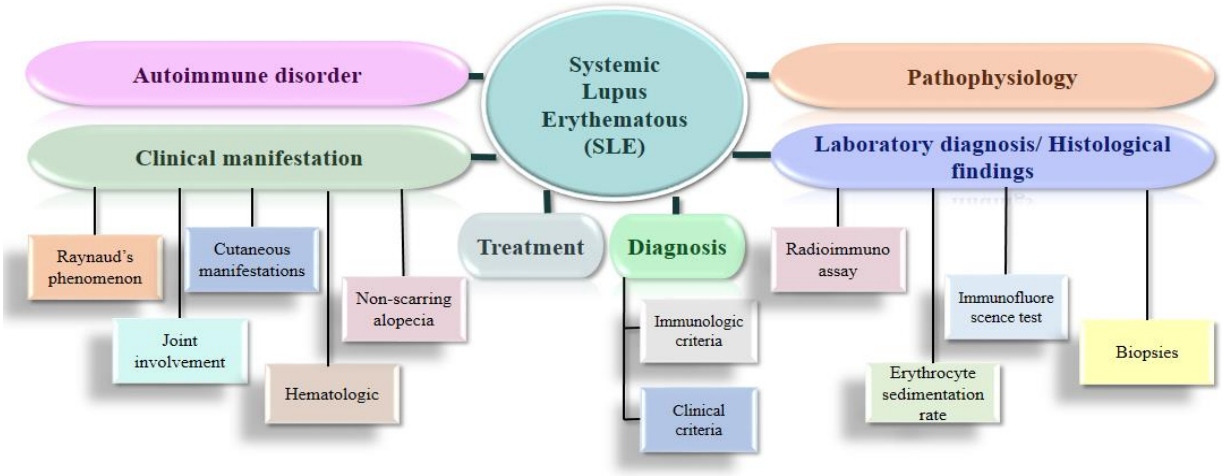
Abstract

Systemic lupus erythematous is an autoimmune disorder with type III hypersensitivity reaction, which spreads over time and eventually affects different organs of the body, making the symptoms worse. Most of the causes of this disease are still unknown; however, its pathophysiology may be assigned to various important components. Clinically, the disease is identified by phases of relapse and remission that can exist as specific and constitutional symptoms of multiple organs. Researchers have studied the disease by clinical findings, immunofluoroscopy, photomicrography, biopsy, and screening tests, and have tried to find the relationship between its inheritance, occurrence, presentation, and advanced manifestations. This review deals with the signs and symptoms, diagnostic criteria, clinical manifestations, histologic findings, and laboratory diagnosis, trying to rule out the basic presentations and pathophysiological effects of this autoimmune disorder.



1. INTRODUCTION

Systemic lupus erythematosus is one of the common types of lupus and is an autoimmune disease that results in the attack of the immune system on own cells and tissues . It causes tissue damage and extensive inflammation in multiple organs by multiple autoantibodies, particularly antinuclear antibodies (ANAs) [1]. The word 'lupus' means any sort of long-term autoimmune disease, making immune system hyperactive; resultantly, attacking the vital organs of the body and 'erythematosus' comes from circular shaped lesions on the face [2, 3]. Multiple organs in the body are affected and damage to the kidneys, joints, skin, and serous membranes impairment is obvious. The signs and symptoms of SLE are variable so multiple set of criteria for the disease have been proposed to help doctors to design and assess several clinical trials. Although the disease frequently appears in the twenties or early thirties, it can be manifested at any age, predominantly affecting women [1]. The hallmark of SLE is the presence of antibodies, especially ANA. These antibodies identify their nuclear and cytoplasmic components and are included in the diagnostic criteria [1].

2. Epidemiology

The American School of Rheumatology (ACR), in 1982, approved an assortment of order standards to achieve a reliable meaning of SLE with the end goal of both research and epidemiological reconnaissance [4]. The Fundamental Lupus Worldwide Working together Centers (SLICC), in 2012, the ACR requirements have been updated to create a more specific and clinically applicable set of criteria [5]. The European League Against Rheumatism (EULAR) and the American College of Rheumatology (ACR) characterization of the disease set up techniques and measures for alteration of the order of lupus nephritis and medication systems. The rate of SLE has been evaluated to be 30-50 for every 1 lac, which is approximately equal to 5 lac patients in Europe and 2.5 lac patients in the USA [6]. A study found that lineage, race, and ethnicity have important effects on the presentation and severity of SLE [7]. The Hispanic, Asians, and Black patients appear to progress lupus earlier and have more serious and active disease, exhibit long-term damage by the disease and have increased mortality, occurrence and prevalence of the disease than in white patients [7,8]. The disparities observed are primarily associated with genetic variance and exposure to the local environment. About 90%, in general, are childbearing females. One of the vital components in clinical indications and visualization of SLE is its stage, while the pinnacle occurs during conceptive years. SLE may occur at any stage in life; mostly affecting grown-up and pediatric partners and the mean age of determination went from 24-32 and 12-17 years, individually [9].

3. Clinical features

Important symptoms include facial rash (butterfly rash; characteristic of SLE), nasopharyngeal ulcers, alopecia, lymphadenopathy, nailfold infarcts, and noninfectious endocarditis. Non-specific symptoms include fever, malaise, fatigue, weight loss, pain with deformity in one or more joints, and photosensitivity.

3.1. Raynaud's phenomenon is common and is usually one of the first symptoms to appear along with arthritis and arthralgia. Digital ulceration can also occur as a result of the severe Raynaud phenomenon [1,10].

3.2. Joint involvement is another important feature of this complex disorder in which two or more joints can be involved. It encompasses joint deformity with or without pain, morning stiffness associated with arthralgia, and most specific for SLE is inflammation of synovial membranes or tenosynovitis.

3.3. Cutaneous manifestations: Exposure to sunlight or UV light predisposes to skin eruptions. Important skin involvements are;

i. **Classic facial rash**
ii. **Discoid rash**, diffuse, usually non-scarring alopecia (occur in active disease)
iii. **Urticarial eruptions**
iv. **Livedo reticularis** [10]

3.4. Hematologic: Neutropenia, lymphopenia, thrombocytopenia, and hemolytic anemia [10].

3.5. Non-scarring alopecia: In this disease, alopecia can be diffuse, nonscarring, patchy, non-scarring, lupus hair, and linear and annular lupus panoculitis of the scalp [11].

3.6. Renal: There may be proteinuria, hematuria, or RBC casts on urine microscopy, which if present, indicates proliferative glomerulonephritis [10].

3.7. Neuropsyciatric: Cerebral lupus presents as headache, fatigue, loss of concentration, and special characteristics of cerebral lupus (chorea, visual hallucinations, lymphocytic meningitis, transverse myelitis, and organic psychosis) [10].

3.8. CVS: Pericarditis, Libman-sacks endocarditis, and myocarditis can occur [10].

3.9. Lungs: Pulmonary symptoms may present as pleuritic pain or pleural effusion or atelectasis, lung volume is reduced, and breathlessness (due to pulmonary fibrosis) occurs [10].

4. Diagnostic criteria

The classification criteria for the diagnosis of SLE according to symptoms are provided in Table 1 [12].

Table 1
Modified classification criteria and symptoms of Systemic Lupus Erythematosus [12].

Criteria	Symptoms
Clinical	
Cutaneous lupus (acute, chronic)	Malar rash, photosensitivity Discoid rash (erythematous and raised edges)
Non scarring alopecia	Hair are thin, easily break and are sparse
Oral ulcers	Nasopharyngeal ulceration
Joint involvement	Non erosive inflammation of a synovial membrane
Serositis	Pleuritis, pericarditis
Renal disorders	Uninterrupted proteinuria (>0.5 g/24 hours) or red cell casts
Neurologic disorders	Seizures, myelitis, psychosis, and neuropathy
Hemolytic anemias	Hemolytic anemias
Lymphopenia or leukopenia	Leukopenia ($<4.0\times10^9$ cells/L) for more than 2 occasions Lymphopenia ($<1.5\times10^9$cells/L) for more than 2 occasions
Thrombocytopenia	Thrombocytopenia ($<100\times10^9$cells/L)
Immunologic criteria	
Antinuclear antibody (ANA)	Abnormal titer of ANA by immunofluorescence
Anti-dsDNA antibody	Abnormal titers
Anti-Smith antibody	Occurrence of antibody to Sm nuclear antigen
Antiphospholipid antibody	+ve findings of antiphospholipid antibodies
Low complement proteins	Low C3, C4 or CH50
Direct Coombs test	Anti-red cell antibody, in the absence of hemolytic anemia

5. Clinical manifestations in contrast to epidemiology

There is significant regional or racial variation in the prevalence of individual clinical indications of the disease. Certain trends have emerged among substantial heterogeneity, even within the fundamental ethnic gatherings. In other parts of the world, Europeans and their descendants generally demonstrate more mucocutaneous signs, particularly photosensitivity, than other ethnic groups [13-15]. The discoid rash affects SLE patients of African origin more often but typically

has less prevailing malar rash and photosensitivity stood out from the European region [14,16]. There are distinctive varieties in the appearance of SLE of changed Asian populaces with an increased rate of renal inclusion recorded in the majority of Chinese, Malaysians, and Indians; however, not in Pakistanis (33%) [17-20]. Patients with Filipino SLE were found to have renal inclusion more commonly (47%) and 30% of patients with a history of nephritic disorder or at the stage of finding the disease [21].

Practically, among versatile ethnic gatherings, lupus nephritis is fundamentally more surprising in SLE patients of European region. African identity is also among the most frequently perceived autonomous risk factors in multiethnic partners in the USA, for renal contribution and active nephritis progression [22-24], than in France [25] and the UK [26,27]. Hispanic ethnicity [22], as Pacific people or Maori ethnicity, is also separately related to renal disease [28]. After change in hypertension and diabetes, renal illness was not considerably more pervasive in African patients than in the European Canadians; however, all other complications happened more frequently in patients of African drop [29].

Indications of the focal and fringe sensory system influence an enormous number of patients with SLE significantly bleakness, and likely mortality. The most prevalent signs of SLE indicate neurological problems such as seizures and psychosis, and these are the most important side effects of neuropsychiatric (NP) SLE. The absence of normalized case definitions before the distribution of the ACR classification and case definitions for the NP lupus disorder in 1999 is because of a large part of this variability. Contrasted with Chinese Canadians (minimal rate), Africans and Americans had CNS involvement more commonly, yet the distinction with European Canadians was not measurably remarkable [29]. Indeed, the recurrence of NP appearance was fairly lower in African and Latin Americans compared to Mestizo and European Latin Americans [16]. The CNS damage estimations were not generously extraordinary for Chinese, European, and African Canadian SLE patients, while CNS contribution was more regular in African than in European Canadian [16]. The ratio of NP-damaged patients was comparable for Texan, Hispanics, European American, and African American, but slightly less for Puerto Rican Hispanic LUMINA cohort participants [29]. In all three ethnic gatherings, compared to Puerto Ricans, the period for NP to occur was limited. In the multivariate investigation, only Caucasian identity was held as an autonomous indicator of the time to NP harm. Other autonomous factors were old age, sickness with a history of infection, diabetes, and lack of ways to deal stress with SLE [29]. In the Maryland Lupus cohort, Caucasian ethnicity was an independent risk factor for NP damage; however, disease activity at the moment of the NP event was the strongest predictor [30]. Antiphospholipid antibodies (aPL) have appeared as an additional risk factor and several investigations have been archived in multiple examinations. In the Maryland test, financial variables were not critical, while destitution was connected to NP damage in the LUMINA accomplice, yet was not held in the multivariate examination [31].

6. Pathophysiology

6.1. Skin and Joints

Among SLE patients, skin involvement is believed to be up to 85% and is the first symptom in a quarter of cases [32-34]. The butterfly or malar rash on the nasolabial fold and photosensitivity are among the most frequently reported symptoms among SLE patients. In SLE patients, other familiar mucocutaneous symptoms include mouth ulcers and alopecia or hair loss. Three forms of hair loss can be identified, including localized diffuse alopecia, frizz or frontal hair loss, and extreme diffuse alopecia with limited signs of new hair growth [35]. Some of the dermatological manifestations reported are also discoid lesions (DL) and maculopapular lesions, splinter hemorrhages, nail base dilated capillaries, bullous lesions, angioneurotic oedema, and livedo reticularis [36].

The discoid lesion repeatedly leads to scarring, and pigmentary changes, either hypopigmentation or hyperpigmentation can result from older lesions. Alopecia scaring can occur in the scalp region in

relation to the discoid rash. SLE has also been associated with ulcers in the mouth, nasal, and genital regions, although they are less common. One of the mildly defined symptoms among SLE patients is Raynaud's phenomenon. Extreme digital ischemia and gangrene maturation may be associated with it. Raynaud's is a disease of the fingers and toes that affects blood vessels. Under cold or stress conditions, blood vessels will narrow around the area due to Raynaud's disease. Unless there is sparing of certain fingers [37], this feature is difficult to recognize among dark-skinned individuals. At the nailfolds and fingertips, vasculitis is commonly identified [35]. It can turn into a tender, profound, frank ulceration when it happens, it can either take months to heal or lead to secondary infections. Maculopapular rash is a type of rash that appears flat and red on the skin and is covered in SLE patients with small confluent bumps that can become infected. Skin injuries are part of lupus erythematosus (LE) with explicit and LE-vague indications dependent on clinical and histological measures [38-39]. Acute cutaneous lupus erythematosus (ACLE), which can appear as a butterfly rash or as a summarized maculopapular exanthema, is the most notable LE-express sign. Discoid lupus erythematosus (DLE) shows a steady course, normally set apart with follicular hyperkeratosis, and scarring provocative erythematous plaques. In bristly zones, it adds to unending alopecia. SLE can appear in around 5 percent of DLE patients who were less symptomatic with no foundational organ contribution. Subacute cutaneous lupus erythematosus (SCLE) is portrayed by polycyclic, annular, symmetric, and papulosquamous/psoriasiform, non-scarring skin injuries to sun-uncovered regions of the arms, chest, back and extensor surfaces. Vascular skin injuries (e.g., Raynaud syndrome, vivedo racemose, and periungual teleangiectasia) are among the LE-non-specific manifestations. In about 90% of patients with SLE, the musculoskeletal system is involved [40]. Arthritis of various joints can arise in addition to arthralgia and myalgia. It is possible to diagnose tendovaginitis and synovitis by imaging. Chronic recurrent arthritis can lead to joint deformation, especially of the finger joints, without radiological signs of erosion, and the resulting disorder is called Jaccoud Arthritis.

6.2. Kidneys

Around 50% of SLE patients develop renal associations [41]. Lupus nephritis is a glomerular nephritis usually associated with urinary sediment proteinuria, erythrocyte cylinders, and erythrocyturia. In a 24-hour urine sample, proteinuria may be quantified; a result of > 0.5 g per day is usually an indicator for renal biopsy. In 2003, the histological classification of lupus nephritis was revised to separate six types (class I-VI) requiring distinct therapy strategies [32,42].

6.2.1. Renal histologic discovery

Renal biopsies [43-44] are productive in most of the patients of lupus with atypical urine as they lessen the renal breaking point since it gives prognostic data and impacts the initial stages of treatment. The most remarkable area for lupus nephritis is its inconstancy between patients and inside biopsies, even inside glomeruli.

6.2.2. Glomerular appearances

Considering light microscopy, the World Health Organization (WHO) request for lupus nephritis [44] is commonly accepted, as it may be an ideal condition, allowing only a general degree of evidence [36]. A basic cause for inconvenience is focal proliferative nephritis (class III), since it encompasses a considerable broad range of appearances. Finally, the degree of patients expressed to each class in various plans from anywhere in the world is an excellent equivalence. WHO class III or IV (diffuse proliferative nephritis) genuinely structures that would be treated vivaciously by most of the clinicians (Fig. 1). In all courses of action, the degree of membranous biopsies (class V) is roughly comparable, i-e. 10-15% (Fig. 1).

IgG is always a general immunoglobulin in immunohistology, with IgG1 and IgG3 being widespread with some patients showing indistinct IgA and IgM in either case. For example, early enhancement fragments, C4 and particularly C1q, close to C3, are usually present. In addition to C3,

C4, and C1q, the discovery of motivation for each of the three isotypes of Ig is accessible in about one-fourth of lupus patients, and never in non-lupus disease [44]. Furthermore, in different patients, other insusceptible reactants, such as supplement sections B, C59, and b1H are present. Fibrin is phenomenally present in different classes in class IV biopsies [44].

6.2.3. Tubulointerstitial nephritis

Healthy totals are open in the adjusted storm cellar layer in about a segment of patients with nephritis, less with class II at any rate and up to 75% class IV. Direct barrel molded immunofluorescence was found in an abnormal patient, reminiscent of antibodies against TBM. Dynamic tubule passage and attack (tubulitis) is a large part of the time discovered in extraordinary pain. The interstitium is associated with a varying amount of collagen in the more consistent ailment. The outrageous tubulointerstitial nephritis in patients without glomerular tainting and can present as outstanding renal bafflement [45].

6.2.4. Intrarenal vessels

There are healthy vascular hyaline aggregates and no inflammatory necrotizing wounds, in fact monocyte and lymphocytic attack, vessel divider vasculitis, even more inconsistent intrarenal arteriolar thrombi [46]. These vascular changes suggest a defenseless awareness and it is important to see them in this way. In histologic and hematologic models, rare patients exhibit plain thrombotic microangiopathy. Relevant audiences have demonstrated associations between the involvement of antiphospholipid antibodies and the intraglomerular thrombus.

6.2.5. Other histologic components

Amyloidosis is exceptional in lupus, the unsurprising way amyloid A and C responsive protein assembly of extreme stage proteins, for example, no rise in plasma during lupus development flares. In abnormal patients, thick store disease and pauciimmune necrotizing glomerulitis have been reflected in lupus. A transition from dispersed proliferative glomerulonephritis (class IV) to an extraordinary membrane plan (class V) under productive treatment is especially common. Proteinuria can become enormous under these conditions as the renal limit improves [47].

Relatively, few subsequent evaluations of such patients have been performed; however, one evaluation found that the majority remained without clinical nephritis for a substantial period of time. In either case, it is almost clear that most of the patients with clinical apparent nephritis are likely to have undergone strangely affliction. Before this becomes self-evident, especially the number of nephritis patients in the beginning are less, it is unknown how many patients go for a subclinical course over the delayed time [47].

6.2.6. Clinico-pathologic relationships

Additional critical forms of glomerular nephritis appear to present severe clinical indications, histologically, it is not possible to predict renal histology with any certainty from the clinical images. The WHO biopsy class is an amazing determinant of results in untreated patients. In any event, this is not true at this stage if therapy that is more dynamic is provided to patients with more severe nephritis [48]. In each histological gathering, anti dsDNA neutralizer titers were comparable in patients, but others noted enormous contrast, although not clinically useful. The patient is also likely to have undergone some immunosuppressive treatment when a renal biopsy is done, which may alter serological outcomes [48-49].

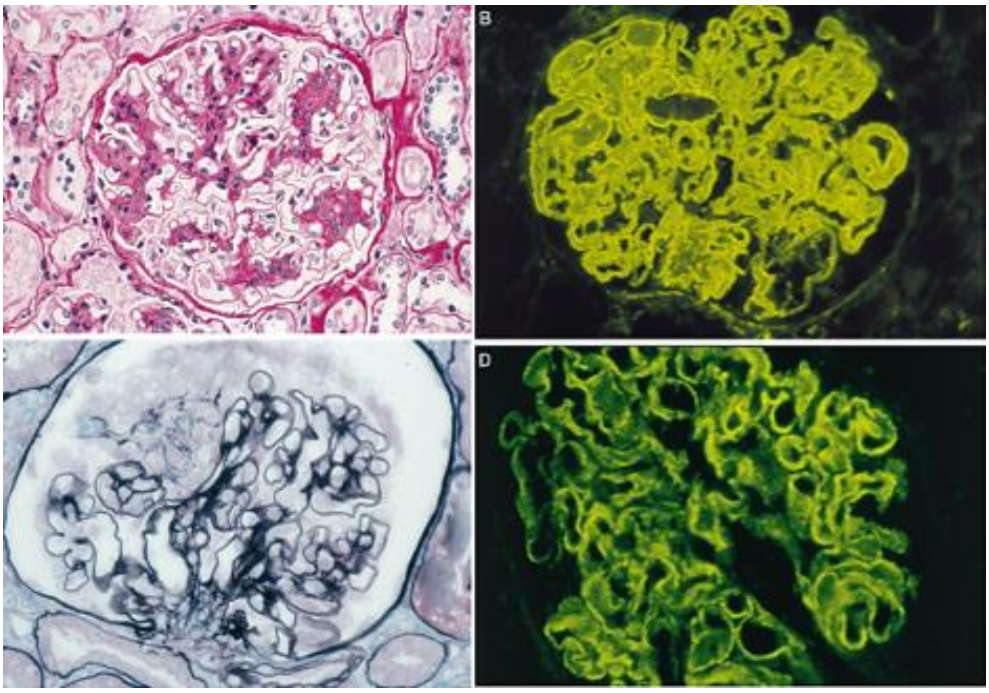


Fig. 1. Glomerular appearance. a) The appearance of patient with glomerulus and lupus nephritis (mesangial type); b) Immunomicrographs of glomerulus depicting the location of IgM in class II LN; c) Photomicrograph revealing the general appearance of class V dispersed membranous glomerulonephritis; d) Immunomicrograph depicting IgG localization in class V LN [47].

6.3. Pleural disease

Systemic lupus erythematosus may also affect the lungs and pleura, which appears to be life threatening. About 30% of patients have pleural effusion which is autoimmune pleuritis,hard to differentiate from other causes, parapneumonic effusion, pulmonary embolism (PE), cardiac disease, viral infections, renal, drug therapy, and tuberculosis [50,51]. Dry or wet, the most widely recognized kind of aspiratory association is pleurisy (40-60%); with less common lupus pneumonitis and aspiratory fibrosis [40].

6.3.1. Interstitial lung disease

In autoimmune rheumatic disease, interstitial lung disease is present but unusually between 1% and 15% of SLE patients [52]. Even if it is present , it has slow clinical progression [53,54].

6.3.2. Acute lupus pneumonitis

It is hard to diagnose the SLE presentation and the cause of acute lupus pneumonitis [55], with a frequency of 1% to 4% [53], and a high mortality rate in which the patient has pleuritic chest pain, cough, dyspnea, hypoxemia, and fever [26]. It can progress to chronic interstitial pneumonitis [56]. The nonspecific diagnosis raises the question of the existence of acute lupus pneumonitis [56].

6.3.3. Vascular involvement

SLE involves in vasculitis through one of two mechanisms. One is the deposition of immune complexes in the vessel wall causing the infiltration of neutrophils and complement activation [57]. Vasculitis will produce with or without antiphospholipid syndrome in the lungs followed by diffuse alveolar hemorrhage (DAH).

6.3.4. Other respiratory problems

About less than 1% of patients face shrinking of lung, a complication of SLE. About 0.5-17.5% of patients have pulmonary arterial hypertension (PAH) [58], 1-5.4% have vascular collagen disorder

[39], while acute reversible hypoxemia syndrome is considered to activate neutrophils which circulate, attach to cells and induce vasculopathy [57], venous thromboembolism, and lung cancer [56]. Biopsy of the lung featuring interlobular edema, lupus pneumonitis, and stiffness with marked vascular obstruction and fibroblastic cells, but no pathogens, viral inclusions, vasculitis, granulomata and pneumonia was noted [59].

6.4. Cardiovascular System

Libman-Sacks endocarditis, myocarditis, pericarditis, and coronary arteritis are the most frequently found SLE cardiovascular manifestations. The primary causes of SLE mortality are the early onset of arteriosclerosis and its related problems [60].

6.4.1. Libman-sacks Endocarditis

In SLE patients, Libman-Sacks vegetation are not unusual, specifically in patients suffering from SLE over the years, increased lupus activity, positive anti-cardiolipin antibodies, and manifestations of secondary antiphospholipid syndrome. Mitral regurgitation is the prevalent injury, while valvular lesion development occurs over a number of years in follow-up stage. Patients of SLE with Libman-Sacks endocarditis during the line of the disease, thrombotic episodes, especially stroke and acute ischemic attacks, appear to occur. As a result, increased understanding of this entity can cause careful cardiovascular evaluation, early prognosis, and effective actions [61].

6.5. Central Nervous System

Approximately, 15-50% of patients with SLE have imapired central nervous system, but their recognition as part of the disease is difficult sometimes because of their less specificity (e.g., headache) and increased variation in symptoms [40,62]. In addition to neuropsychiatric indications, for example, psychological shortage, psychosis, and melancholy, there may be natural mind conditions, epileptic seizures, and crossover myelitis, such as vascular occasions (for example, vasculitis, antiphospholipid antibodies, blood vessel/venous apoplexy) and fringe neuropathy [62-63].

7. Laboratory diagnosis

Research facility testing can validate the determination if the disease is considered dependent on clinical discovery. Initially, a research center test is recommended for screening [40-41,64-65]. For active SLE, a high rate of erythrocyte sedimentation is characteristic; the C-reactive protein is usually normal or just slightly increased. Cytopenias (thrombocytopenia and/lymphopenia and leukopenia) and more haematological changes like autoimmune hemolytic anemia, can be revealed in a normal blood count in the differential manner [63]. Creatinine, serum, urinary status, and sediment should be included in renal parameters. Indirect immunofluorescence experiments can analyze ANA.

It is beneficial to evaluate dsDNA antibodies in patients with positive artificial intelligence and homogeneous fluorescence design. Radioimmunoassays (RIA, otherwise called Farr examine; high particularity) upholds the after-effects of ELISA [32,66]. The Crithidia luciliae immunofluorescence test is an option in contrast to the RIA test, which is not accessible in all research centers due to its prerequisites of radioactive material. Despite dsDNA, and antibodies (70-98% predominance), hostile to Sm antibodies (14-40% pervasiveness) are SLE specific marker antibodies [33,66-67]. As markers of supplement use or deficiency, C3 and C4 must be calculated. The progression of SLE is marked by exacerbations and remission, but there is no connection between the AI A titer and the activity of the disease.

Anti-dsDNA antibodies, on the other hand, frequently increase several months before the onset of the disease, along with a decrease in complementary factors [68-69]. Therefore, disease activity, especially with regard to renal involvement, should be closely monitored.

8. Treatment

SLE treatment depends on the impact of only a couple of randomized controlled preliminaries. Likewise, with heterogeneous sickness frequency and low patient numbers, the incorporation models of these investigations are not normalized.

First-line treatment is hydroxychloroquine or chloroquine, however, acts as a starting non-steroidal drugs or potential glucocorticoids [70-72]. If no response or no long-term reduction of glucocorticoids by 7.5 mg is necessary, then the better options azathioprine or methotrexate or mycophenolate mofetil [73]. Autoantibody-positive SLE adjunct therapy with increased activity of disease among the standard care – belimumab [74-76]. Expert opinion indicates that not only low-dose prednisone, but also hydroxychloroquine and azathioprin can be administered during pregnancy (particularly in lupus nephritis [68,75]. The bioavailability of mycophenolate mofetil is decreased in the case of comedy with mycophenolate mofetil and proton pump inhibitors; it is advisable to turn to mycophenolate acid [75]. Proton pump inhibitors can decrease the effectiveness of hydroxychloroquine/chloroquine [76].

9. Survival and mortality rate

With an improvement in five-year endurance from around 50% during the 1950s to approximately 95% during the 2000s, the endurance of SLE patients has expanded in recent years [77]. The standardized mortality proportion (SMR) for SLE is 2.6-3.0 times higher than in everyone; this is likely identified with renal sickness (SMR = 4.7), higher rates of cardiovascular (SMR = 2.3) and infections (SMR = 5.0) [78-79]. Since the mid-1970s, SLE has recognized a bimodal mortality pattern in patients who pass on from the start throughout the illness because of dynamic sickness and additional contamination, while individuals who kick the bucket late over the span of infection are auxiliary to cardiovascular illness and also have an inert infection at the hour of their passing [80]. The factors associated with mortality are gender, ethnicity, etc., time of disease diagnosis [81-82] and damage caused by disease, particularly renal impairment, which has high mortality rate [82-83]. Neurological [84-85], hematologic disorders [85] including thrombocytopenia [86-87], hemolytic anemia [88-89], pulmonary [90], the antiphospholipid condition and comorbidities including coronary conduit sickness [91] and hemophagocytic syndrome [90] are the cause of high risk of mortality.

It is also recognized that the therapies used to control lupus affect mortality, which may mirror the gravity of the condition, yet may likewise mirror the medications themselves, for example, enormous portions of glucocorticoids [87] and immunosuppressive drugs [91], including cyclophosphamide. Antimalarials, then again, have appeared to broaden life [91-93], perhaps in a time-dependent way [84].

10. Conclusions

Systemic lupus erythematosus is an autoimmune disease in which cells and tissues of the body are destroyed by the own immune system. It usually manifests as multiorgan disease and genetic inheritance, female gender, young/childbearing age, and Asian ethnicity are more at risk for the disease. The common presentations in addition to facial rash are alopecia, oral ulcers, pericarditis, arthritis, anemia, thrombocytopenia, and there may be renal or pleural involvement such as; persistent proteinuria or pleuritic, respectively. In this review, we highlighted the disease and tried to focus on the diagnostic criteria and the few most common outcomes of this disease along with their histological and microstudies.

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Conflict of interest

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