REVIEW ARTICLE

Systemic Lupus Erythematosus and its Major Organ Complications: A Narrative Review on Available Literature

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Abstract

Systemic lupus erythematosus (SLE) is a complex chronic autoimmune disease that is well-recognized by its dermal manifestations, like skin lesions, rash, and photosensitivity. Symptoms may vary among individuals ranging from mild to severe because of its erratic pattern of remission and flare. SLE is a systemic disease that has the tendency to influence multiple organs of the body like lungs, brain, heart, and blood, *etc.* which is also one of the most important reasons for SLE-mediated mortality in young and old age groups, apart from renal complications and various infections. Because SLE is an autoimmune ailment, the formation of autoantibodies is considered to be the main cause of multiple organ system effects and systemic inflammation. The presence of hyperactive B cells produces autoantibodies in combination with the removal of apoptotic cellular material, resulting in immune complex formation. This leads to an inflammatory reaction in the microvasculature – causing multi-organ complications. In this review article, the main focus is on the complications of SLE among which renal disorder is one of the most life-threatening complications. Apart from this, cardiovascular, neurological, gastrointestinal and hepatic, muscular, osteoarticular and pregnancy complications have also been discussed. It has been concluded that timely identification and targeted therapy to manage these patients is the only solution.

Keywords: *Systemic Lupus Erythematosus, autoimmune disease, cutaneous manifestations, multi-systemic disease, Photosensitivity.*

INTRODUCTION

Systemic lupus erythematosus (SLE) is a multifaceted autoimmune disease ranging from mild to severe life-threatening ailment because of multiple manifestations and chronic relapsing-remitting course [1-4]. The clinical presentation of the disease is associated with a mixture of genetic predisposition, hormonal, environmental, and immunological aspects, with a consistent preference for females of the childbearing stage [5-8]. The disease progression of SLE is due to the formation and deposition of autoantibodies and immune complexes, eventually leading to organ damage [9, 10]. Majority of the SLE patients signify the skin and musculoskeletal signs as the initial and frequent complaints but it may affect any organ including skin, hematologic, kidney, neurological, respiratory, and/or cardiovascular systems [11, 12]. It may take months or years for all the manifestations to appear concurrently; consequently, heterogeneity is observed in the clinical appearance and pathogenesis of SLE, that's why it is a disease that is still problematic to explain [7, 13].

SLE is very widespread in North America (241/100000 persons (95% CI: 130, 352) with minimal prevalence in Northern Australia (0 cases among 847 people) [14, 15]. SLE being predominant in young women, the signs and symptoms are at their peak in the middle age of adulthood but in late ages in males. Additionally, the presence of black skin further increases the probability of the disease's presence in comparison to Caucasian ethnicity [12, 14]. Even after years of studies spent gathering deep insight into the disease, the information about the risk factors of SLE is imprecise [16]. It was proposed that programmed cell death (apoptosis) as the beginning of the disease progression [17] causes the cell fragments to be deposited at multiple organ sites with an enhanced B and T lymphocyte activity [18]. Furthermore, autoantibodies against nuclear antigens are also formed as a result of neutrophil-mediated cell death [19, 20]. SLE is also responsible for the development of antiphospholipid syndrome which is of concern in pregnant females that increases the chances of fetal loss [20]. The diagnostic workup is a combination of the clinical presentation along with laboratory investigations and tissue biopsy. The autoantibodies recognition is highly significant for making the correct diagnosis because of their high sensitivity; they include anti-nuclear antibody (ANA), anti-double-stranded DNA (anti-Ds-DNA) [21, 22] anti-Smith antibodies, anti-Sjögren's-syndrome-related antigen A (anti-SSA) and anti-Sjogren's syndrome B (anti-SSB) autoantibodies. Besides this, the identification of reduced levels of C3

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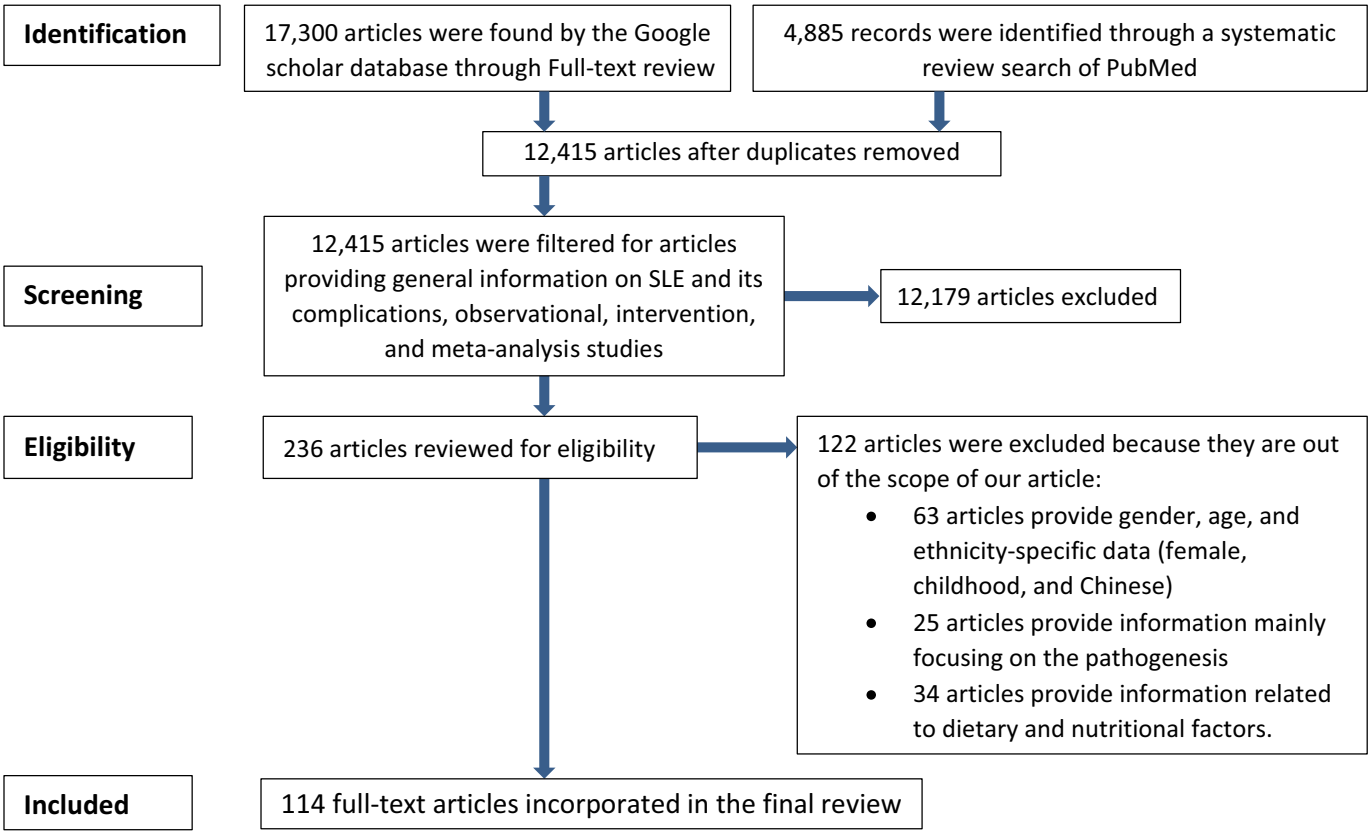

Fig. (1): Article selection flow diagram.

and C4 further aids in diagnosis. The crucial guide for the management of SLE is compliance with medications to achieve remission, patient counseling, and education concerning the pathogenesis of the disease and modifications in lifestyle [2].

METHODOLOGY

An electronic database search was performed on Pubmed. Search terms used were: systemic lupus erythematosus, complications of SLE, and management of SLE. Articles published in English were selected. All interventional, observational, and meta-analysis studies were included in which renal, cardiovascular, neurological, gastrointestinal, hepatic, muscular, osteoarticular, and pregnancy complications were reported in patients with SLE. All references were downloaded to EndNote X8 and duplicates were removed.

RESULTS

There were 17,300 articles identified in the search, of which 114 are included in the review. The complications category includes renal (13), cardiovascular (12), neurological (7), gastrointestinal and hepatic (21), muscular(4), osteoarticular(4), and pregnancy complications (18) articles (**Fig. 1**) (**Table 1**).

Table 1: Literature frequency of organ complication.

SLE-mediated multiorgan complications	No. of articles included
Renal	13
Cardiovascular	12
Neurological	7
Gastrointestinal and Hepatic	21
Muscular	4
Osteoarticular	4
Pregnancy	18

DISCUSSION

Epidemiology

SLE incidence and prevalence have vast differences as per demographics, socioeconomic factors, and according to some ethnicities like Asian and Hispanic populaces. Within similar contexts, people of African origin [9, 10], American Indians, and Alaska natives (particularly in Europe and North America) have greater tendencies and inferior outcomes from SLE than Caucasians. So, there are numerous signs for cases of European ancestry than Asian, African, and certain “Hispanic” or different aboriginal populations for having less severe SLE. It is to be noted that SLE among aboriginal/indigenous people is 2-4 fold more widespread in Australia, Canada, and the USA, as compared to non-aboriginal people. Additionally, cases of Asian and African origin as compared to white populaces are also expected to have a larger number of clinical indications, active SLE commencement, and greater mortality [23]. The assertions that SLE is infrequent in Africa can be due to the clinical technicalities and complexity of its diagnosis. Yet, evolving reports point out that the prevalence of SLE in sub-Saharan Africans is lower than in the Asian–

Pacific states *i.e.* 1.7% [24]. Across Asian–Pacific states, the overall incidence and prevalence of SLE went from 0.9 to 3.1 and 4.3–45.3 per 100,000, correspondingly. Also, SLE incidence in North America and Europe went from 3.7 to 49 and 1.5 and 7.4 per 100,000 person-years, correspondingly [25]. Data also implies that in North America, Europe, and Asia, there is a slow rise in SLE prevalence. Yet, there can be a variation in the proportion of the SLE disease population because of study design, reporting bias, case descriptions, and disease categorization criteria [26].

There have been articles reporting a prevalence of 72.1 to 74.4 per 100,000 individuals and incidence rates of 5.6 per 100,000 individual years in Caucasian and African-American populations, as per Georgia and Michigan lupus registries [27]. Nevertheless, African-Americans reported the highest rates of SLE [28].

An earlier onset age of the ailment is seen, which is more serious in African-American populations. SLE has a female: male ratio of 9: 1 and it mostly influences the females of childbearing age [29]. However, the risk declines in females after menopause, but it exists twice in comparison to males. Research has revealed that lupus in males is likely to be more serious, although rare. Moreover, males have a greater tendency than females for more frequent skin problems, renal disease, serositis, cytopenias, neurologic association, cardiovascular disease, hypertension, thrombosis, and vasculitis. Age is another important factor for SLE; while SLE is observed more in the childbearing stage of females, it has been well-stated for elderly and pediatric populations, too. SLE is reported to be more serious in pediatrics as compared to adults, having a greater incidence of malar rashes, pericarditis, nephritis, hematologic abnormalities, and hepatosplenomegaly. Nevertheless, it may have a more stealthy onset in the elderly with further pulmonary association and serositis and less Raynaud's, malar rash, nephritis, and neuropsychiatric problems [20, 30].

Pathogenesis

A complex interaction exists amid gene susceptibility, hormonal impacts, and environmental activators [31] with immune tolerance collapse, leading to autoantibody creation, following dysregulation of the inflammatory reaction, ensuing generation, and conservation of the ailment [32].

SLE Related Complications

The following figure defines the frequencies of organ involvement in SLE.

- **Renal Complications**

They are among the most severe complications of SLE [33-35]. Lupus nephritis may appear due to the deposition of the immune complexes in the kidneys [36]. For lupus nephritis, the universally accepted categorization of ISN RPS 2004 is as follows (**Table 2**).

Table 2: Lupus nephritis categories based on ISN RPS (2004).

Classes	Description
I	Marginal mesangial lupus nephritis, with typical glomeruli (*via* microscopic evaluation) but mesangial immune deposits on immunofluorescence
II	Mesangial proliferative nephritis, with mesangial hyperplasia (*via* optical microscope) but mesangial immune deposits on immunofluorescence
III	Focal proliferative nephritis comprising less than half of the glomeruli, with or without mesangial involvement. The subgroups are:
	IIIA (active lesions)
	IIIA/C (active and chronic lesions)
	IIIC (inactive lesions)
IV	Diffuse proliferative nephritis, comprising more than half of the glomeruli
V	Membranous nephritis
VI	Sclerotic nephritis without active lesions

Further uncommon types of lupus renal sickness are interstitial nephritis, drug-induced, and vascular illness and these may occur when the renal vessels are influenced [37, 38]. The factors assessed on a para-clinical basis include 24 hours of proteinuria with values more than 0.5 g per 24 hours; active urinary sediment with diffraction cell cylinders and red blood cells and; low creatinine clearance; serum creatinine more than lab's upper limit; low complement and high titer of double-stranded DNA antibodies [37]. Approximately 25% to 50% of lupus sufferers are usually found to have abnormalities of kidney function, initiating early or during disease development. The renal problems may be revealed in the first three years after SLE diagnosis [39, 40]. As per ethnicity, research has shown that renal damage with lupus nephritis is considerably worse in black individuals than in white ones [41, 42].

A critical part of the lupus nephritis structure is proteinuria. An exploration report on lupus nephritis demonstrated proteinuria in all of the cases, with 45% to 65% of cases having the nephrotic syndrome. Through the disease course, around 80% of patients exhibited microscopic hematuria [37]. Retrospective research, discovering the determinants of initial renal ailment in SLE cases, revealed that factors like young age, male gender, and non-European origin have too much effect on the ailment [43]. Regardless of therapy, end-stage renal failure is developed in about 10–15% of patients with lupus nephritis [37]. The characteristics that can predict end-stage renal disease in cases with severe lupus nephritis consist of greater baseline serum creatinine and inability to attain remission, through higher potassium levels, which may result in arrhythmic happenings. In this phase of disease progression, there is a higher anesthetic risk for such a patient for whom stringency perioperative management is necessary to avoid additional impediments [44].

- **Cardiovascular Complications**

In comparison to non-SLE patients, heart involvement encompasses many situations in SLE patients *e.g.* the

most common being pericarditis, valve ailments, coronary artery diseases (CADs) because of early atherosclerosis [9], and cardiac failure [45], carrying a greater mortality rate [46, 47]. Mortality from cardiovascular diseases has come to be more noticeable, though mortality from the disease activity has declined [48].

Cardiovascular association in SLE, compared to the general population, is the result of a combination of pathogenic mechanisms, leading to the progression of many cardiac events in young people [49]. Research published in 2003 had a sample populace of n=134 with similar age, ethnicity, and gender; 65 individuals with SLE having a mean age of 40.3 years, and 69 controls with a mean age of 42.7 years had no identified medical history of CADs. To check the presence of coronary artery calcification, electron beam computed tomography was employed and the Agatston score was used to evaluate the degree of calcification. The outcomes of this study exhibited that coronary-artery calcification was more in SLE cases (p=0.002) *i.e.* in 20 out of 65 patients, as compared to controls *i.e.* in 6 out of 69 individuals. Hence, the mean score of calcification for cases was 68.9±244.2 and for controls, it was 8.8±41.8 (p<0.001). This study established an increased prevalence of coronary artery atherosclerosis occurring at an earlier age in SLE cases [2, 50].

It is to be noted that the overall target for maintenance of blood pressure in SLE cases should be considered under 140/90 mm Hg because it can reduce vascular events [51]. Yet, if SLE patients have blood pressure >130/80 mm Hg with cardiovascular disease or increased estimated CVD risk (>10%), then they should be treated with a target of < 130/80 mm Hg [52, 53]. According to the study from the Taiwan National Health Insurance Research Database, on ensuing percutaneous coronary angioplasty (PCI), SLE was considered an independent predictor of in-hospital mortality and was independently associated with overall mortalities, repeat revascularisation, and major cardiovascular adverse events. Furthermore, this study highlights the necessity to increase care and apply secondary prevention tactics for these high-risk cases [54].

• Neurological Complications

SLE is generally linked with neurological problems according to studies [55]. The prevalence of neurological problems due to SLE ranges between 14 and 95%, with more common manifestations in children. Moreover, neurological problems can take place in the absenteeism of serologically active SLE and can be exhibiting symptoms in 39–50% of patients. A widely used nomenclature system for the neuropsychiatric syndromes linked with SLE was suggested by the American College of Rheumatology (ACR). The connection of the nervous system was noticed to be related to worse consequences and death rates reaching from 2 to 45% [55-57].

For the progression of neuropsychiatric SLE, the disturbance of the blood-brain barrier is considered to be significant. Headaches are not explicit or related to active disease, yet, they are a common neurological issue in SLE cases. Still, the existence of red flag signs should be assessed, signifying a subarachnoid haemorrhage, meningitis, and venous sinus thrombosis. Other common complaints include a decline in cognitive function, 'brain fog, depression, seizures, psychosis, mononeuritis multiplex, myelitis, peripheral and cranial neuropathy, and acute confusional state [32, 58].

To unify terminology, for use in clinical practice and research initiatives, the ACR also proposed a set of classifications for 19 NPSLE syndromes in 1999 [57]. These syndromes were divided into focal or diffuse associations, as well as central (there were a total of 12) and peripheral (there were a total of 7) [58]. This grouping showed diagnostic difficulty, mainly for the reason that this can existent, lacking anomalies in the characteristic markers of active lupus *i.e.* anti-dsDNA and complement [58].

• Gastrointestinal and Hepatic Complications

Gastrointestinal (GI) symptoms, though common in SLE, are generally minor. Such problems due to SLE can result from gut vasculitis, and if not diagnosed and treated, they may lead to increased morbidity [59]. A possibly fatal gastrointestinal complication is lupus enteritis, having varying symptoms from those of an acute abdomen to pseudo-obstruction or protein-losing enteropathy [60, 61]. GI symptoms can come about in around half of SLE patients, frequently activated by an underlying infection or by adverse effects of medication [62-64]. The most widespread GI symptoms are nonspecific, *e.g.* nausea and vomiting, anorexia, and abdominal discomfort [65]. A report from Alves *et al.*'s stated that inflammatory bowel disease can exist formerly or after SLE diagnosis, with a prevalence of ulcerative colitis (0.4–0.7%) and Crohn's disease (<0.4%); 0.3–2.4% of SLE patients suffer from primary biliary cirrhosis [62, 66]. In 3–10% of SLE patients, autoimmune hepatitis had been reported with a grander incidence in patients with juvenile SLE [11, 67, 68]. At a stage in the disease course, the liver may be affected in 19.4% to 60% of SLE patients out of which cirrhosis accounts for approximately 1-2% only [69-72]. Other autoimmune illnesses can co-occur with SLE *i.e.* secondary Sjögren's syndrome [73], hypothyroidism, antiphospholipid syndrome, overlapping syndromes with characteristics of rheumatoid arthritis, and scleroderma [32].

An underlying esophageal motility illness was found in about 20-70% of SLE patients. Esophageal motility complaints do not seem to be linked with SLE's duration, activity, and management. While the Raynaud phenomenon and the existence of antiribonucleoprotein antibodies have been associated with esophageal motility problems, it is uncertain if this relationship is due to the existence of other connective tissue illnesses. The

mechanism by which SLE gives rise to an esophageal motility disorder may be due to an inflammatory reaction in the esophageal muscles or due to ischemic or vasculitic changes to Auerbach's plexus [74, 75].

- **Muscular Complications**

Muscular pain may often come about in SLE patients with progression, frequently leading to a misdiagnose. As reported in several studies, the histologic modifications observed in muscle biopsies of SLE cases and myositis exhibited vasculitis, vacuolar myopathy, and necrosis. The associations between SLE disease activity and deposits of immune complexes in skeletal muscle biopsies were highlighted *i.e.* a great proportion of SLE cases were noted with myositis who had pathological changes in consistency with dermatomyositis and polymyositis [76, 77]. Pain is usually present in the scapular-humeral belt and indications like myalgia and muscle weakness are found often [78]. Lastly, franc myositis is related to the surge in levels of serum muscle enzymes, aminotransferases, or creatine kinase. In many cases, the pain is very severe requiring multimodal therapy [37].

- **Osteoarticular Complications**

Arthritis in SLE symmetrically affects the small joints of the hands and it resembles rheumatoid arthritis but arthritis of SLE is non-erosive. Jaccoud arthropathy is a specific type of SLE arthritis, with "swan neckline" deformations. Erosive arthritis arising in SLE is termed rhupus. In SLE cases, osteoporosis can often arise which is straightly connected with greater fracture risks [79]. There are complications in the surgical management of such fractures, particularly due to bone fragility, necessitating restricted periosteum stripping to avoid further bone injury [80]. Osteoporosis may be linked with the adverse effects of cortisone management in affected persons [37].

In SLE cases, there is a disagreement among diverse immune cell subsets like Th1/Th2 and Th17/regulatory T (Treg) cells with an uncommon expression of several proinflammatory cytokines, particularly IL-1, IL-6, and TNFα. This cytokine setup affects both osteoclast and osteoblast activities, boosting the expression of nuclear factor-Kb ligand (RANKL) by osteoblasts and osteocytes. Also, TNFα substantiates bone resorption indirectly in combination with IL-6, due to the upregulation of RANKL expression and directly by promoting the osteoclasts differentiation in association with RANKL. Therefore, there is a rise in RANKL production and a RANKL/osteoprotegerin (OPG) imbalance in SLE cases, leading to hurried osteoclastogenesis. IL-17 is another pro-inflammatory cytokine increasing bone resorption and several Th17 cells and elevated serum IL-17 levels are defined in SLE patients. IL-17 complements inconsistent RANKL/OPG by the expression of RANKL in osteoblasts or activated T cells and it could act in association with TNFα and other cytokines to affect osteoclast resorption [81].

- **Pregnancy Complications**

One of the most common immunological disorders related to pregnancy is SLE. A complex interaction exists between both SLE and pregnancy and they tend to influence each other. Literature is variable about the impact of pregnancy on SLE flares [82]. There is an association of greater risk of adverse pregnancy outcomes (APOs) with pregnant SLE patients, comprising of intrauterine growth restriction, fetal loss, prematurity, preeclampsia, and surge in mother illness and death [8, 83, 84], and neonatal lupus syndrome due to transplacental passage of autoantibodies [85-88].

APOs' clinical and laboratory forecasters in SLE females and mild or inactive illness were investigated in the PROMISSE research. PROMISSE study is a multicenter, prospective, multiethnic, observational cohort [89]. Greater clinical SLE disease activity at baseline, non-Caucasian ethnicity, the usage of antihypertensive drugs at baseline, existence of lupus anticoagulant and thrombocytopenia, as predictors of APOs were identified in a 2015 study of this populace [90-92]. Lately, PROMISSE researchers found a relationship between APOs and unusual triggering of the alternate complement pathway [93]. Tumor necrosis factor (TNF) was discovered to be a crucial intermediary between complement activation and foetal loss in a previous investigation with a mouse model of obstetric antiphospholipid syndrome (APS). In this paradigm, either TNF suppression or TNF shortage led to visible foetal protective effects [94]. Similar effects of complement activation were seen in women from the general population who developed preeclampsia between 10 and 20 weeks of pregnancy. Females with severe preeclampsia had complement split products in their amniotic fluid [95-97]. PROMISSE Complement activation indicators (Bb and sC5b-9) were found in blood among SLE/APS subjects who acquired APOs early in pregnancy, and they continued to rise through 31 weeks compared to those with normal outcomes in their cohort [85].

SLE Management

The main goal of therapy is to manage the activity of illness. Mild activity can be controlled by the use of non-steroidal anti-inflammatory drugs (NSAIDs) or low-dose steroids, however more severe indications demand timely therapy with moderate-to-high doses of steroids to reduce organ injury. Steroid-sparing immunosuppressive therapy should be deemed as soon as possible to avoid steroid-related illnesses [98].

Hydroxychloroquine is an efficacious medication in SLE, particularly for arthritis and rash. It has a protective impact in diminishing harm accrual in the long term and presents a survival benefit in SLE cases. The medication is well tolerated with rare ocular toxicity up on proper dosing [99].

Immunosuppressive medications like cyclophosphamide and mycophenolate for lupus nephritis have been used

as steroid-sparing agents in SLE, while azathioprine and methotrexate are used generally. Belimumab is a human monoclonal antibody that prevents B-cell activation by interference with a protein essential for B-cell activity. This monoclonal antibody has been newly granted by the Australian Therapeutic Goods Administration to treat moderately severe SLE [98].

More common measures to be considered in SLE cases involve cardiovascular risk decline and bone protection optimization. SLE cases are at substantially higher risk of premature atherosclerosis, thus smoking termination and management of hypertension, dyslipidemia, obesity, and hyperglycemia are greatly advised. Approaches to avoid osteoporosis should be deemed in most cases because many patients possibly need long-term glucocorticoid treatments [98].

It has been observed that approximately 10% of patients with lupus nephritis (LN) develop end-stage kidney disease. There can be a considerable risk of progressive irreversible damage buildup and a higher death rate due to insistent disease activity, comorbidities, and medication toxicity [1, 100, 101]. Sustained remission has been recently suggested as the eventual goal of SLE management which is rarely attained [102]. A broad range of medications are used for treatment owing to the organ system affected by SLE and the severity of tissue injury, the most common being hydroxychloroquine which permits the cases to attain the remission state [28, 103]; furthermore, as stated above, glucocorticoids and immune-suppressive therapies are employed often [20, 104].

The Lupus Low Disease Activity State (LLDAS) is evolving as a more realistic target state that pools both low SLE activity and a low prednisone (PDN) dose (≤ 7.5 mg daily) [105]. The LLDAS is related to a lesser danger of fresh injury buildup and better health-related quality of life (HRQoL) [106, 107]. So far, the goals of management for SLE patients have been modified over the past two decades. Earlier, patient survival was much focused but now, the focus is diverted to the decreased therapy-related adverse effects and organ injury, thereby significantly considering HRQoL [108]. Hence, it is vital to have worldwide control of this ailment with sufficient tolerability and safety of all existing management [7].

SLE and New Findings

Due to the multi-organ association, the use of both global and organ-specific, validated disease activity indices is essential to manage the disease and related outcomes for clinical trials. SLE Disease Activity Index (SLEDAI); British Isles Lupus Activity Group (BILAG) index and the Safety of Estrogens in Lupus Erythematosus National Assessment (SELENA)-SLEDAI Physician Global Assessment (PGA) are the three most widely used instruments [109]. Every index scores common signs and symptoms of disease activity in various organs, along with the SLEDAI scoring lupus serology, like anti-dsDNA and serum complement levels. The SLEDAI is weighted, while BILAG presents a comprehensive set of definitions for mild, moderate, and severe activity in different organs and as per the intention-to-treat concept (BILAG A includes the use of high-dose glucocorticoids and/or immunosuppressives). PGA should add to objective activity indices, as the latter can oversight some items of disease activity or lack sensitivity to longitudinal changes. Realistically, the SLEDAI-2K version of SLEDAI (which allows continued activity in alopecia, mucosal ulcers, rash, and proteinuria to be recorded) combined with PGA and the SELENA-SLEDAI definitions for flares has been generally used. Lately planned SLE Disease Activity Score (SLE-DAS; accessible at http://sle-das.eu/) with additional items comprising less common, yet severe appearances like myositis, haemolytic anaemia, cardiopulmonary and gastrointestinal indications, is considered to have better sensitivity to variations in comparison to the SLEDAI, with the upkeep of high specificity and simple for usage [110, 111].

Disease-related and treatment-related factors, both Are linked with the net risk of SLE infections. Vaccinations should be given to the patients according to the EULAR guidelines [112]. During stable disease, immunization against seasonal influenza and pneumococcal infection (both PCV13 and PPSV23) should be given more rapidly. Herpes zoster vaccination with the live vaccine (Zostavax) is available for the general population. Zostavax was reported to be well-tolerated in 90 stable SLE cases and triggered an immune response in those who were not given intensive immunosuppression [113]. SLE patients may have variable net immunosuppression, therefore if the infection is suspected, it should be managed since there is a more probable bacterial infection than a disease flare when C reactive protein is elevated [114]. There should be speedy detection and management of sepsis and authenticated scores like the quick Sepsis-related acute Organ Failure (SOFA) score identifies cases at greater risk for a meager outcome in the emergency or hospitalised cases, *via* scoring of three variables *i.e.* altered mental status, tachypnoea and hypotension [111].

CONCLUSION

SLE is an autoimmune ailment with loss of self-tolerance and polymorphic manifestations, representing a genuine defy in terms of its diagnosis and management. The disease frequently implicates serious complications in different body systems, thereby declining the quality of life of the affected persons. One of the most severe complications is renal impairment which may result in end-stage renal failure. Currently, the emphasis should be on timely diagnosis and targeted therapy for adequate management of such patients.

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CONFLICT OF INTEREST

None.

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