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Beyond The Classic: Unraveling the Diverse Clinical Manifestation of Systemic Lupus Ererythromatous

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ABSTRACT
Systemic Lupus Erythematosus (SLE) is a chronic autoimmune disease which may present with many atypical clinical features. This review explores variations in symptoms, challenges in making a diagnosis and treatment customization based on rare presentation of SLE titled "Beyond the Classic: Unravelling the Diverse Clinical Manifestations of Systemic Lupus Erythematosus". It begins by giving an overview about this uncommonly understood auto-immune disorder and its effects on different organs and tissues throughout the body. That being said, there are some common signs and symptoms which can be used as a platform to appreciate diverse nature of this illness such as constitutional problems like skin involvement joint pains among others.The main focus of this review article is therefore centered around strange or infrequent presentations of SLE. Neurological manifestations like seizures and cognitive dysfunctions challenge our traditional understanding about these diseases being limited only to joints or skin. Haematological features manifest themselves through clotting abnormalities together with various forms anaemia thus reflecting systemic character inherent in SLE. Dermatological findings also extend beyond what is typically referred to as butterfly rash; more so there exists numerous types with differing clinical appearances.The entanglement and variety of these expressions is illustrated by the use of case studies and examples in this investigation.A vital part of this exploration is about diagnosing diagnostic troubles in recognizing atypical SLE presentations. However, they have pointed out the disadvantages of relying solely on conventional criteria that highlights a broader clinical perspective as well as certain diagnostic criteria for unusual presentations. Recognizing genetic susceptibility may be complicated by environmental factors Knowing what triggers different clinical manifestations is important when considering causes.Treatment strategies are discussed here but adjusted for atypical manifestations. It has been brought out before that even though traditional methods used in treating joint and skin symptoms still apply there should be individualized therapeutic interventions. Unusual manifesting pathways can be targeted with biologic therapies that are emerging or other targeted treatments which work along specific abnormal pathways related to atypical manifestation management. The impact of various SLE manifestations on quality life among affected persons centers around one idea- Different forms come with diverse levels Psychosocial effects coping with less common symptoms not only physical cost but also mental effect. The talk emphasizes the need to implement a comprehensive approach towards patient care that takes into account the physiological as well as psychological needs. The review looks at new advances in research and technology that could change our understanding and treatment of different manifestations of SLE. The field of SLE is evolving, which creates opportunities for more accurate diagnosis and targeted treatments. These may involve better diagnostic tools and further investigation into genetic or environmental causes of the disease."Beyond the Classic: Unraveling the Diverse Clinical Manifestations of Systemic Lupus Erythematosus" ends with an in-depth exploration into many facets surrounding SLE. This evaluation brings out hidden signs by name so doing contributes to better understanding of what is known about this sickness while identifying it in different forms than usual ones . Therefore, paving way for enhanced accuracy during diagnosis; individualized plans for treatment among others which would improve general management skills among those who deal with such patients (SLE).

Keywords: Systemic Lupus Erythematosus, autoimmune disease, diverse clinical manifestations, atypical symptoms, neurologic manifestations, hematologic manifestations, dermatologic manifestations, diagnostic challenges, genetic predisposition, environmental triggers, tailored treatment strategies, personalized medicine, quality of life, psychosocial impact, emerging research, advanced diagnostics, targeted treatments.

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I. INTRODUCTION

Systemic Lupus Erythematosus (SLE) is a strong foe in the world of autoimmune disorders, distinguished by its varied influence on many organs and systems inside the body. This chronic autoimmune condition occurs when the immune system, which is normally the first line of defence against external threats, turns inward and attacks healthy tissues and organs. As a result, inflammation occurs, resulting in a variety of symptoms in the joints, skin, kidneys, heart, lungs, brain, and blood cells(BengtssonAA,R. 2017).

SLE can be a complex disease that has a wide range of clinical manifestations. Its start is influenced by a complex interaction between hereditary and environmental factors. SLE is thought to develop as a result of a confluence of environmental factors and genetic predisposition, while the precise origin is yet unknown. The illness primarily strikes women, frequently manifesting in the reproductive years, underscoring the significance of hormonal and gender-related variables(Arkema,S. 2017).

There's an extensive constellation of symptoms associated with SLE, from minor to severe. Frequent signs and symptoms include fever, exhaustion, rashes on the skin, and joint pain. But the fact that almost any organ system could be involved emphasises how varied it can be. SLE diagnosis is a complex procedure that frequently combines imaging examinations, blood tests detecting antibodies, and clinical criteria. To help in diagnosis, the American College of Rheumatology has developed classification criteria that include particular symptoms and lab results(BengtssonAA,R.2017).

A multimodal strategy customised for each case is required to manage SLE. Although there is no known cure, there are a number of therapeutic options that try to reduce organ damage, manage symptoms, and stop flare-ups. Antimalarial medicines and nonsteroidal anti-inflammatory drugs (NSAIDs) are frequently used to treat skin complaints and joint discomfort. For more severe inflammation, corticosteroids can be used, and they provide quick relief. Immunosuppressive medications and disease-modifying antirheumatic medications (DMARDs) constitute essential parts of the treatment regimen for those with severe or persistent SLE(Arkema,S.2017).

Treatment options have increased because to advancements in biologic medicines, such as monoclonal antibodies that target particular immune response pathways. With an increasing understanding of how genetic factors influence treatment response and susceptibility to specific consequences, personalised medicine is becoming more and more popular(ChaigneB,F. 2017).

It is essential to have regular medical monitoring in order to address changing symptoms and modify treatment plans as necessary. Beyond pharmaceutical interventions, lifestyle modifications such as sun protection and stress management are part of the management of systemic lupus flare-ups(ChaigneB,F.2017)

SLE patients requires continuous medical attention due to its chronic nature. Patients may communicate closely with nephrologists, rheumatologists, and other experts to treat organ involvement in particular. Even though SLE has no known cure, developments in the field and in available treatments help those who suffer from this difficult autoimmune disease live longer and with better projections(Gobeaux,D.2017)

In conclusion, systemic lupus erythematosus is a complicated autoimmune disease that requires extensive knowledge for successful treatment. Research on the disease is still ongoing and is helping us understand it better, which gives us hope for better treatment plans and better outcomes for people who suffer from this complex illness(FeldmanCH,L.2017)

1.1 Phathogenesis

Systemic Lupus Erythematosus (SLE) is a complicated aetiology that combines immunological, environmental, and genetic components. Here's a thorough rundown:

Genetic Elements:

Genetic predisposition: SLE is inherited. There is a genetic component that increases the likelihood of having SLE, namely immune system gene polymorphisms(Gobeaux,D. 2017)

HLA Complex: Susceptibility to SLE is associated with variations in the Human Leukocyte Antigen (HLA) complex, a collection of genes involved in immune system regulation(Gullstrand,L.2017).

Environmental Stressors: Infections: Epstein-Barr virus infections in particular have been linked to SLE susceptibility. Autoimmunity can arise as a result of immune system activation brought on by infections.(Gullstrand,L.2017)

Hormonal Factors: Changes in hormones, especially in women going through menopause,

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puberty, and pregnancy, might affect the causa of SLE.

An immune system dysregulation:

Loss of Self-Tolerance: Autoantibodies against the body's own cells and tissues are produced when the immune system in SLE is unable to discriminate between self and non-self(Kyttaris.2017).

B-Cell Hyperactivity: In SLE, B cells are essential. They generate autoantibodies that attack parts of the cell nucleus, such as anti-nuclear antibodies (ANA).

An imbalance of cytokines: Inflammation and tissue damage are two components of SLE that are caused by the dysregulation of cytokines, signalling molecules that control immune responses(Kyttaris.2017)

T cells' function:

CD4+ T Cells: The inflammatory response in SLE is influenced by the aberrant activation and function of CD4+ T cells, particularly T-helper cells.

Tolerance Breakdown: The autoimmune response is made worse by regulatory T cells' inability to control atypical immunological responses(Kyttaris.2017).

Activation of the Complement System:

Complement Cascade: In SLE, abnormal activation of the complement system, a component of the immune system involved in inflammation, contributes to tissue damage(BachenEA,2009).

Endothelial Dysfunction:

Vasculopathy: Endothelial cells lining blood arteries may be destroyed in SLE, resulting in vasculopathy and contributing to organ involvement(BachenEA,2009).

Organ Involvement:

SLE may affect various organs, including the skin, joints, kidneys, heart, lungs, and central nervous system. The precise processes of organ-specific injury differ(Kyttaris.2017)

Factor	Description
Genetic Factors	- Hereditary predisposition. -Variations in immune-related genes, particularly in the HLA complex.
Enviromental triggers	-Viral infections, notably Epstein-Barr virus. - Hormonal changes, especially in women duringpuberty,pregnancy,and menopause.
Immunological Dysregulation	- Loss of self-tolerance, leading to the production of autoantibodies. -B-cellhyperactivity,producing autoantibodies like anti-nuclear antibodies (ANA). - Dysregulation of cytokines contributing to inflammation and tissue damage.
Role of T Cells	- Abnormal activation and function of CD4+ T cells, especially T-helper cells. - Breakdown of immune tolerance due to regulatory T cell dysfunction.
Compliment system Activation	- Abnormal activation of the complement system, contributing to tissue damage.
Endothelical Dysfunction	-Vasculopathy due to damage to endothelial cells lining blood vessels.
Organ invlovement	- Multi-organ impact, affecting the skin, joints, kidneys, heart, lungs, and CNS.

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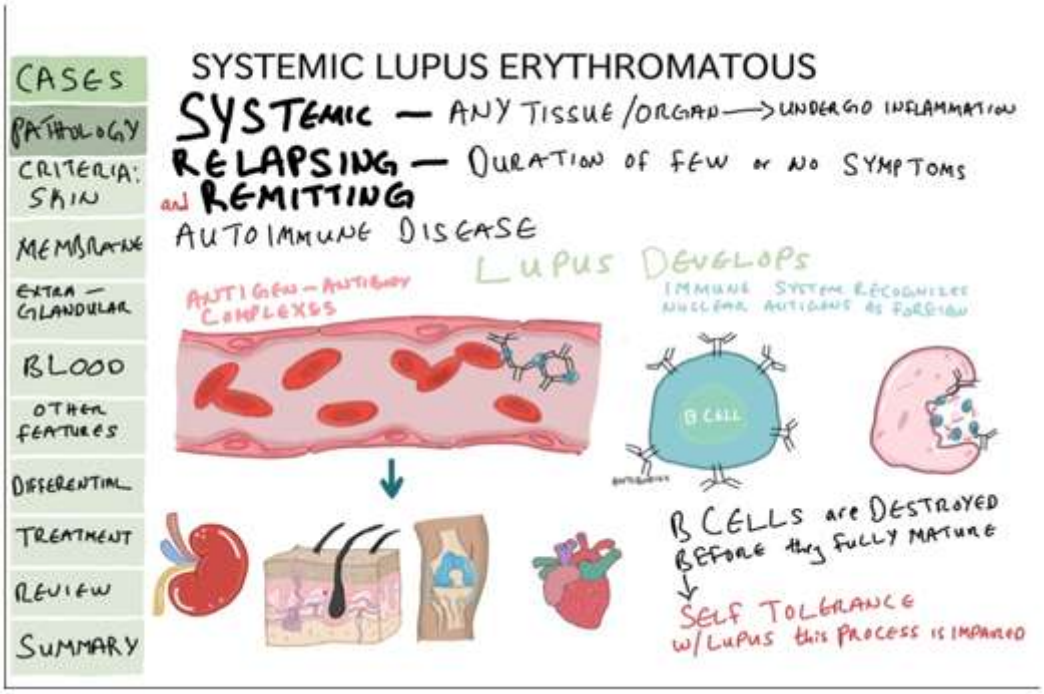

Figure: Pathophysiology of systemic luouserythromatosus

1.2 Diagnostic Criteria:

To diagnose systemic lupus erythematosus (SLE), a combination of laboratory experiments and clinical examples are usually used. The updated 2019 American College of Erdology (ACR) guidelines is the most preferred method of diagnosing SLE. SLE can often be diagnosed when an individual meets any four of the following criteria:(KesslerRC,2003)

Malar Rash: Fixed erythema, flat or raised, over the malar eminences, tending to spare the nasolabial folds(SeawellAH,2004)

Discoid Rash: Erythematous raised patches with adherent keratotic scaling and follicular plugging; atrophic scarring may occur in older lesions(SeawellAH,2004)

Photosensitivity: Skin rash as a result of unusual reaction to sunlight, by patient history or physician observation(SeawellAH,2004).

Oral ulcers: Oral or nasopharyngeal ulcerations, usually painless, observed by a physician(SeawellAH,2004).

Arthritis: Nonerosive arthritis involving two or more peripheral joints, characterized by tenderness, swelling, or effusion(ShihM,2006).

Serositis: Pleuritis or pericarditis documented by ECG or rub or evidence of effusion(ShihM,2006).

Renal disorder: Persistent proteinuria >0.5 g/day (or >3+ if quantitation not performed) OR cellular casts — may be red cell, hemoglobin, granular, tubular epithelial, or mixed(ShihM,2006).

Neurologic disorder: Seizures — in the absence of offending drugs or known metabolic derangements; psychosis — in the absence of offending drugs or known metabolic derangements(ShihM,2006).

Hematologic disorder:

a. Hemolytic anemia— with reticulocytosis OR

b. Leukopenia <4k/mm^3 on ≥2 occasions OR

c. Lymphopenia <1.5k/mm^3 on ≥2 occasions OR

d. Thrombocytopenia <100k/mm^3 in the absence of offending drugs.

Immunologic disorder:

a. Antibody to dsDNA OR

b. Antibody to Sm nuclear antigen OR

c. Positive finding of antiphospholipid antibodies based on (1) an abnormal serum level of IgG or IgM anticardiolipin antibodies, (2) a positive test result for lupus anticoagulant using a standard method, or (3) a false-positive serologic test for syphilis known to be positive for at least 6 months and confirmed by Treponema pallidum immobilization or fluorescent treponemal antibody absorption test(ShihM,2006).

A positive antinuclear antigen test shows increased quantity of antigens which combine with cell nucleus(Enocsson,W.2017).

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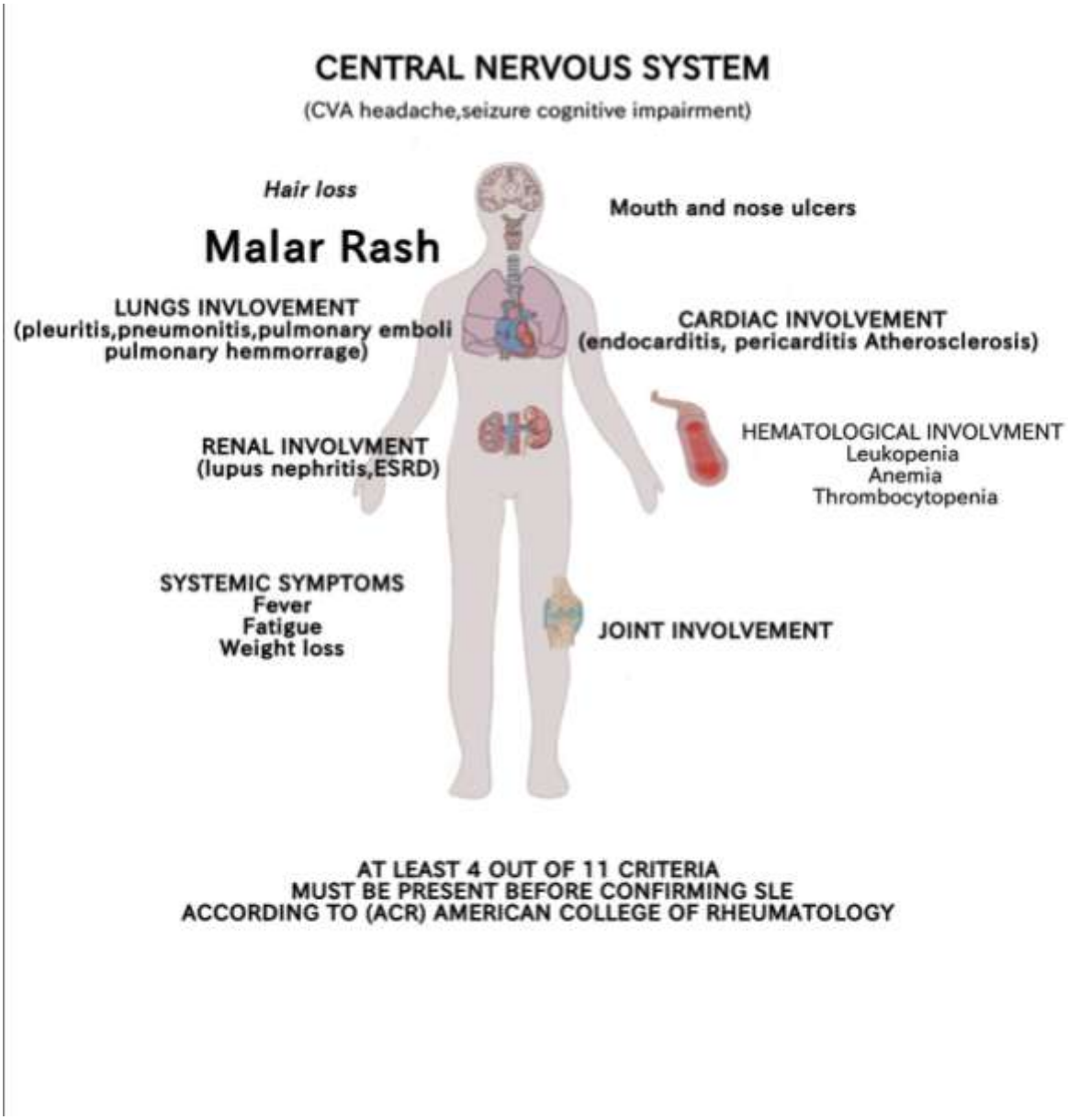
It is important to remember that these guidelines are followed strictly according to the clinical doctrine of a doctor, not all requirements need to be met at once and there may be some odd or overlapping symptoms displayed by people. Besides, it may take a lot of effort and time before getting accurate diagnosis for SLE(Enocsson,W.2017).



1.3 Biomarkers

An intriguing aid appears to come from the traditional biomarkers before tackling the new emergent ones. In fact, it has been observed that SLE patients typically have lower platelet activation; in addition, the decreased platelet volume significantly correlates with anticardiolopin antibodies (Gullstrand, 2017). A cross-sectional, longitudinal, and predictive analysis was conducted to investigate the clinical value of anti.sm antibodies in the diagnosis and monitoring of SLE. The results showed that 14.8% of anti.ds DNA negative patients had anti.sm positivity, and 51.4% of anti.ds DNA positive patients had anti.sm positivity as well. Although the longitudinal and

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predictive study found no link with lupus activity(McKinleyPS,1995).

In SLE patients who have not yet begun treatment, hypovitaminosis D is more common than in healthy controls (38.6% vs. 4.8%), according to reports on the measurement of vitamin D status in these individuals. A greater ANA titer and high ANA titer have been linked to treatment-naive SLE with hypovitaminosis D (Shahnin D. El-Farahaty RM, 2017).cardiovascular disease is one of the leading causes of death in SLE patients, yet the Framingham score frequently understates this population's CVD risk. According to a cross-sectional controlled investigation, there is a connection between elevated levels of the cardiac protein high sensitivity cardiac troponin T (HS-cTnT) and carotid plaques (assessed by ultrasound). Accordingly, high serum HS-cTnT levels are linked to carotid plaques in SLE patients who, based on the Framingham score, appear to have a low risk of CVD(Gobeaux, 2017).As was already said, one of the most popular applications of biomarkers is their capacity to forecast disease activity. For instance, a study revealed that serum osteopontin levels, a protein of the extracellular matrix with immunomodulatory capabilities, are typically increased fourfold. p0.0001 when comparing SLE cases to controls. Osteopontin corresponds with disease activity in cases of recent onset, according to the date. SLE is a reflection of related syndrome and overall organ damage (Enocsson, 2017).

SLE patients have been reported to have considerably higher serum and urine levels of IFNgamma chemokine (CX-L Motif) Ligand 16 (CXCL-16) and soluble urokinase-type-

plasminogen activator receptor (SUPAR) than healthy controls between those markers. The link between SUPAR and disease activity was stronger(WardMM,1999).

1.4 Neurological Involvement:

Patients with SLE exhibit a widerange of central nervous system (CNS) and peripheral nervous system (PNS) neuropsychiatric characteristics. other psychiatric illnesses. Polyneuropathy was the first PNS manifestation in SLE patients, followed by non-compression mononeuropathy, cranial neuropathy, myasthenia gravis, and Gultain-Barre syndrome (Pinto, 2017). SLE is a demonstrated independent positive predictor of epilepsy after accounting for several confounding factors (age, sex and socioeconomic status). Epilepsy was more prevalent in SLE patients than in the control group (40.3% vs 0.87%) (S.BRAGAZZI W. , 2018).

A Swedish study found that SLE patients had a relative risk of ischemic stroke that was more than twice as high as that of the general population. The high risk group included females and adults over the age of 50. The highest significant relative risk was noted within the first year following the diagnosis of SLE and remained largely stable up to another 11 years of follow-up. When compared to healthy controls, SLE patients' mean age at stroke was lower (68.42% vs.
73.3%) (Arkema S. , 2017).

1.5 Renal involvement:

A retrospective cohort analysis with a 20-year time span that included 249 SLE patients with renal involvement was recently conducted (proved by renal biopsy). Hypertension, nephrotic syndrome, and renal failure were among the signs of renal flare in these individuals, accounting for 40%, 30%, and 69.4% of the cases, respectively(Da CostaD,1999).

1.6 Cardiovascular and pulmonary involvement

The pericardium, myocardium, valvular tissue, and coronary arteries may all be involved in the heart in SLE. A serious ailment, lupus myocarditis. That might happen as the condition progresses. Recent research suggests that patients with SLE may develop myocarditis more easily due to the lack of particular treatment. Luckily, the outlook for this manifestation over the long term is generally favorable (Thomas G C. , 2017).

Hematological parameters

The clinical spectrum of the disease may already show hematological involvement at the time of diagnosis, or it may appear later or be brought on by treatment. SLE has been discovered to be independently linked to a larger percentage of malignancies, especially hematologic ones. NonHodgkin lymphoma was the most prevalent, followed by Hodgkin lymphoma and multiple myeloma(S.Watada, 2017).

(Bachen EA,2009)**,** Demographic and clinical characteristics of the sample are shown in Table 1. Subjects were diagnosed with SLE an average of 15 years prior to study participation. At the time of the interview, 46% reported using psychotropic medications, the most common of which were antidepressants (41%). The mean SLAQ score for the sample was 14.0+/−7.6 and ranged from 0 to 35, reflecting a wide range of self-reported disease activity in SLE. Twenty-six percent of the sample

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had a history of renal involvement, which is consistent with other Caucasian SLE cohorts (35).

Demographic and clinical characteristics of 326 SLE study subjects(KesslerRC,1998).

Characteristic	Mean (SD) or N (Proportion)
Age, mean (SD), yr	47.9 (11.3), range 18–83
Education level, N (%)	
High school/GED or less	136 (41.7)
Associate degree	54 (16.6)
College degree	89 (27.3)
Master's degree or higher	47 (14.5)
Marital status, N (%)	
Single	104 (31.9)
Married	222 (68.1)
Working outside the home, N (%)	
Yes	131 (43.7)
No	169 (56.3)Yes
	131 (45.5)
$50,000 or higher	157 (54.5)
Age at SLE diagnosis, mean (SD), yr	32.5 (12.2), range 1–73
SLE duration, mean (SD), yr	15.4 (9.7), range 1–47
SLE medications*, N (%)	
Non-steroidal anti-inflammatory drugs	185 (56.7)
Prednisone	136 (41.7)
Hydroxychloroquine	169 (51.8)
Methotrexate	29 (8.9)
Other disease-modifying antirheumatic drugs	57 (17.5)
History of renal involvement**, N (%)	
Yes	83 (25.5)
No	239 (74.2)

GED = General Equivalency Diploma.
Current SLE medications at time of interview.

History of renal involvement was defined as meeting ACR renal criterion or renal biopsy consistent with lupus nephritis.(Bachen EA,2009)

1.7 Camorbidities

Since understanding of the SLE's pathogenic process has grown Many immunosuppressive medications are now frequently utilized in clinical settings, and infections are becoming one of the leading causes of death. The combined effects of the immunological treatments, particularly in situations of high disease activity and LN, are likely to be to blame for the increased risk of infections in this population. According to Chen et al analysis.'s of the adverse events linked to long-term glucocorticoid usage in 11288 Chinese SLE patients, greater glucocorticoid doses were linked to a higher incidence of bacterial infections in addition to other non-infectious problems. In few recent articles, hospital-acquired infections in SLE were the main topic. 3956 Chinese SLE patients with hospital-acquired infections participated in a case-control study, which was discovered that bloodstream infections (10.9%) and the respiratory tract (58.8%) were the most frequently affected. The majority of episodes (50%) were caused by bacteria, followed by viral (34.8%) and fungal infections (15.2%) infections. Hospital acquired infections were more likely to occur in patients with an SLE DAI score, LN high dose of GC, and CYC medication(NymbergJH,1994).

According to a cross-sectional population-based study that included 5018 SLE patients and 25090 controls, the diagnosis of SLE was independently associated with increased proportions of malignancies, particularly hematological diseases but also cervix, uterine cancer, and genital organ cancers(GrantBF,2004).

Two recent large cohort studies have demonstrated a substantial increase in the incidence of cervical cancer in SLE patients taking

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immunosuppressing medications as compared to those receiving antimalarial medication (Feldman CH, 2017).

Therapy:

Baff levels have repeatedly been reported to be greater following B-cell depletion using anti CD20 methods, and they have also been reported to be higher in SLE patients who relapse after receiving rituximab as compared to those who maintain illness remission. A possible therapy approach for increases in BAFF levels brought on by the reduction of auto-reactive B cells in B cells is sequential treatment with RTX followed by belimumab. New biologic medicines that target B cells have emerged as a result of the prominent role that B cells play in the pathology of SLE. Eprahizumab, a humanized mAb that targets CD22 ON B cells, is one of them(BrownTA,1992).

Bortezomib, a hematological medication licensed for multiple myeloma and lymphoma, is one of the new B-cell depleting drugs. It is a proteasome inhibitor that has the ability to suppress antibody secretion is caused by plasma cell processes.

Roy-ByrnePP, report that Bortezomib and GC were used to treat 5 cases of SLE patients with refractory LN, with positive outcomes for renal function, proteinuria, immunological parameters, and minimal adverse effects (Roy-ByrnePP, 1999).

In summary, belimumab, an anti-Blys agent having a limited impact on disease activity, is the only biological medication currently licensed for the treatment of SLE. Despite early encouraging results, other B-cell targeted treatments failed to demonstrate efficacy in significant phase III trials (Kyttaris, 2017).

Inhibitors of IFN-I

It is well recognized that SLE is a specific autoimmune condition that is IFN-I mediated. Serum IFN-I levels were shown to be higher in SLE patients who were older than 30 and were linked to disease activity (Psarras A, 2017).In contrast to healthy people, the majority of SLE patients exhibit a prolonged activation of the IFN-I system, which is caused by an overexpression of type I IFN-regulated genes or an IFN-signature. The course of autoimmune illness is significantly influenced by the constant production of IFN-1, which stimulates both the innate and adaptive immune systems. High dosages of GC and Hydroxychloroquine are two examples of common treatments for SLE that downregulate IFN-signature. Hydroxychloroquine, in particular, appears to affect IFN-1 via preventing TLR7 and TLR9 activation (Bengtsson AA, 2017).

II. QUALITY OF LIFE AND PATIENT REPORTED OUTCOMES & TREATMENT

SLE is a chronic, unpredictable illness that has a big influence on patients' day-to-day lives.

Although the prognosis for SLE as a whole has improved, patients' quality of life has not. Patients with SLE typically have lower health-related quality of life than the general population(IshikuraR,2001).

In a recent study by Chaigne et al., the medical outcomes study short form was used to assess the health-related quality of life in patients with SLE and rheumatic diseases who were matched by age, sex, and disease duration (SF-36). Rheumatoid disease patients had lower physical component summary scores than SLE patients, and these differences persisted even after adjusting for patient characteristics, treatment, and disease activity, as well as after more than a year of follow-up. Accordingly, the findings imply that SLE and rheumatoid arthritis have fundamentally different effects on quality of life (Chaigne B, 2017).Another example illustrates two Italian studies that shown how significantly musculoskeletal involvement affects a patient's day-to-day activities. According to research by Piga et al., fibromyalgia, jaccouds deformities, and active arthritis are all linked to lower quality of life(ZorillaEP,2001). Also, it was discovered that active arthritis, deformities, and fragility fractures adversely impacted the HAQ's assessment of impairment perception(Piga M, 2018) .Similar to this, Tani et al. investigated the effect of joint involvement in a cohort of 50 consecutive SLE patients and discovered a significant correlation between the presence of arthritis (established by clinical and ultrasound evaluation) and patients' VAS scores for pain, patients' perception of disease activity, and global health(Zandman-GoddardG,2007)

III. CONCLUSION:

Finally, this investigation into the many clinical presentations of Systemic Lupus Erythematosus (SLE) has revealed a rich tapestry of intricacies that extend beyond the conventional symptoms. A more comprehensive view of SLE arises by scuba diving into less-explored components such as renal involvement,

neuropsychiatric symptoms, cardiovascular obstacles, haematological aberrations, dermatological problems, gastrointestinal challenges, and respiratory realities(McCartyDJ,1995).

The complexities of lupus nephritis emphasise the need of monitoring renal function in SLE patients and understanding the influence on overall well-being. Neuropsychiatric symptoms ranging from cognitive impairment to seizures highlight the importance of thorough examinations, given their severe effect on patients' quality of life.

The increased risk of cardiovascular problems in SLE emphasises the significance of comprehensive treatment, including consideration of the heart and blood arteries. Haematological abnormalities, such as autoimmune cytopenias, provide another degree of complexity to SLE, demanding specialised treatment methods.

Exploring cutaneous signs other than the typical butterfly rash emphasises the diagnostic importance of skin manifestations. Furthermore, the less-explored gastrointestinal and respiratory issues add to the multidimensional character of SLE, necessitating increased clinical awareness.

Navigating these many clinical landscapes reveals that SLE is a dynamic autoimmune illness with systemic consequences. The difficulties in diagnosing and managing these many forms underline the importance of continuing research and a multidisciplinary approach in clinical practice.

In a nutshell this study argues for a paradigm change in our knowledge of SLE, acknowledging that its effects go well beyond the traditional triad of symptoms. We enable medical professionals, researchers, and SLE patients to promote better diagnostic accuracy, individualised treatment plans, and ultimately improve overall care and outcomes for those navigating the complexities of this autoimmune disorder by embracing the nuances of these varied clinical manifestations(RioloSA,2005).

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