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Systemic Lupus Erythematosus: Historical Perspective, Global Epidemiology, Immunological Dysregulation, and Multifactorial Etiology

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الذئبة الحمراء الجهازية: منظور تاريخي، وبائيات عالمية، واضطراب مناعي، وأسباب متعددة العوامل

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ABSTRACT

Background:

Systemic lupus erythematosus (SLE) is a multisystemic autoimmune inflammatory disease, that is chronic and complex. Its pathogenesis is a complex of intrinsic factors and environmental stimuli, hormonal effects, and epigenetic processes, and it causes a severe deregulation of the immune system. The main symptoms of the disease is an abnormal immune response, where autoregulated T cells trigger B cells to produce auto pathogenic autoantibodies. The clinical presentation of SLE is so diverse, covering mild and self-limiting manifestations, such as fatigue, skin rash, and joint pain, to severe and life-threatening organ involvement, which may include lupus nephritis, neurological problems and heart diseases. The disease can either attack one organ or manifest itself as a systemic disorder with more than one organ at a time. This broad range of clinical and serological heterogeneity is one of the distinguishing features of SLE and creates a great difficulty in the diagnosis of the disease, its monitoring, and treatment .It is crucial to gain a complete picture of the immunopathological processes and heterogeneity of SLE to enhance the accuracy of the diagnostic process, at the same time optimizing the personalized approach to treatment, and minimizing morbidity and mortality in the course of long-term follow-up of the disease.

Key word: Systemic Lupus History, Epidemiology, Immunological, Etiology.

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INTRODUCTION

Systemic lupus erythematosus (SLE) is an autoimmune disease of a chronic and multisystemic nature involving inappropriate immune reactions and chronic inflammation that may result in a progressive destruction of tissues and organs (1). The loss of self-tolerance is a typical immunological characteristic of SLE that leads to the generation of a wide range of autoantibodies, specifically nuclear antigens, and immune complexes that cause inflammatory damage in various tissues (2). It is known that SLE is a highly heterogeneous disorder clinically with patients presenting with a great range of manifestations and disease pathways, from mild cutaneous and musculoskeletal disease up to severe and fatal organ disease (2).SLE may involve a large number of different organs and systems. According to study carried out in (2021), SLE may affect the skin, joints, kidneys, lungs, nervous system, serous membranes, and other organs, which is a symptom of the systems-wide character of immune-mediated inflammation and the presence of immune-complexes (3). Although much is known about SLE, its etiology has not been fully understood yet. Existing data favors a multifactorial approach that is based on genetic susceptibility interacting with environmental and hormonal factors to elicit autoimmunity and clinical disease (4,5). A number of environmental causes have been attributed such as ultraviolet (UV) radiation exposure, some of the medications that are demethylating effects, infectious agents (endogenous or exogenous viruses), exposure to antibiotics and frequent infections (4,5). It is also believed that hormonal factors, especially those associated with the sex hormones, are also factors that make a person susceptible and active in diseases, and this may be one of the reasons why females are far more susceptible to SLE (4,5).

Musculoskeletal involvement and lupus arthritis

Among the most common and clinically notable features of SLE are musculoskeletal, which are all covered in all major classification criteria that have been offered in the past four decades (5,6). Lupus arthritis is known to be an early manifestation and may also be the initial manifestation and may appear at any point in the course of the disease (5,6). According to the new EULAR/ACR classification criteria, arthritis is considered synovitis, which involves two or more joints, where pain is present in at least two joints, there is objective evidence of swelling or effusion, and it is accompanied by at least 30 minutes of morning stiffness (7). It is this

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definition that brings out the significance of inflammatory joint disease as a diagnostic and classificatory component of SLE. Musculoskeletal signs are a critical part of SLE disease activity indexes and are commonly employed to determine the burden of disease and response to treatment (7). Though arthralgia and arthritis in the case of SLE are typically intermittent and may disappear with proper treatment, the symptoms may mimic the signs and symptoms of rheumatoid arthritis (RA), especially when they become chronic or recurrent. Patients can be affected by chronic joint pain, stiffness, swelling, and functional limitation, in some cases and limited range of movement and loss of everyday activity. The most affected joints are the hands, wrists, and knees, but in some patients, the symptoms may affect other joints and patterns may be different (5). Moreover, it is not rare that the inflammatory activity of the periarticular structures takes place; inflammation in tendons and synovium can be present and can significantly cause pain and disability (5). Notably, although most of them are non-erosive, a long period of inflammation may still result in deformities or impaired functioning, underlining the necessity of early diagnosis and successful management of the disease.

Historical perspective of SLE

The evolution of the concept of SLE is associated with the observation of the slow transformation of lupus into a systemic illness and not just a cutaneous disorder. One of the earliest doctors to discover that internal organs could be affected by lupus erythematosus was Moriz Kaposi (18371920), who coined the term systemic lupus erythematosus as asystemic disease as opposed to lupus confined to the skin (LE) (8). His findings supported the groundbreaking knowledge that SLE is defined by the internal organ involvement, and cutaneous types of lupus can manifest mostly with the skin symptoms. Subsequently, Sir William Osler (18491919) developed clinical observations of diseases involving the systemic involvement, such as those of the kidney now considered lupus nephritis. His reports implied that severe kidney disease may be temporally associated with systemic illness and this helped in increasing the value of nephritis as a significant complication and contributor to morbidity and mortality in SLE (9). In the past, it used to be thought that infections might be the major cause of mortality in lupus, however, later developments made it plain that renal activity- especially severe lupus nephritis- may be the focus in prognosis and early mortality (9). However, the magnitude of Osler has occasionally been

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exaggerated, with subsequent reviews pointing out that of the cases that he characterized between 1895 and 1904, only a few were obviously typical of what is today known as SLE, with other conditions (including HenochSchonlein purpura) taking the place of many of them (9). In general, these initial developments formed the background of contemporary ideas of the SLE as a systemic autoimmune disease that can have a potentially severe organ involvement.

Epidemiology

Systemic lupus erythematosus (SLE) is a significant health burden that is significantly uneven both by sex and by geographic location. As estimated by Tian and colleagues (2023), an estimated 0.4 million individuals globally are exposed to SLE every year, and the incidence of SLE is 5.14 per 100,000 person-years globally (10). The estimates under stratification according to sex show strong female preponderances: women have approximately 0.34 million new cases every year and an incidence rate of 8.82 per 100,000 person-years, men have approximately 0.06 million new cases every year and an incidence rate of 1.53 per 100000 person-years (10). These data highlight the fact that SLE is prevalent in women, but the gap is also significant on the level of the new cases per year. Besides incidence, prevalence (the number of people living with SLE) is also significant. The authors found that approximately 3.41 million SLE patients exist within the global population, which is equivalent to 43.7 per 100,000 (10), as estimated by Tian et al. (2023). Once again, sex-based stratification shows obvious inequality: a total of 3.04 million women were estimated with a prevalence of 78.73 per 100,000, and males numbered 0.36 million with a prevalence of 9.26 per 100,000 (10). All these data points to the fact that women bear the largest proportion of the long-term disease burden across the globe in the number of new cases and overall cases. Another interesting characteristic of the epidemiology of SLE is the geographic variation. According to Tian and associates (2023), the United States, Barbados, and Poland were ranked highest with the highest estimated occurrences that had been assessed among the regions analyzed (10). The causes of such differences between countries are typically understood in terms of a combination of interacting factors such as population genetics, environmental exposures, access to healthcare, disparities in study methodology and case ascertainment, but the pattern observed in the first place reminds us of the fact that SLE is not evenly distributed worldwide (10).

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Sex disparity and reproductive-age predominance

The commonly accepted and persistent epidemiological feature of SLE is that it is highly female predominant, especially at the age of reproduction. There are numerous reports of an estimated 9:1 ratio between women and men with the disease being most commonly seen in women who are of reproductive age (11). The trend corroborates the hypothesis that biological factors (such as hormonal and genetic processes) of sex related susceptibility, immune activation, and disease expression are involved in SLE.

Proposed genetic explanation: X chromosome–linked mechanisms

In addition to the effect of hormones, genetic processes attributed to the X chromosome have also been suggested as causing the sex difference in SLE. A study indicated that females tend to develop SLE more often than males and implied that this predisposition can be associated with X-linked abnormalities and/or changed expression of the genes that are related to the X chromosome (11,12). Zucchi et al. (2022) also emphasized how B cells in SLE contribute to X-linked gene expression patterns which could cause gender bias (12). This has a biological basis since females have two X chromosomes and the differing degrees in X-linked gene dosage regulation (including genes which might not be fully inactivated) may enhance expression of immune-related genes, which in turn would modulate autoimmune risk and immune responsiveness (12).

Immunological Abnormalities of Systemic Lupus Erythematosus

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responsiveness (12).

Etiology:

Even though the exact etiology of systemic lupus erythematosus (SLE) is not fully understood, there has been a lot of evidence that has shown that the disease is caused by complex interactions between environmental exposures, hormone effects, and genetic susceptibility. The sum of these interacting factors finally leads to a common pathogenic event: loss of self-tolerance, that permits the immune responses against the body parts (14). Immune activation in genetically susceptible persons promoted by environmental factors and immune responsiveness by hormonal effects can contribute to breaking immune homeostasis and triggering the process of immune autoimmunity (14).One of the most important immunological abnormalities in SLE is the ineffective disposition of cellular debris, especially nuclear material, which is released during abnormal or dysregulated cell death. In the normal physiological dynamics, the body has tissue homeostasis to regulate the continuous elimination of dying cells and cell debris in a highly regulated fashion. Nevertheless, defects the removal of necrotic material and apoptotic debris contribute to the survival of intracellular constituents in the extracellular space in SLE (15). Since most of these components are nuclear (e.g., DNA, histones, and ribonucleoproteins), the excessive exposure of these components to the immune system predisposes the immune system to treat them as antigens. It is closely associated with cell death pathway modification, such as apoptosis, and is believed to be involved in autoantibody formation in response to nuclear self-antigens that define SLE (15).

In the real world, phagocytic clearance is very important in the removal of dying cells sometimes known as efferocytosis. In apoptosis, the dying cells undergo production of certain surface cues, often defined as the eat-me patterns, that allow the phagocytes (e.g. macrophages) to recognize and ingest them to ensure the prevention of inflammation and organic balance (16). In the event of an effective recognition and clearance process, the apoptotic material is quickly internalized and transported to lysosomes. Effective engulfment of dead cells Apoptotic cells within lysosomes are broken down into basic biological building blocks, and hence, reused, thus, preventing the release of potentially inflammatory intracellular material (16).Conversely, unsuccessful engulfment of the dead cells has relevant pathological implications. Failing to clear the apoptotic cells in time may result in secondary necrosis of the apoptotic cells, which causes the release of intracellular and nuclear material into the tissues. The emitted materials are danger

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signals that have the potential to activate innate immune sensors and facilitate an inflammatory response. This means that inefficient efferocytosis may be one of the primary triggers of immune response, which connects a failure to clear dying cells with the subsequent autoimmune and chronic inflammation.

Clinical and Serological Diversity and Classification Criteria in SLE

The presence of a broad range of autoantibodies and a great variety of clinical manifestations are the features of systemic lupus erythematosus (SLE). This clinical and serological heterogeneity is one of the most important issues in establishing the correct and timely diagnosis. Patients can have various combinations of symptoms that involve a variety of organ systems and autoantibodies can change significantly across patients and across time. As a result, there is no clinical characteristic or laboratory examination that allows making a diagnosis, and SLE is a complicated disorder that needs to be clinically judged carefully. These are clinical manifestations, serology, imaging and histopathology in case it is present. The fact that all these parameters are to be considered jointly points to the multifactorial character of SLE and highlights the necessity that misdiagnosis or delayed diagnosis is widespread, especially at the early stages of this disease. To facilitate the diagnostic process, a number of classification criteria have evolved over the decades and major biomarkers have been discovered to assist the clinicians and researchers.

Standards for Classification of SLE

To standardize the selection of patients in clinical research, a number of sets of classification criteria for SLE have been developed in the first place. However, these criteria are frequently applied in the field of clinical practice as a guideline or a baseline in diagnostic assessment [17,18]. The major limitation of them as a diagnostic tool, however, was the comparatively low sensitivity of about 83 percent, implying that a considerable number of patients with actual SLE would be overlooked [19]. This was especially high in early disease where the sensitivity decreased to approximately 66, meaning that one in six patients were not correctly diagnosed on disease occurrence. The other significant limitation of the 1997 ACR standards was that several of its clinical and immunological items are usually gradually built up during the disease progression. Consequently, the early-stage patients might not be reached by the necessary number of criteria, even though they have the active disease, which restricts its use in early

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diagnosis. To address these limitations, the 2012 Systemic Lupus International Collaborating Clinics (SLICC) classification criteria were introduced. These standards broadened and narrowed clinical as well as immunological elements and emphasized more on the immunological abnormalities. The SLICC criteria were showing better results with a higher sensitivity of approximately 84, and the sensitivity was 97 across the board. This increased sensitivity though, came at the expense of specificity which declined to 84 percent when the SLICC criteria were used compared to 93 percent with the ACR criteria [19]. Nevertheless, the SLICC criteria were a major step forward in the process of capturing a wider scope of SLE manifestations. The latest one is the 2019 European League Against Rheumatism/American College of Rheumatology (EULAR/ACR) classification criteria, which were aimed to strike a better balance in sensitivity and specificity and enhance applicability in early disease [18]. Such criteria mandate a positive antinuclear antibody (ANA) test as a condition of entry, which is an indication of the central place of ANA in SLE. As a follow-up on the criterion of this entry, patients undergo an evaluation of seven clinical areas and three immunological areas, which comprise complement proteins, antiphospholipid antibodies, and SLE-specific autoantibodies. The weighted nature of each criterion and a cumulative score being used to classify a disease allow a more subtle and quantitative evaluation of disease characteristics [17,18]. In general, the development of the criteria of classification in SLE has been facilitated by an increased knowledge of the immunopathogenesis and clinical complexity of the disease. Even though classification criteria are not supposed to substitute clinical judgment, they offer useful advice on diagnosis, and early identification of the problem, and enhance uniformity in clinical research and patient care.

Genetic Susceptibility in SLE

Systemic lupus erythematosus is mainly developed by genetic predisposition. Various investigations have found links between SLE and certain genetic loci, especially those connected to immune regulations, complement systems and interferon signaling. Overall, changes in genes HLA-DR, STAT4, IRF5, and PTPN22 have been highly associated with vulnerability to the diseases. Such genetic factors are not independent and, instead, they interplay with environmental triggers in order to facilitate immune dysregulation and autoimmunity(20,21).

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Role of Cytokines and Interferon Pathway

SLE is characterized by cytokine imbalance, and type I interferon (IFN) pathway is of particular focus. High concentrations of IFN-a help in improving antigen presentation, B-cell activation and production of autoantibodies. There are other cytokines, such as BAFF (B-cell activating factor), IL-6, and TNF-a that are also very critical in maintaining inflammation and immune activation. The idea of the interferon signature is now a significant biomarker of disease activity and therapeutic targeting of SLE(22,23).

Complement System Abnormalities

A defect of the complement system is frequently seen in SLE and is closely linked to the disease activity. The missing early complement fragments like C1q, C2, and C4 have a close connection with the development of SLE. Low complement levels frequently indicate active immune complex formation and degradation especially in lupus nephritis. Complement proteins are thus useful as pathogenic factors as well as useful biomarkers in clinical practice(24,25).

Lupus Nephritis: Pathogenesis and Clinical Importance

One of the most serious forms of systemic lupus erythematosus (SLE) is lupus nephritis (LN), which is a significant predictor of prognosis and outcome in the long term (26). It is mainly caused by the deposition of circulating immune complexes and in situ development of immune complexes in the kidney glomeruli causing the activation of the complement system, inflammatory cell recruitment, and eventual kidney damage (27). The involvement of complement pathways, specifically the classical pathway, in the development of tissue inflammation and progressive tissue damage (26) is also associated with histopathology that is characterized by mesangial-proliferation, endocapillary-proliferation, immune complex deposition, and crescent formation (28) and sclerosis in severe cases of lupus nephritis. Lupus nephritis can clinically manifest itself through proteinuria, hematuria, hypertension, and poor renal performance, which is essential to determining the choice of treatment and predicting the course (29). The most common serological markers in supporting diagnosis and assessing disease activity are the anti-dsDNA (anti-doubles stranded antibodies) and complement (C3, C4) levels (26). Nevertheless, renal biopsy is the best tool for absolute diagnosis, categorisation and evaluation of disease severity (28).Early diagnosis and timely administration of

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immunosuppressive therapy are critical to avoid irreversible renal destruction. Late treatment is a serious risk factor contributing to chronic kidney disease (CKD), end-stage renal disease (ESRD) and death (26). Thus, lupus nephritis is still a severe clinical case that should be promptly identified and treated.

Neuropsychiatric Manifestations of SLE

The neuropsychiatric systemic lupus erythematosus (NPSLE) represents a wide range of neurological and psychiatric manifestations such as seizures, cognitive dysfunction, psychosis, mood disorders, and cerebrovascular disease (30,31). The heterogeneity of SLE is demonstrated by the fact that the American College of Rheumatology (ACR) has identified 19 distinct neuropsychiatric syndromes linked to this condition, which offers a multifactorial pathogenesis (30). Neuronal damage and thrombotic events have been implicated in the use of autoantibodies like anti-NMDA receptor antibodies and antiphospholipid antibodies (32,33). Moreover, neuroinflammation and neuronal dysfunction are also mediated by inflammatory cytokines and complement activation, which adds to the exacerbation of neural injury (32,34). They are also clinically important, impairing quality of life, cognitive functioning, and psychosocial functioning and are difficult to diagnose because of the overlapping symptoms, absence of specific biomarkers, and the necessity to rule out other etiologies such as infection or drug effects (31,35). Treatment of management is a multidisciplinary approach that should be based on the mechanism of pathogenesis, such as immunosuppressive therapy, anticoagulation (where it is necessary) and symptomatic psychiatric or neurological treatment (31,35).

CONCLUSION

Despite the fact that lupus care and long-term outcomes have been greatly enhanced over the last 2 decades, there are still numerous enigmas concerning the condition. The treatment of the chronic diseases, predominantly lupus, requires the best commitment and adherence. The results of the lack of decent medication compliance are example of relapses, resistance cases, and poor results, which are classical. In order to restrict mass and then fat phases then avoid the cardiovascular complications, patients ought to be instructed on the disease pathology, indications and symptoms, medication adherence, the necessity of the tedious supervision, and

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the protective responses such as frequent exercises, nutritional modifications, and lifestyle changes.

Conflict of interests.

There are non-conflicts of interest.

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الخلاصة

الخلفية: الذئبة الحمراء الجهازية (SLE) مرض التهابي مناعي ذاتي متعدد الأجهزة، مزمن ومعقد. تتكون آلية حدوثه من مجموعة من العوامل الداخلية والمحفزات البيئية، والتأثيرات الهرمونية، والعمليات فوق الجينية، مما يؤدي إلى خلل شديد في تنظيم الجهاز المناعي. تتمثل الأعراض الرئيسية للمرض في استجابة مناعية غير طبيعية، حيث تحفز الخلايا التائية ذاتية التنظيم الخلايا البائية لإنتاج أجسام مضادة ذاتية ممرضة. تتنوع الأعراض السريرية للذئبة الحمامية الجهازية بشكل كبير، بدءًا من الأعراض الخفيفة والمحدودة ذاتيًا، مثل التعب والطفح الجلدي وآلام المفاصل، وصولًا إلى إصابة الأعضاء الشديدة والمهددة للحياة، والتي قد تشمل التهاب الكلية الذئبي، والمشاكل العصبية، وأمراض القلب. يمكن أن يصيب المرض عضوًا واحدًا أو يظهر كاضطراب جهازي يصيب أكثر من عضو في الوقت نفسه. يُعدّ هذا التباين الواسع في الأعراض السريرية والمصلية أحد السمات المميزة لمرض الذئبة الحمراء، ويُشكّل صعوبة كبيرة في تشخيص المرض ومتابعته وعلاجه. من الضروري تكوين صورة شاملة للعمليات المناعية المرضية وتباين أعراض الذئبة الحمراء لتعزيز دقة التشخيص، وتحسين النهج العلاجي المُخصّص، وتقليل معدلات الاعتلال والوفيات خلال المتابعة طويلة الأمد للمرض.

الكلمات المفتاحية: الذئبة الحمراء، التاريخ، علم الأوبئة، المناعة، المسببات.