Functional Alterations of Immunological Profiles in Systemic Lupus Erythematosus

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Abstract

The intricate nature of autoimmune disorders and their profound influence on individuals' lives have made them a focal point of substantial research. Systemic Lupus Erythematosus (SLE) is a complex disease that exhibits various immunological profiles, distinguishing it from other disorders. SLE is a persistent autoimmune disorder characterized by the production of autoantibodies, which leads to inflammation and harm to several organs and tissues. Gaining knowledge about the mechanisms and indicators linked to Systemic Lupus Erythematosus (SLE) is essential for precise identification, efficient treatment, and enhanced outcomes for patients. This review article will examine the immunological characteristics of systemic lupus erythematosus (SLE), specifically focusing on the changes in immunological biomarkers and their correlation with disease activity. This review paper was thoroughly compiled using reputable academic resources such as Scopus, Web of Science, Google Scholar, and specialized biomedical databases (PubMed, MEDLINE, and NCBI) to guarantee the inclusion of the most recent and reliable scientific discoveries. The review concluded that Systemic Lupus Erythematosus (SLE) is a complex autoimmune illness with various immunological markers. Comprehending the causes and biomarkers of a condition is essential for precise diagnosis, effective disease control, and enhanced patient results. More research is required to cultivate specific therapeutic interventions and individualized treatment strategies.

Keywords: Autoimmune diseases, Autoantibodies, Immunological profile, Biomarkers

1. Introduction

Systemic Lupus Erythematosus (SLE) is a long-lasting autoimmune illness where the body produces autoantibodies that cause inflammation and harm to many organs and tissues. This condition is intricate and has a broad range of clinical symptoms, which makes it difficult to diagnose and treat. Systemic lupus erythematosus (SLE) primarily impacts women between the ages of 15 and 44, with a greater occurrence among individuals of African, Asian, and Hispanic ancestry. Research suggests that systemic lupus erythematosus (SLE) predominantly affects women, with a gender bias of 9:1. This bias is believed to be influenced by estrogen hormone receptor-1 and unspecified immunomodulatory genes (Tsokos et al., 2016).

The development of SLE is characterized by a disruption in the body's immunological tolerance, resulting in a production of autoantibodies that specifically attack the body's own antigens (self-antigens). The presence of autoantibodies, such as anti-nuclear antibodies (ANA) and anti-double-stranded DNA (dsDNA) antibodies, leads to the formation of immunological complexes. These complexes then accumulate in different tissues and organs, initiating inflammation and causing

damage to the affected tissues and Organs. The disruption of both innate and adaptive immune responses is a contributing factor in the pathogenesis and progression of SLE (Pan et al., 2020).

The disease's etiology is influenced by a combination of genetic predisposition and environmental variables, with gender exerting a notable influence. The autoimmunity of SLE is influenced by several factors, including the initial disruption of tolerance, the presence of B and T lymphocytes that target autoantigens (self-antigens), abnormalities in cell death mechanisms, tissue inflammation, and inadequacies in immune control (Feng et al., 2020).

There are no known methods to prevent or permanently cure Systemic Lupus Erythematosus (SLE). The primary approach for treating SLE is through the use of Immunosuppressive medication. The management of SLE often entails a combination of pharmacotherapy, adjustments to a patient's lifestyle, and regular medical surveillance (Kuhn et al., 2015).

SLE is characterized by a diverse array of symptoms that can vary in intensity from moderate to severe and may not always become apparent. Typical symptoms encompass joint discomfort, skin rashes (often in a symmetrical pattern resembling a butterfly over the face), tiredness, and fever. Additional issues might arise when vital internal organs, such as the lungs, kidneys, heart, and brain, are impacted. People with Systemic Lupus Erythematosus (SLE) may undergo episodes of symptom exacerbation followed by periods of symptom relief, known as remission (Zharkova et al., 2017).

Researchers have examined the biological roles of specific types of white blood cells, including B lymphocytes, T Lymphocytes, and myeloid cells, in the onset and progression of systemic lupus erythematosus (SLE). The initiation and advancement of autoimmunity in Systemic Lupus Erythematosus (SLE) are acknowledged to be impacted by several factors. These factors encompass: dysfunctions in the processes of programmed cell death (apoptosis) or removal of waste stuff, as well as the continuous production of self-antigens also called self-immunogens; inflammation in tissues and deficiencies in immune regulation, along with mechanisms that sustain long-term persistence to promote the development of lupus-related immune disorders; the initial disruption of immune tolerance and the formation of autoantigen-specific active B and T lymphocytes, followed by the production of antinuclear antibodies (ANAs) (Feng et al., 2020, Pan et al., 2020, Tsokos et al., 2016).

The evaluation of systemic lupus erythematosus (SLE) activity strongly depends on the assessment of inflammation in different organs. However, the available routine laboratory markers for inflammation have their limitations. ESR is a rudimentary indicator, but anemia and reduced serum albumin are indicative of intricate connections with inflammation. C-reactive protein (CRP) is more effective in detecting infections compared to monitoring SLE activity. Procalcitonin (PCT) is specifically employed for diagnosing serious bacterial infections. Cytokines, such as IL-18 and TNF-alpha, have a correlation with the activity of inflammatory conditions. However, accurately and promptly measuring these cytokines poses a challenge. Urine proteinuria is crucial for evaluating kidney involvement, but it can also occur as a consequence of renal injury (Aringer, 2020).

This review seeks to examine and unify the existing knowledge about functional alterations in the immune profile of individuals diagnosed with Systemic Lupus Erythematosus (SLE). The objective is to analyze crucial immunological components and pathways in order to discover potential diagnostic and therapeutic insights for a more precise approach in controlling SLE.

2. Immunological Basis of SLE

Systemic Lupus Erythematosus (SLE) is a complex autoimmune condition that affects multiple systems in the body. It is caused by a malfunctioning immune system, which produces autoantibodies and aberrant immune complexes that attack the body's own tissues. The pathogenesis of systemic lupus erythematosus (SLE) is characterized by an intricate interaction of genetic, environmental, and immunological components, resulting in an autoimmune response (Tsokos et al., 2016).

The fundamental characteristic of Systemic Lupus Erythematosus (SLE) is the generation of autoantibodies, specifically antinuclear antibodies (ANAs), as indicated by scientific literature. Autoantibodies specifically attack different constituents of the cellular nucleus, such as dsDNA, RNA, and Nucleoproteins. Autoantibodies create immune complexes by attaching to self-antigens. The immune complexes have the ability to circulate through the bloodstream and accumulate in different tissues, resulting in chronic inflammation and potential harm to the tissues (Lou et al., 2022).

The dysregulation of T and B lymphocytes plays a major role in the development of SLE. T cells become aberrantly activated, contributing to the generation of autoantibodies by plasma cells derived from activated B cells. Disruptions in the communication between T and B cells, such as impairments in CD8+ regulatory T cells, can potentially lead to the loss of immunological tolerance which is the major hallmark in the pathogenesis of SLE (Lou et al., 2022, Pisetsky, 2020). Moreover, research results unequivocally demonstrate that a distinguishing characteristic of systemic lupus erythematosus (SLE) is the existence of overactive B cells and an impairment of B cell tolerance (Gottschalk et al., 2015).

The complement system, a collection of proteins synthesized by the liver and tissue macrophages, serves to augment and control the immune response. In individuals with SLE, this system is frequently activated in an aberrant manner. Dysregulation of the complement system can result in heightened inflammation and harm target tissues. Research has demonstrated that the classical route, activated by the interaction between C1q and immunological complexes, is the primary mechanism for complement activation in systemic lupus erythematosus (SLE). Active illness is associated with reduced amounts of complement proteins from the classical pathway, including C1, C2, and C4 (Sharma et al., 2020).

Another immunological component in SLE is cytokine dysregulation, which causes pro-inflammatory and anti-inflammatory cytokine abnormalities and inflammation. SLE patients have elevated levels of pro-inflammatory cytokines, such as IL-6 and TNF-α. (Zhou et al., 2019). In addition, SLE commonly overproduces Type I interferons, potent antiviral cytokines. Overactive interferon signaling activates immune cells and produces autoantibodies. Research indicates that type I IFNs (e.g., IFN-α and IFN-β) not only contribute to early vulnerability but also contribute to the ongoing disease activity in SLE. Previous cross-sectional studies have linked disease activity to increased type I IFN in the blood (Postal et al., 2020).

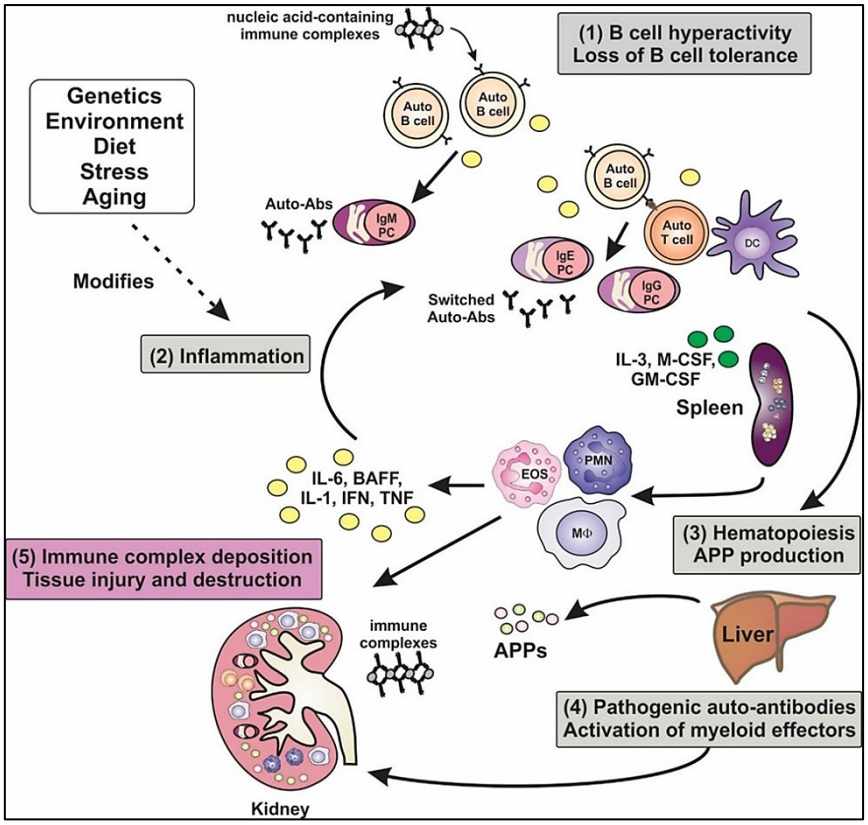

Figure 1: Chronic inflammation is the key factor in the pathogenesis of SLE. The scheme is adapted from (Gottschalk et al., 2015)

3. Cellular Players in SLE

Systemic Lupus Erythematosus (SLE) is a condition where there are anomalies in the way different immune cells work. This leads to chronic autoimmune response and inflammation, which are typical features of the disease.

3.1. T Lymphocytes

SLE affects CD4+ T cells, particularly Helper T cells. T cell activation and differentiation dysregulation produces proinflammatory cytokines and autoantibodies. SLE pathogenesis also involves Th1/Th2 imbalance. An enhanced Th2 response typically shifts the state of balance between Th1 and Th2. This imbalance can cause autoantibodies and immunological complexes (Chen and Tsokos, 2021). Th17 cells and their cytokines, such as IL-17, are also linked to SLE pathogenesis. Therefore, Th17 cells also cause inflammation and tissue damage (Suárez-Fueyo et al., 2016).

3.2. B Lymphocytes

B lymphocytes have a crucial function in systemic lupus erythematosus (SLE) by generating autoantibodies, namely antinuclear antibodies (ANAs). The autoantibodies specifically attack different cellular biological components, such as DNA, RNA, and nucleoproteins. Dysfunctions in B cell tolerance pathways might result in the persistence and stimulation of self-reactive B cells, hence leading to the generation of autoantibodies. Multiple studies have demonstrated that dysregulation in B cell signaling pathways, specifically the B-cell receptor (BCR) and Toll-like receptor (TLR) pathways, can amplify B cell activation and the generation of autoantibodies (Canny and Jackson, 2021).

3.3. Dendritic Cells

T cell receptors (TCRs) receive self-antigens from dendritic cells, which is an important antigen-presenting cells. Dendritic cell dysfunction may cause and maintain SLE autoimmunity. Additionally, SLE patients' dendritic cells may generate higher quantities of type I interferons, notably IFN-α and IFN-β. For that reason, increased interferon signaling activates immune cells and produces autoantibodies (Kaewraemruaen et al., 2020).

3.4. Macrophages

Macrophages phagocytose foreign antigen, present it to T cells, and eliminate cellular debris during inflammation. In SLE, aberrant macrophage clearance pathways may allow apoptotic cells to survive and form immunological complexes, which are crucial to SLE pathogenesis. SLE macrophages produce proinflammatory cytokines and chemokines that cause persistent infection and tissue damage (Ma et al., 2019).

3.5. Natural Killer cells

Natural Killer (NK) cells, which play a role in the innate immune response, may have modified functionality in Systemic Lupus Erythematosus (SLE). Research suggests that alterations in NK cell function may play a role in immunological dysfunction and the ongoing presence of chronic inflammation (Zahran et al., 2019).

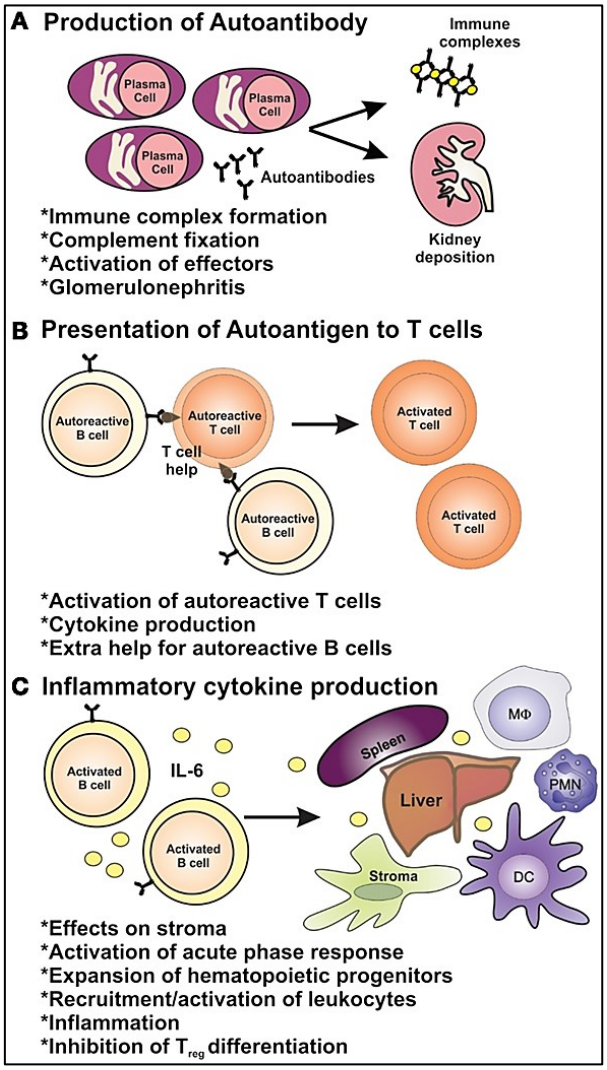

Figure 2: The role of B cells in SLE. The scheme is adapted from (Gottschalk et al., 2015)

4. Dysregulation of Cytokines

SLE is characterized by imbalance in proinflammatory and anti-inflammatory cytokine production and signaling, which causes chronic inflammation. Targeted SLE treatment requires understanding proinflammatory cytokine production and signaling changes. Treatments for SLE frequently control cytokine activity to reduce inflammation and symptoms (Zhou et al., 2019, Gottschalk et al., 2015).

4.1. Interleukin-6

SLE patients frequently have increased IL-6, a proinflammatory cytokine. Immune cells including T, B, and macrophages create it. High IL-6 levels cause systemic inflammation, promote the acute-phase response, and worsen SLE symptoms (Chun et al., 2007). Tang et al. found a positive correlation between serum IL-6, IL-17, and hsCRP levels and cytokine activation in SLE patients, supporting the role of proinflammatory cytokines and complement proteins in SLE pathogenesis (Tang et al., 2019).

4.2. Tumor Necrosis Factor-α

TNF-α is a potent cytokine that regulates the immune system and causes inflammation. Dysregulated TNF-α production in SLE leads to persistent inflammation and tissue damage. TNF-α signaling abnormalities may increase the production of inflammatory mediators such prostaglandins, leukotrienes, and platelet activating factor (PAF) (Jin et al., 2021). Research indicates a genetic relationship between TNF-α gene polymorphism and SLE susceptibility, with clinical symptoms highly linked with TNF-α expression. TNF-α, a powerful B-lymphocyte growth factor, is elevated in SLE patients and associated to disease activity. Dysregulation of TNF-α causes tissue damage, lymphocyte mortality, and poor cell clearance, leading to self-antigen presentation and autoantibody production (Ghorbaninezhad et al., 2022).

4.3. Aberrant signaling of Type 1 Interferons

Type I interferons, such IFN-α and IFN-β, are essential for immune control and antiviral responses. SLE is linked to type I interferon overproduction, which activates immune cells and perpetuates autoimmunity. Interferon signaling increases with autoantibodies and SLE activity (Postal et al., 2020). Type I interferons, such as IFN-α and IFN-β, are critical in the development of Systemic Lupus Erythematosus (SLE), according to various studies. They activate JAK1, TYK2, STAT1, and STAT2, which upregulate IFN stimulated genes (ISGs) transcription, worsening SLE (Gallucci et al., 2021).

4.4. IL-23/IL-17 axis cytokines

SLE is linked to inflammation and autoimmune responses via the IL-17 and IL-23 axis proinflammatory cytokine signaling pathways. In SLE, T helper 17 and regulatory T cell imbalances cause immunological dysregulation. SLE may potentially be caused by IL-23, which promotes T helper 17 cell survival and growth (Izati et al., 2020).

Scientific literature shows that IL-12 and IL-23 are heterodimeric with p40 as a shared globular domain. Chronic inflammation and Th17 cell activation by IL-23 cause IL-17 production, whereas IL-12 stimulates Th1 response and differentiation. New autoimmune disease treatments have emerged from understanding the IL-12-IL-23/Th17 axis. Thus, these findings suggest both mice and humans develop lupus nephritis via IL-23/Th17 axis signaling. (Larosa et al., 2019).

4.5. Dysregulation of IL-1β

SLE is caused by cytokine production, mainly IL-1, IL-1B, and IL-10 family cytokines, according to recent studies. The most researched cytokines are IL-1, IL-1β, IL-18, IL-33, IL-36, IL-37, and IL-38 (Wu et al., 2022). A pluripotent proinflammatory cytokine, IL-1β regulates inflammation and immunological responses, notably in autoimmune disorders. Some studies reported SLE patients have

IL-1β production and processing dysregulation. In SLE, altered IL-1β signaling causes inflammation and tissue damage (Mende et al., 2018).

5. Environmental and Epigenetic factors

Women, racial and ethnic minorities, and individuals with a family history of SLE or associated autoimmune disorders are at higher risk for Systemic Lupus Erythematosus (SLE), which is a multisystem disease with a complicated etiology. SLE susceptibility is influenced by genetics and environment throughout life. Crystalline silica, current cigarette smoking, oral contraceptives, and postmenopausal hormone replacement therapy raise SLE risk, but alcohol usage decreases it. Solvents, pesticides, heavy metals, air pollution, UV light, illnesses, and immunizations may be linked. Environment-induced SLE is linked to epigenetic changes, oxidative stress, systemic inflammation, inflammatory cytokine overexpression, and hormonal consequences. Understanding environmental exposures and SLE development may reveal modifiable risk factors and etiologies.
SLE is also linked to epigenetic modification, which alters gene expression without changing the DNA sequence. Aberrant DNA methylation and histone changes can influence immune-regulating gene expression (Wu et al., 2020).

6. Diagnostic Biomarkers

Diagnosis and monitoring Systemic Lupus Erythematosus (SLE) often requires evaluating a mix of clinical symptoms, laboratory analyses, and Imaging examinations. Several biomarkers are essential for diagnosing, monitoring, and follow-up of SLE. This study aims to provide an illustration and categorization of Biomarkers into three distinct classes: Current Biomarkers, Emerging Biomarkers, and Urine Biomarkers.

6.1. Current Biomarkers

6.1.1. Antinuclear Antibodies (ANAs)

The immune complexes formed by antinuclear antibodies (ANAs) in Systemic Lupus Erythematosus (SLE) usually cause tissue deposition or proinflammatory cytokine production. Some ANAs bind DNA or nucleosome proteins, whereas others bind RNA and RNA-binding protein complexes (RBPs). Currently in clinical settings, ANA autoantibodies are a screening test for SLE due to their high levels. (Pisetsky and Lipsky, 2020).

6.1.2. Anti-dsDNA Antibodies

Antibodies against double-stranded DNA (anti-dsDNA) are unique to SLE and linked to Lupus nephritis (LN), a kidney inflammatory consequence. The most studied lupus autoantibodies are anti-dsDNA. However, some studies designate that anti-dsDNA antibodies may be one of numerous SLE-related autoantibodies with low diagnostic and prognostic value (Fu et al., 2015).

6.1.3. Anti-Sm Antibodies

Anti-Smith (anti-Sm) antibodies exhibit a high degree of specificity towards systemic lupus erythematosus (SLE) and are frequently employed with other autoantibodies associated with lupus for diagnostic purposes. According to literature, there is evidence indicating that in persons who have recently developed systemic lupus erythematosus (SLE), the presence of anti-Sm antibodies is associated with both the initial level of disease activity and subsequent changes in disease activity. Consequently, monitoring the titer of Anti-Sm antibodies helps evaluate the level of disease activity in Systemic Lupus Erythematosus (SLE) (Ahn et al., 2019).

6.1.4. Complement protein levels

SLE pathogenesis and autoimmunity have several causes. Failure of early complement system components that remove immune complexes and apoptotic debris is important. Scientific literature suggests that monogenic anomalies in early complement components such C1q, C1s, C1r, C2, or C4 might cause severe Lupus manifestations. SLE often reduces complement proteins, especially C3 and C4, which may suggest disease activity (Sharma et al., 2020).

6.1.5. Erythrocyte Sedimentation Rate

Active SLE has long been associated with elevated Erythrocyte Sedimentation Rate (ESR). Serum protein changes like hypergammaglobulinemia and fibrinogen can raise ESR. SLE patients with erythrocytopenia and Microcytosis may also have increased ESR level. Thus, ESR monitoring can reveal SLE disease activity and inflammation (Aringer, 2020, Dima et al., 2016).

6.1.6. C-reactive Protein

CRP is a key Biomarker for inflammation. In SLE patients, CRP indicates severe infections rather than disease activity. CRP levels are affected by IL-6, which is increased in active SLE. Active SLE generally has normal CRP values. Scientific data proven that patients with active serositis, arthritis, or myositis have elevated CRP (Aringer, 2020, Dima et al., 2016).

6.1.7. Urinalysis and Proteinuria

Lupus nephritis (LN) is a common and serious SLE complication. Laboratory monitoring is essential for diagnosing new-onset renal involvement and monitoring nephritis activity throughout treatment. Albuminuria, proteinuria, and cellular casts can indicate renal inflammation and illness (Chedid et al., 2020).

6.2. Emerging Biomarkers

6.2.1. Biomarkers for Type 1 Interferons

High blood levels of type I (IFN-α), type II (IFN-γ), and type III (IFN-λ1) IFNs are associated with greater disease activity in SLE patients. Additionally, SLE family members often show increased type I IFN levels, and additional genetic risk factors for SLE exist along the type I IFN pathway, supporting the concept that enhanced type I IFN activity is heritable. IFN-γ levels increase years before SLE diagnosis, but autoantibodies and high type I IFN activity are later symptoms, discovered earlier in the disease. IFN-α is the primary agent of functional type I IFN activity in SLE sera, whereas IFN-β may potentially play a role. Additionally, evidence indicates that the combined advantages of IFN-α, IFN-γ, and IFN-λ1 are substantial in SLE pathogenesis (Postal et al., 2020, Oke et al., 2019).

6.2.2. Proinflammatory Cytokines and Chemokines

Researchers are studying cytokines and chemokines related with inflammation, including IL-6, IL-17, and TNF-α, to gain insights into disease activity and inform therapy options. Numerous studies show that SLE patients have elevated levels of TNF-α, IL-6, IL-4, IL-5, IL-9, IL-12, IL-13, IL-1RA, and IL-10 (Damiati et al., 2023).

6.2.3. MicroRNA Profiles

Small, highly conserved non-coding RNA molecules called microRNAs regulate gene expression. MicroRNAs control B cell growth and activity, which may contribute to SLE pathogenesis. Genome-wide miRNA expression analysis identified microRNAs as possible indicators for disease severity and novel treatment targets in SLE patients. Some miRNAs were strongly up-regulated (such as miR-29b and miR-494) whereas others were dramatically down-regulated in SLE patients. (Duroux-Richard et al., 2015). Carlsen et al. discovered increased miRNA-142-3p and miR-181a in SLE patients, reduced

miR-106a, miR-17, miR-20a, miR-203, and miR-92a, and a substantial reduction in active nephritis patients. These data suggest dysregulated pathways in SLE and distinguish circulating miRNA patterns from other immunoinflammatory illnesses (Carlsen et al., 2013).

6.2.4. Biomarkers for Cardiovascular Risk

SLE increases cardiovascular risk, thus researchers are studying biomarkers of vascular inflammation and endothelial dysfunction. Significant atherosclerosis, which causes asymptomatic premature cardiovascular disease, affects 10% of SLE patients annually. SLE biomarkers include High-density lipoprotein (piHDL), Leptin, Homocysteine, and Sclerostin, which are connected to subclinical atherosclerosis (McMahon et al., 2014, Garcia-de los Ríos et al., 2022).

6.2.5. Autoantibodies against specific targets

Unique autoantibodies targeting particular cellular and tissue targets are being investigated and discovered for diagnostic and prognostic purposes. Lewis et al. found new SLE subgroups using autoantibodies targeting TLR and SMAD pathways and novel autoantigens. The autoantibody clusters showed SMAD2, SMAD5, and MyD88 protein networks. These autoantibodies were linked to organ involvement patterns and increased diagnostic accuracy over traditional testing. Autoantibody clusters may predict organ involvement and stratify SLE patients for certain treatments (Lewis et al., 2018, Yu et al., 2021).

6.2.6. Genetic and Epigenetic Markers

Genetic and Epigenetic markers linked to SLE susceptibility and progression are being studied for diagnosis and prognosis. Research links genetic susceptibilities, environmental factors, and epigenetic control to immune system illnesses. MHC and C1q risk genes cannot explain the increased lupus frequency in homozygous twins. DNA methylation, histone modifications, and non-coding RNA regulations are epigenetic alterations involved in Lupus pathogenesis and potential biomarkers and therapeutic targets (Wu et al., 2020).

6.3. Urine Biomarkers

Lupus nephritis (LN) causes considerable morbidity and death in SLE patients. To screen for LN, guide treatment response, and measure renal flare, urine protein, red blood cells, white blood cells, and cellular casts are tested. Urine biomarkers such as TNF-like weak inducer of apoptosis (TWEAK), MCP-1/CCL2, neutrophil gelatinase-associated lipocalcin, and vascular cell adhesion molecule-1 can predict histopathology, renal flare, and treatment response, according to various research. LN-related SLE patients had greater urinary angiostatin, a proteolytic fragment of plasminogen, than healthy controls. These novel biomarkers may predict LN, SLE, renal flares, treatment response, and histology (Arriens et al., 2016, Yu et al., 2021).

7. Alteration of Immunological Biomarkers in SLE

SLE affects immunological markers such T and B lymphocyte counts and function. Pathogenesis involves autoreactive T cell activation and B cell autoantibodies. SLE relies on monocytes and macrophages to produce inflammatory mediators. Understanding these functional changes is crucial for designing targeted medicines and improving SLE management.

Immune complexes cause inflammation in systemic lupus erythematosus (SLE). Based on their size, specificity, and charge, these complexes produce proinflammatory cytokines and activate immune system effector cells such plasmacytoid dendritic cells (pDC), monocytes, and macrophages, causing organ dysfunction. Most SLE organ activity is inflammatory. Cytokines and chemokines also affect immune dysregulation and function. SLE activity is linked to type I interferons (IFN-α, IFN-β) and

interleukins (IL-6, IL-8, IL-10, IL-15, IL-18, BAFF/BLyS, and TNF-α) (Aringer, 2020, Arriens et al., 2016, Kuhn et al., 2015).

7. Conclusion

In conclusion, this review reveals the Systemic Lupus Erythematosus' immunological complexities. SLE is a complicated chronic autoimmune illness caused by genetic predisposition, environmental variables, and dysregulated immune responses.

B cells, T cells, dendritic cells, macrophages, and natural killer cells are dysregulated in SLE, highlighting the numerous cellular actors implicated in autoimmunity. Genetic factors, Epigenetic alterations, and abnormal cytokine signaling complicate the disease's immunology.

Diagnostic biomarkers established and emerging are essential for SLE diagnosis and monitoring. Traditional indicators like antinuclear antibodies (ANAs) and anti-double-stranded DNA antibodies are important, but new biomarkers including microRNA profiles, genetic and epigenetic markers, and urine biomarkers are promising for more nuanced diagnosis and prognosis.

Targeted therapy is possible by identifying immunological biomarkers and understanding their functional changes in SLE. Future study in this area may improve our understanding of SLE pathophysiology and lead to individualized and effective treatments.

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