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Dealing with Lupus (SLE) and nursing intervention plan: An updated review

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***Abstract*---Background:** Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by periodic flare-ups and remissions, causing multi-organ damage. It is marked by the production of autoantibodies that lead to inflammation and tissue injury. While advances have been made in understanding SLE's pathogenesis, treatments continue to rely on broad immunosuppressive therapies, with more targeted therapies emerging as promising options. This review explores the mechanisms behind SLE and evaluates current and future therapeutic strategies. **Aim:** This review aims to provide an updated overview of the pathogenesis of SLE, recent advances in diagnostic approaches, and the development of targeted treatments, focusing on the potential for personalized therapy. **Methods:** The review synthesizes recent literature on SLE epidemiology, pathogenesis, diagnostic criteria, and therapeutic advancements. It explores the roles of adaptive and innate immunity, mitochondrial dysfunction, apoptosis, and interferon involvement in disease progression. **Results:** SLE's pathogenesis involves dysregulated immune responses, with significant contributions from B and T lymphocytes, type-I interferon (IFN) production, neutrophil dysfunction, and mitochondrial abnormalities. Advances in diagnostic tools, including anti-dsDNA and anti-ENA antibodies, have improved disease identification. Therapeutic strategies now include both traditional immunosuppressive treatments and newer targeted therapies aimed at specific immune pathways, with the goal of reducing reliance on broad immunosuppressants. **Conclusion:** SLE remains a complex, multifactorial disease, with advancements in understanding its pathogenesis leading to promising therapeutic innovations. Targeted therapies and personalized treatment strategies are poised to significantly improve patient outcomes. However, challenges remain in

optimizing treatment approaches and addressing disease variability across different populations.

***Keywords*---**Systemic lupus erythematosus, autoimmune disease, pathogenesis, targeted therapies, immune system, mitochondria, interferon, B cells, T cells, lupus nephritis.

Introduction

Systemic lupus erythematosus (SLE) is a complex autoimmune condition marked by alternating periods of flare-ups and remission, causing significant damage to various organs and tissues. The kidneys, nervous system, joints, and skin are the most frequently affected. A defining characteristic of SLE is the production of circulating autoantibodies that form immune complexes, precipitating in blood vessels and triggering potent inflammatory responses responsible for multi-organ damage [1,2]. Advances in understanding SLE pathogenesis over recent decades have highlighted the dysregulation of both innate and adaptive immune systems, with type-I interferon (IFN) playing a central role. This cytokine induces the overactivation of pro-inflammatory gene expression, a phenomenon referred to as the type-I IFN signature [3]. Despite therapy for SLE still relying on nonspecific immunosuppressive and immunomodulatory drugs [4], targeted therapies focusing on specific immune pathways have recently emerged, with some receiving regulatory approval [5]. However, these newer treatments often require combination with traditional therapies to achieve satisfactory disease control. This review delves into recent advancements in understanding SLE mechanisms, the therapeutic potential of current targeted drugs, and prospects for personalized therapies aimed at minimizing reliance on conventional treatments.

Epidemiology

The global incidence and prevalence of SLE have risen in recent decades, attributed to improved diagnostic techniques and comprehensive international registry data. The estimated incidence of SLE varies significantly by region, ranging from 0.3 to 23.2 cases per 100,000 person-years [6]. North America reports the highest incidence, while sub-Saharan Africa, Europe, and Australia exhibit lower rates, influenced by genetic predisposition [7], socioeconomic factors [8], and environmental conditions [9]. Women of reproductive age are disproportionately affected, with female-to-male incidence ratios between 8:1 and 15:1 [9]. Among ethnic groups, African Americans have the highest SLE incidence and mortality, followed by Hispanic and Asian populations, whereas Caucasians show the lowest rates [10]. African populations, however, display greater susceptibility to SLE and reduced responsiveness to systemic treatments such as corticosteroids and immunosuppressants [11]. Overall, SLE patients face a mortality risk approximately 2.6 times higher than the general population [12], with delayed diagnosis, renal complications, heightened disease activity, infections, and major cardiovascular events identified as primary mortality predictors [11].

Diagnosis, Management, and Disease Activity Criteria for SLE

According to the 2019 classification criteria from the European Alliance of Associations for Rheumatology (EULAR) and the American College of Rheumatology (ACR), a positive antinuclear antibody (ANA) test is required as an entry criterion for SLE diagnosis [5]. However, ANA presence is not exclusive to SLE and may be detected in healthy individuals and other autoimmune or non-autoimmune disorders [13,14,15]. Furthermore, approximately 30% of clinically diagnosed SLE patients are ANA-negative [16]. The anti-dsDNA antibody is considered diagnostic for SLE and is strongly correlated with disease activity [17]. Anti-extractable nuclear antigen (ENA) antibodies, particularly anti-Sm antibodies, are more specific markers for SLE. These are often found alongside anti-U1-ribonucleoprotein (U1-RNP) antibodies, which bind small nuclear ribonucleoproteins (snRNP) and are characteristic of mixed connective tissue disease [18]. Anti-SSA and anti-SSB antibodies, common in Sjögren's syndrome, appear in 24–60% of SLE cases and are associated with neonatal lupus [19]. Anti-histone antibodies are indicative of drug-induced lupus, while anti-ribosomal antibodies are linked to the disease itself. Antiphospholipid antibodies, including lupus anticoagulant, anti-cardiolipin, and anti-β2 glycoprotein 1, serve as markers for vascular inflammation and thromboembolic risk, and are implicated in recurrent pregnancy loss, thrombosis, and neurovascular complications [20,21]. The diagnostic process requires a cumulative score of at least ten points from clinical and immunological domains, with the criteria demonstrating 96.1% sensitivity and 93.4% specificity [22]. Continuous monitoring and evaluation are crucial for the long-term management of SLE patients. Assessing disease activity poses challenges due to the multisystem nature of SLE. The SLE Disease Activity Index-2K (SLEDAI-2K) is the most widely used tool, categorizing disease severity with scores ranging from ≤6 for mild to ≥12 for severe cases [24]. The 2004 British Isles Lupus Activity Group (BILAG) index, which evaluates eight organ systems, offers a more comprehensive systems-based measure [25]. For organ damage evaluation, the internationally recognized Systemic Lupus International Collaborating Clinics (SLICC)/ACR Damage Index (SDI) is frequently utilized [26]. Clinical trials often rely on the SLE Responder Index (SRI), which integrates criteria from SELENA-SLEDAI, Physician Global Assessment (PGA), and BILAG 2004 [27].

Pathogenesis of Systemic Lupus Erythematosus (SLE)

Role of Adaptive Immunity

B lymphocytes, distinguished by the presence of B-cell receptors (BCRs) on their surface, are essential for identifying pathogens and generating specific antibodies [28]. However, during B-cell development, autoreactive B cells may emerge. Although mechanisms of immunological tolerance, such as clonal deletion and peripheral anergy, typically regulate these cells, these systems can fail, leading to the expansion and activation of autoreactive B cells and potentially triggering autoimmune diseases [29,30,31]. The survival and proliferation of B cells, including self-reactive ones, rely on soluble factors, particularly the B-cell activating factor (BAFF), also termed B lymphocyte stimulator (BLys) [32,33]. Autoreactive B cells predominantly produce autoantibodies targeting nuclear antigens. Toll-like receptors (TLRs), particularly TLR7 and TLR9, play a pivotal

role in this process, driving the production of autoantibodies against double-stranded DNA (dsDNA) and RNA-associated antigens in SLE [34–37]. Long-lived plasma cells (LLPCs), derived from terminally differentiated B cells, are major contributors to sustained autoantibody production. Interaction with CD4+ T cells in lymph node germinal centers transforms short-lived plasmablasts into high-affinity plasma cells, which migrate to bone marrow niches, ensuring their longevity and continued autoantibody secretion [38]. Spontaneous germinal center formation, observed in murine and human SLE, underscores its critical role in autoantibody generation [39]. Additionally, B cells can act as antigen-presenting cells (APCs) to autoreactive T lymphocytes, as evidenced in mouse models [40,41].

The pathogenic role of autoantibodies remains a debated subject. Although autoantibodies can be detected years before clinical manifestations of SLE, suggesting their utility as biomarkers, substantial evidence supports their role in disease progression. For instance, immune complexes in lupus nephritis, comprising autoantibodies such as anti-dsDNA antibodies, contribute to glomerular pathology; their removal is associated with disease improvement [42–44]. Moreover, neonatal lupus erythematosus (NLE) arises from passive transfer of maternal autoantibodies, emphasizing their role in SLE pathogenesis [45]. Thus, autoantibodies are implicated in the clinical manifestations of SLE to a significant extent. Self-reactive T cells are central to SLE development. T-helper 1 (Th1) cells exacerbate SLE through oxidative stress mediated by interferon-gamma (IFNγ) production [46]. Conversely, IL-4-producing T-helper 2 (Th2) cells are reduced in SLE patients, suggesting their protective role and linking disease activity to an increased IFNγ/IL-4 ratio [47]. T-helper 17 (Th17) cells, major producers of the pro-inflammatory cytokine IL-17, also contribute to SLE by inducing neutrophil recruitment, innate immune activation, and B-cell enhancement [48]. Elevated IL-17 levels correlate with lupus nephritis severity and disease activity [49,50]. Regulatory T cells (Tregs) maintain peripheral tolerance to self-antigens, but their role in SLE remains controversial due to conflicting findings. However, Tregs' ability to suppress effector T cells suggests their potential in SLE cell therapy [51–53]. T-follicular helper (Tfh) cells facilitate autoreactive B-cell clone generation in germinal centers, contributing to lupus nephritis through localized aggregation with B cells [54,55]. Interactions between CD4+ T cells and B cells are vital in sustaining autoimmunity, promoting autoreactive B-cell survival and differentiation into autoantibody-producing plasma cells. CD8+ T cells are also implicated in SLE immunopathogenesis, exhibiting functional impairments such as reduced cytolytic activity due to diminished granzyme and perforin production [56]. This phenotype correlates with lower disease flare rates but increases infection susceptibility, compounded by immunosuppressive therapies [57,58]. Finally, the elevated presence of γδ-T lymphocytes in SLE patients highlights their role in autoimmunity [59,60].

Role of Innate Immunity

Neutrophils in SLE exhibit significant dysfunction, including impaired phagocytosis and defective clearance of apoptotic cells, which serve as sources of autoantigens [61–63]. Genetic variations in ITGAM, NCF1, and NCF2 exacerbate these dysfunctions by disrupting phagocytosis and reactive oxygen species (ROS)

regulation [64–66]. Furthermore, neutrophils contribute to abnormal B-cell development through type-I interferon (IFN-I) production, independent of TLR stimulation [67,68]. Low-density granulocytes (LDGs), a subtype of neutrophils enriched in SLE patients, are associated with IFN signature and disease severity. LDGs also exhibit heightened neutrophil extracellular trap (NET) formation, contributing to autoantigen exposure and endothelial damage via ROS [69–73]. Genetic polymorphisms promoting increased NET formation further amplify inflammasome activation in macrophages, driving inflammatory responses [74–78]. Plasmacytoid dendritic cells (pDCs), key producers of IFN-I, are integral to SLE pathogenesis [79–81]. pDCs internalize circulating nucleic acids via FcγRIIa and activate TLR7 and TLR9 pathways, triggering Myd88- and IRAK4-mediated signaling cascades for IFN-I production [82–85]. This production links innate and adaptive immunity by promoting extrafollicular B-cell differentiation into plasmablasts and activating pro-inflammatory T cells, thereby exacerbating SLE [86–89]. Despite their pathogenic role, pDCs also have tolerogenic functions, inducing regulatory T cells (Tregs) and IL-10-producing regulatory B cells (Bregs), which suppress IFN-I production. Dysregulation of this feedback loop is a pivotal factor in SLE development, making pDCs a potential target for therapeutic intervention [90–93].

The Role of Mitochondria

Mitochondria, essential organelles for energy production, generate ATP vital for cell metabolism. During apoptosis, damaged mitochondria release mitochondrial DNA (mtDNA), which is unstable and prone to degradation into antigenic fragments. MtDNA can activate autoreactive T cells in SLE, subsequently stimulating B cells to produce anti-DNA antibodies. Resembling bacterial DNA, mtDNA can also engage toll-like receptors (TLRs), eliciting inflammatory responses such as type-I interferon (IFN) production, contributing to immune tolerance breakdown. Additionally, mitochondrial genetic variants linked to SLE increase oxidative stress, as evidenced by oxidized mtDNA accumulation in neutrophils of affected individuals. During NETosis, oxidized mtDNA may trigger type-I IFN production through plasmacytoid dendritic cells. Furthermore, mitochondrial RNA (mtRNA) serves as another autoantigen, with higher autoantibody levels against mtRNA observed in SLE patients [94-104].

The Role of Apoptosis

Apoptosis, crucial for removing cellular debris and maintaining immune tolerance, relies on nucleases for nucleic acid digestion. Deficiencies in these enzymes, such as DNASE1L3 or TREX1, lead to lupus-like symptoms in animal models. In SLE, impaired apoptotic clearance results in undigested DNA accumulation, immune complex formation, and autoimmune responses. Pattern recognition receptor (PRR) activation by apoptotic remnants exacerbates this defect. Studies confirm that SLE patients exhibit reduced efficiency in apoptotic cell clearance. Furthermore, neutrophil extracellular traps (NETs) complicate the digestion of DNA, promoting type-I IFN secretion by plasmacytoid dendritic cells [105-118].

The Role of Interferons in SLE

Type-I IFN plays a pivotal role in SLE pathogenesis. IFNα and IFNβ are key drivers, activated by PRRs such as toll-like receptors (TLRs) and retinoic acid-inducible gene I (RIG-I). Plasmacytoid dendritic cells, significant producers of type-I IFN, trigger downstream signaling via the IFNα receptor (IFNAR), leading to an inflammatory cascade mediated by JAK1, TYK2, and STAT transcription factors. This pathway induces genes that amplify inflammation. Observations in patients treated with IFNα for other conditions demonstrate a direct link between IFN exposure and lupus-like symptoms, which often resolve upon discontinuation of therapy. Genetic polymorphisms in IFN signaling components, such as STAT and IRF genes, further underscore the genetic predisposition to SLE. Additionally, the IFN signature is emerging as a potential biomarker for tailoring anti-IFN therapies. This intricate interplay between mitochondria, apoptotic pathways, and interferon signaling highlights their collective contribution to SLE's complex etiology and presents avenues for targeted therapeutic interventions [119-138].

SLE Treatment
The EULAR/ACR Recommendations

Systemic lupus erythematosus (SLE) treatment has yet to replace traditional therapies decisively, according to the European League Against Rheumatism (EULAR) and the American College of Rheumatology (ACR). Their guidelines emphasize achieving remission or low disease activity using the "treat-to-target" approach adapted from rheumatoid arthritis management [4, 139, 140, 141]. Hydroxychloroquine (HCQ) is widely recommended for all SLE patients due to its long-term safety, ability to prevent flares, and low cost, making it particularly viable in low-income regions. Its dosage is limited to 5 mg/kg body weight to avoid retinal complications [142, 143, 144]. Glucocorticoid usage is recommended at doses below 7.5 mg/day, or their complete discontinuation if possible, due to risks such as osteoporosis, diabetes, and infection [145]. Immunosuppressants like azathioprine, methotrexate, mycophenolate mofetil, and cyclophosphamide (CYC) serve as steroid-sparing agents, but their application is limited by side effects, including teratogenic risks with mycophenolate and cancer risks with CYC [146, 147, 148]. These considerations necessitate combining innovative biologics with conventional therapies when standard treatments are insufficient [149].

Rituximab

Rituximab, a monoclonal antibody targeting CD20 for B-cell depletion, has theoretical benefits in SLE. However, the EXPLORER and LUNAR trials failed to meet their endpoints due to flawed designs and incomplete depletion of tissue-resident CD20+ B cells [150, 151, 152]. Further analyses revealed benefits, such as reduced proteinuria in lupus nephritis (LN) and clinical improvements in African American and Hispanic subgroups [150, 151]. Innovative anti-CD20 agents, like obinutuzumab, show promise for more effective B-cell depletion and improved renal response in LN, though infection risks remain a concern [156, 157, 158].

Belimumab

Belimumab, an anti-BAFF monoclonal antibody, gained FDA approval in 2011 for moderate to severe SLE in adults and was later approved for pediatric use and LN treatment. The BLISS-52 and BLISS-76 trials demonstrated statistically significant benefits of belimumab in combination with standard therapy, particularly for patients with high disease activity [159, 160, 161]. Subcutaneous administration proved effective in the BLISS-SC study, leading to its approval in 2017 [165]. In LN, the BLISS-LN trial showed improved primary and complete renal responses with belimumab compared to placebo, confirming its kidney-preserving effects [167, 168].

Anifrolumab

Anifrolumab, an anti-type-I interferon (IFN) receptor monoclonal antibody, is approved for moderate to severe SLE at 300 mg every four weeks. Its efficacy was established through the MUSE trial and two phase III TULIP studies. Although TULIP-1 failed to meet its primary endpoint, a protocol modification in TULIP-2 to focus on BILAG-based composite lupus assessment (BICLA) criteria led to successful outcomes, including reduced disease relapse and glucocorticoid use [169, 170, 171]. Pooled analyses confirmed anifrolumab's efficacy, particularly in patients with high type-I IFN signatures, making it a valuable addition to SLE management [173, 174].

Nursing Interventions for Systemic Lupus Erythematosus (SLE)

Systemic lupus erythematosus (SLE) is a complex, chronic autoimmune disorder that involves inflammation of various organ systems. Effective nursing interventions in SLE aim to manage symptoms, prevent complications, and improve the patient's overall quality of life. These interventions are multifaceted, focusing on education, symptom management, psychosocial support, and collaboration with interdisciplinary teams to provide comprehensive care.

Patient Education and Self-Management

A cornerstone of nursing interventions in SLE is patient education. Nurses play a vital role in ensuring that patients understand the nature of their condition, the treatment regimen, and strategies for managing symptoms. First and foremost, nurses should educate patients on the importance of medication adherence. Medications commonly prescribed for SLE, such as hydroxychloroquine, corticosteroids, and immunosuppressants, can have significant side effects and require consistent use to maintain disease control. Nurses must ensure that patients are aware of potential side effects, such as retinal toxicity from hydroxychloroquine or increased susceptibility to infections from immunosuppressive therapies, and emphasize the importance of regular follow-up appointments to monitor these effects. Additionally, patient education should address the avoidance of disease triggers, particularly ultraviolet (UV) light, which can exacerbate symptoms. Nurses can advise patients to use sunscreen and wear protective clothing to limit UV exposure. Furthermore, promoting a healthy lifestyle is essential; nurses should encourage balanced nutrition, regular

exercise, and smoking cessation, as smoking has been shown to worsen the symptoms of SLE and reduce the effectiveness of treatment.

Symptom Monitoring and Management

Symptom management is central to nursing care for SLE patients. Nurses must conduct regular assessments to monitor disease activity and ensure early intervention when symptoms worsen. Key symptoms to monitor include fatigue, joint pain, skin rashes, and systemic involvement such as renal or cardiovascular issues. Using validated tools like the SLE Disease Activity Index (SLEDAI), nurses can objectively assess disease activity and track changes over time. Pain management is often a significant concern for patients with SLE, especially those experiencing arthritis or musculoskeletal discomfort. Nurses are responsible for administering prescribed analgesics and recommending complementary therapies such as heat or cold applications, and physical therapy, to manage pain and improve mobility. Furthermore, nurses must monitor for signs of organ damage, particularly renal involvement, which is common in SLE patients. Regular screening for proteinuria, blood pressure monitoring, and assessing for signs of lupus nephritis are essential components of care. Early detection and management of organ damage are critical to preventing irreversible damage and improving long-term outcomes.

Psychosocial Support

Living with a chronic, unpredictable disease such as SLE can take a toll on a patient's mental and emotional well-being. Therefore, nursing interventions must also address the psychosocial aspects of the disease. Nurses should provide emotional support by creating a safe and supportive environment for patients to express their concerns. Active listening and empathetic communication are essential in fostering trust and alleviating feelings of isolation. Nurses should be proactive in referring patients to counseling services if they experience significant emotional distress, anxiety, or depression. Cognitive-behavioral therapy (CBT) has proven effective in helping patients develop coping mechanisms for dealing with the chronic nature of their illness. Additionally, connecting patients with support groups can provide valuable social interaction and an opportunity for patients to share experiences with others facing similar challenges. These peer interactions often lead to a sense of empowerment and emotional resilience, which are critical for managing a chronic illness.

Preventing and Managing Complications

SLE is associated with a range of potential complications, including cardiovascular disease, infections, and osteoporosis. Nurses play an integral role in preventing and managing these complications. Cardiovascular health is a particular concern, as patients with SLE have a higher risk of developing heart disease. Nurses should monitor patients for signs of hypertension, hyperlipidemia, and other cardiovascular risk factors, encouraging regular physical activity and a heart-healthy diet. In addition to cardiovascular risks, the use of immunosuppressive medications can increase susceptibility to infections. Nurses must educate patients on the signs and symptoms of infection, promote

hand hygiene, and encourage timely vaccination (excluding live vaccines in immunocompromised patients) to prevent illness. Furthermore, long-term use of glucocorticoids, commonly prescribed to manage SLE symptoms, increases the risk of osteoporosis. Nurses should promote calcium and vitamin D supplementation, weight-bearing exercises, and regular bone density screenings to minimize bone loss and fracture risk.

Promoting Medication Safety

Given the complexity of SLE treatment, medication safety is a key nursing responsibility. Nurses must monitor for side effects associated with commonly used medications, such as hydroxychloroquine, glucocorticoids, and immunosuppressive drugs. Hydroxychloroquine, while effective in controlling disease activity, can cause retinal toxicity, requiring regular ophthalmologic examinations. Nurses should educate patients on the signs of potential ocular issues, such as blurred vision or difficulty seeing at night, and the importance of reporting these symptoms promptly. Corticosteroids, another mainstay of SLE treatment, can cause a range of side effects, including weight gain, mood changes, and elevated blood glucose levels. Nurses should regularly assess for these side effects and offer strategies for managing them, such as dietary modifications or adjusting the timing of medication administration. Additionally, immunosuppressive medications can cause hematological and hepatic side effects. Nurses should monitor for abnormal blood counts or signs of liver dysfunction, such as jaundice, and ensure patients adhere to routine laboratory testing to detect these complications early.

Enhancing Coping and Stress Management

Chronic illness often results in significant stress, which can exacerbate symptoms of SLE. Nurses should support patients in managing stress by teaching stress-reduction techniques such as mindfulness, meditation, and deep breathing exercises. These techniques have been shown to reduce symptoms of anxiety and improve overall well-being. Nurses should also encourage patients to engage in social support systems, including family involvement in care. Providing emotional support and fostering a sense of community can alleviate feelings of isolation and improve coping strategies. Additionally, nurses can help patients set realistic goals and expectations regarding their illness, thereby reducing anxiety and promoting a more positive outlook on disease management.

Collaboration with Interdisciplinary Teams

The management of SLE requires a team approach, and nurses are essential in coordinating care among interdisciplinary team members. Nurses should collaborate with rheumatologists to ensure that treatment plans are adjusted based on disease activity and laboratory findings. In addition, dietitians can help develop nutrition plans tailored to the patient's needs, such as managing weight or addressing nutrient deficiencies associated with long-term medication use. Physical therapists may assist in maintaining mobility and improving quality of life, while occupational therapists can help patients adapt to limitations caused by joint pain or fatigue. Effective communication and collaboration with these

professionals ensure that patients receive comprehensive, holistic care that addresses all aspects of their condition. Nursing interventions for patients with SLE are multifaceted, involving education, symptom management, psychosocial support, prevention of complications, and collaboration with interdisciplinary teams. Nurses play a crucial role in improving patient outcomes by empowering patients to manage their condition and preventing complications through early intervention and ongoing monitoring. With a focus on both the physical and emotional needs of the patient, nursing care helps enhance the quality of life for individuals living with SLE, ensuring that they can manage their disease and maintain functional independence. By providing individualized care, nurses contribute significantly to the overall well-being of SLE patients.

Conclusion

Systemic lupus erythematosus (SLE) is a multifaceted autoimmune disorder with complex interactions between genetic, environmental, and immunological factors. Recent advancements in understanding the disease's underlying mechanisms have significantly improved our knowledge of its pathogenesis, which involves the dysregulation of both innate and adaptive immune systems. Key drivers of disease progression include the overactivation of B and T lymphocytes, particularly through autoantibodies such as anti-dsDNA and anti-ENA, and the crucial role of type-I interferons in sustaining inflammation. Additionally, defects in mitochondrial function and the accumulation of apoptotic cells exacerbate immune responses, contributing to tissue damage. This highlights the significance of a dysregulated immune tolerance mechanism in SLE, which allows the survival and activation of autoreactive cells that target self-antigens. The epidemiology of SLE has revealed its higher prevalence in women, particularly those of African, Hispanic, and Asian descent, with significant geographical variations in incidence. Mortality risk is notably higher in SLE patients due to complications like renal failure, infections, and cardiovascular events. The diagnostic process, though challenging due to the multisystem nature of the disease, has benefited from refined classification criteria and the development of specific biomarkers such as anti-dsDNA and anti-ENA antibodies. These biomarkers not only aid in diagnosis but also serve as indicators of disease activity, providing valuable insights into patient prognosis. Treatment strategies for SLE have evolved from nonspecific immunosuppressive agents to the introduction of targeted therapies that focus on specific immune pathways. Drugs such as belimumab, which targets B cell-activating factor (BAFF), have shown promise in reducing disease activity and improving patient outcomes. However, these therapies are often used in combination with traditional treatments to achieve optimal disease control. Despite these advances, there is still a need for personalized treatment approaches, as individual responses to therapy vary significantly. This underscores the importance of a tailored treatment plan based on patient-specific factors such as genetic predisposition and disease manifestations. In conclusion, while significant progress has been made in understanding and managing SLE, further research is required to develop even more effective, personalized therapeutic options. Continued exploration of immune pathways, alongside improvements in diagnostic techniques and patient stratification, will be crucial in addressing the remaining challenges in SLE management. Advances in personalized medicine hold promise for reducing the

reliance on broad immunosuppressive treatments, thus enhancing the quality of life for SLE patients and improving long-term outcomes.

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التعامل مع الذئبة الحمراء (SLE) وخطة التدخل التمريضي - مراجعة محدثة

الملخص:

الخلفية :الذئبة الحمراء الجهازية (SLE) هي مرض مناعي ذاتي مزمن يتميز بتفاقمات دورية وانخفاضات في الأعراض، مما يؤدي إلى تلف متعدد الأعضاء. يتميز المرض بإنتاج الأجسام المضادة الذاتية التي تسبب الالتهاب وإصابة الأنسجة. وعلى الرغم من التقدم في فهم آلية تطور المرض، فإن العلاجات ما زالت تعتمد على العلاجات المناعية العامة، مع ظهور علاجات مستهدفة جديدة تعد خيارات واعدة. تستعرض هذه المراجعة الآليات الكامنة وراء مرض الذئبة الحمراء وتقييم الاستراتيجيات العلاجية الحالية والمستقبلية.

الهدف :تهدف هذه المراجعة إلى تقديم نظرة محدثة حول آلية تطور الذئبة الحمراء، والتقدم الأخير في أساليب التشخيص، وتطور العلاجات المستهدفة، مع التركيز على إمكانيات العلاج الشخصي.

الطرق :تجمع هذه المراجعة الأدبيات الحديثة المتعلقة بالوبائيات، وآلية تطور المرض، ومعايير التشخيص، والتقدمات العلاجية المتعلقة بالذئبة الحمراء. كما تستعرض أدوار المناعة التكيفية والفطرية، واضطراب الميتوكوندريا، والموت الخلوي المبرمج، واشتراك الإنترفيرون في تقدم المرض.

النتائج :تتضمن آلية تطور الذئبة الحمراء استجابة مناعية غير منظمة، مع مساهمات كبيرة من الخلايا اللمفاوية B وT، وإنتاج الإنترفيرون من النوع الأول(IFN) ، واضطراب في الخلايا العدلية، والاختلالات في الميتوكوندريا. وقد حسَّنت التقدمات في أدوات التشخيص، مثل الأجسام المضادة ضد الـ dsDNA والأجسام المضادة ضدENA ، من قدرة التعرف على المرض. تشمل الاستراتيجيات العلاجية الآن العلاجات المناعية التقليدية بالإضافة إلى العلاجات المستهدفة الأحدث التي تركز على مسارات مناعية محددة، بهدف تقليل الاعتماد على الأدوية المناعية العامة.

الخلاصة :تظل الذئبة الحمراء مرضًا معقدًا ومتعدد العوامل، حيث أدى التقدم في فهم آليتها إلى ابتكارات علاجية واعدة. من المتوقع أن تحسن العلاجات المستهدفة واستراتيجيات العلاج الشخصي بشكل كبير نتائج المرضى. ومع ذلك، لا تزال هناك تحديات في تحسين طرق العلاج والتعامل مع التباين في المرض عبر الفئات السكانية المختلفة.

الكلمات المفتاحية :الذئبة الحمراء الجهازية، المرض المناعي الذاتي، آلية تطور المرض، العلاجات المستهدفة، الجهاز المناعي، الميتوكوندريا، الإنترفيرون، الخلاياB ، الخلاياT ، التهاب الكلى الذئبي.