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An overview on the concept of Autoimmunity w. s. r. to Systemic Lupus Erythematosus (SLE) from Modern and Ayurveda perspective

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Abstract: Systemic lupus erythematosus (SLE) is a disease that can affect persons of all ages and ethnic groups and both sexes, but more than 90% of new patients presenting with SLE are women in the childbearing years. SLE is a disease that affects multiple systems. This article attempted to provide an overview on Autoimmunity from Ayurveda and modern perspective w. s. r. to Systemic Lupus Erythematosus. SLE can be correlated with *Uttana* and *Gambhira Vatarakta* based on *sthana* involved in pathogenesis and *Raktadhika Vatarakta* based on predominant *doshas* involved which presents the similar symptoms as that of SLE. Further research over this less explored topic is expected.

Keywords: systemic Lupus Erythematosus, Autoimmunity, *Vatarakta*, Ayurveda

Introduction:

Systemic lupus erythematosus (SLE) is a chronic systemic autoimmune disease with some immunological and laboratory abnormalities, non-specific clinical pathogenicity and special diagnostic testing. The clinical manifestations of SLE differ not only from patient to patient, but also show considerable geographic or ethnic variation between populations [1,2]. In Ayurvedic science, SLE is correlated with specific pathological conditions called *Vatarakta*.

This article attempted to provide an overview on Autoimmunity from Ayurveda and modern perspective w.s.r. to Systemic Lupus Erythematosus.

Autoimmunity:

Autoimmune diseases occur when the immune system attacks endogenous molecules as a result of the breakdown of immune tolerance towards self-reactive immune cells. Many autoimmune diseases are strongly associated with genetic,

infectious, and/or environmental predispositions. It comprises multiple diseases and conditions, ranging from organ-specific to systemic. Autoimmune diseases include insulin-dependent diabetes, rheumatoid arthritis, systemic lupus erythematosus, scleroderma, thyroiditis, and multiple sclerosis. There is also an impact of autoimmune pathology in common health problems such as atherosclerosis, inflammatory bowel disease, schizophrenia, and certain types of infertility. It is an autoimmune disease of poorly understood etiology that affects approximately 3% of the population in North America and Europe, and more than 75% of those affected are women[3].

Systemic lupus erythematosus (SLE):

It is a typical systemic autoimmune disease characterized by the production of a variety of autoantibodies and the formation of immune complexes with a chronic disease course and diverse organ involvement. It is multisystem microvascular inflammation with the generation of numerous autoantibodies, particularly antinuclear antibodies (ANA). The disorder was recognized as early as the Middle Ages, with the 12th-century physician Rogerius first applying the term lupus to the classic malar rash, and Moric Kaposi in 1872 first recognizing the systemic nature of the disease. SLE affects the immune system, reducing the body's ability to prevent and fight infections. Additionally, many of the drugs used to treat SLE also suppress the functioning of the immune system, further reducing its ability to fight infection. SLE affects the immune system, reducing the body's ability to prevent and fight infections. Additionally, many of the drugs used to treat SLE also suppress the functioning of the immune system, further reducing its ability to fight infection. The most common infections affect the respiratory, urinary and skin organs and do not require hospitalization if treated promptly. Other opportunistic infections, particularly salmonella, herpes zoster, and candida infections, are more common in her SLE patients due to altered immune status. Patients with lupus often require special diets related to medical conditions that may occur during the course of the disease. These conditions include steroid-induced osteoporosis or diabetes, cardiovascular disease, and kidney disease. Complications from lupus can be serious and even life-threatening.[5,6,7,8, 52].

Incidence and prevalence and gender distribution of SLE:

Among all the types of autoimmune diseases, SLE is a rare condition which has a prevalence rate of 6%. Various prevalence and incidence of SLE have been reported, and the differences are mainly due to population differences. The lupus registries of Georgia and Michigan report a prevalence of 72.1–74.4 per 100,000 and an incidence of 5.6 per 100,000 person-years in predominantly Caucasian and African American populations. African-Americans have the highest percentage, with more Asian and Hispanic populations than whites. The disease tends to have an earlier onset and more severe disease in African Americans. SLE primarily affects women of childbearing age, with a female-to-male ratio

of 9 to 1 for her. However, although the risk decreases after menopause, women are still at twice the risk of men[9,10].

Genetics[11]:

There is clearly a genetic component to disease susceptibility in SLE. Early evidence in support of this theory came from epidemiological studies of affected twins – monozygotic twins have a concordance rate of about 25%,compared with 2% in dizygotic pairs[12]**.** The genes implicated will have immune functions. Areas of interest include genes that encode proteins involved in antigen presentation (the HLA genes), apoptosis, the Fc receptor, B and T cell function and the production of cytokines and complement. A second area of potential linkage is mapped to the chromosome 1q region, which seems to stand out in affected sibling pair studies [13,14]**.** The loss of tolerance with subsequent immune dysregulation is a consequence of genetic factors, in the setting of environmental triggers and stochastic events with recent studies implicating over 30 genetic loci in the disease pathogenesis [15,16]**.**

Etiopathogenesis:

- Although virtually any organ or system can be affected, SLE commonly affects the skin, joints, hematopoietic system, kidneys, lungs, and central nervous system.
  There is no simple answer to the question 'what causes lupus?' This heterogeneous disease is caused by the complex interaction of a variety of abnormalities which cause disease susceptibility, and/or provoke disease onset or exacerbation. Autoantibodies - At the core of this process is immune dysfunction, and the production of autoantibodies. B lymphocytes from patients with SLE display a lack of self-tolerance, and an inappropriate overproduction of antibodies[17].
- **Immune dysfunction** - Powerful new evidence for the strength of the role of B cells in disease development comes from a recent study of B-cell depletion therapy in patients with SLE, resistant to conventional therapies. Although only a small number of patients have been treated so far, results suggest a beneficial response in the majority. There is good data to show that immune cell signaling is abnormal in SLE[18,19].
- **Complement-** Complement is involved in the clearance of immune complexes, and its function is somehow intertwined with the development of lupus[20].
- **Cytokines** - Cytokines are low-molecular-weight proteins which act as the chemical modulators of the immune system. IL-10 is secreted by T-helper cells, and stimulates B-cell proliferation and antibody production. There is an increasing body of research to suggest that this cytokine may be central to the overproduction of antibodies seen in SLE. The serum concentration of IL-10 in lupus patients is significantly higher than that seen in normal controls. Stimulating lupus

mononuclear cells with IL-10 causes significantly increased production of antibodies [21,22].

- **Apoptosis -** Apoptosis plays an important role in the development of autoimmunity. Casicala-Rosen et al. We showed that the intracellular components that often make up the spectrum of lupus target autoantigens accumulate in vesicles on the surface of apoptotic cells. There is evidence that her T cells in some lupus patients overexpress the oncogene bcl-2, promoting cell survival by reducing apoptosis. This could potentially allow autoreactive T cells to persist, propagating the autoimmune response[23].
- **Hormonal Factors-** Sex hormones play an immunomodulatory role in the development of autoimmune disease. SLE, in particular, predominantly affects women, with females commonly affected up to ten times more than males. Estrogen is further implicated in the pathogenesis of lupus by the observation that SLE tends to affect women in the years between their menarche and menopause[24].
- **Environmental Factors -** The disease model that proposes SLE pathogenesis to be a combination of genetic susceptibility followed by exposure to an environmental trigger, viral infection provides a convenient putative target. Many possible culprits have been investigated. Epstein-Barr virus (EBV) is among the most popular candidates. Ultraviolet light Photosensitivity is a common presenting symptom of SLE. Ultraviolet (UV) light exposure causes rash and even systemic flare in susceptible individuals[25].

Clinical Features:

Patients with SLE can present with a variety of systemic symptoms. Common symptoms include: Fever, malaise, joint pain, muscle pain, headache, loss of appetite, weight loss. Nonspecific fatigue, fever, joint pain, and weight change are the most common symptoms in new cases or recurrent active SLE flares. Fatigue, the most common systemic symptom associated with SLE, may be due to active SLE, medications, lifestyle choices, or concomitant fibromyalgia or mood disorders. commonly occurs in association with other clinical and laboratory markers. These symptoms may mimic other autoimmune diseases, infections, endocrine disorders, chronic fatigue, and fibromyalgia. [26,27].

Manifestations of Systemic Lupus Erythematosus [49]-

1. **Musculoskeletal manifestations:** Joint pain is one of the most common reasons for the initial clinical presentation in SLE patients [28,29]. Joint pain, arthritis, osteonecrosis (avascular necrosis) and myopathy are the main symptoms. Arthritis and joint pain have been identified in up to 95% of SLE patients [30,31,32].
2. **Dermatological manifestations:** The characteristic lupus, or butterfly rash on the nose, occurs in only 30% of SLE patients. 1. malar rash

characterized by erythematous rashes on the cheeks and bridge of the nose 2. Photosensitivity 3. Discoid rash 4. Alopecia (common in the temporal region). Lupus panniculitis as the first manifestation of systemic lupus erythematosus. Other skin manifestations associated with, but not specific to, SLE include Raynaud's phenomenon, reticular derma, panniculitis (deep lupus), bullous lesions, vasculitic purpura, telangiectasia, and urticaria [33,34].

- **Renal Manifestation:** Kidney disease is particularly common in SLE patients in developing countries and is a major cause of morbidity and mortality[35]. Renal failure and sepsis are two of the leading causes of death in SLE patients. The kidney is the most commonly affected organ in SLE. Although only about 50% of patients with SLE develop clinically apparent renal disease, lupus nephritis is a common and potentially devastating manifestation of
- SLE.[36,37].
- **Pulmonary Manifestation**: Pleuritis with pleuritic chest pain, with or without pleural effusion, is the most common feature of acute pulmonary involvement in SLE. Shortness of breath and difficulty breathing can have many causes. Serositis due to pericardial or pulmonary effusion, pulmonary embolism, lupus pneumonia, chronic interstitial lupus lung disease, complement-mediated pulmonary leukoagglutination, or infections associated with lupus disease [38, 37].
- **Gastrointestinal manifestation:** Mouth ulcers are a common feature of SLE. Gastrointestinal symptoms and drug side effects secondary to primary SLE are common in SLE patients. Abdominal pain in SLE is important because it can be directly related to active lupus, including peritonitis, pancreatitis, mesenteric vasculitis, and intestinal infarction. Lupoid hepatitis is another entity in which the liver is the major organ involved.[39,40].
- **Cardiac manifestation:** Autoimmune vascular injury in SLE may predispose to atherosclerotic plaques. Patients with SLE should be carefully evaluated for heart failure or chest pain. Pericarditis, which presents as chest pain, is the most common cardiac symptom of SLE, presenting as positional chest pain that is often relieved when the patient bends over [37].
- **Ocular manifestation:** The ocular manifestations of lupus reflect systemic disease. The presence of ocular symptoms should alert clinicians that disease activity may be present elsewhere. The most common ocular manifestation of SLE is *keratoconjunctivitis sicca* (KCS), occurring in approximately 25% of patients. Conjunctivitis, stromal keratitis, *episcleritis*, and less common diffuse or nodular *scleritis* [41,42].

- **Obstetrics manifestation:** Thyroid dysfunction is more common in SLE patients than in the general population and may be hereditary. Furthermore, SLE patients with antibodies to thyroid peroxidase (anti-TPO) were more likely than controls to have thyroid dysfunction, with 14% of SLE patients having anti-TPO and anti-thyroglobulin (anti-Tg), 68% patients with SLE and thyroid disease vs. 5-6% of the general population[43].
- **Hematology manifestation-** Patients with SLE have complex abnormalities that affect their immune system. A history of multiple *cytopenias* such as leukopenia, *lymphopenia*, anemia, or thrombocytopenia may be suggestive of SLE, among other etiologies. Leukopenia, especially *lymphopenia*, is common in SLE, and due to this and *hypocomplementemia*, SLE patients tend to get frequent infections [44,45].
- **Neurological manifestations:** Neurological symptoms of lupus have been reported in 25-75% of patients and can affect any part of the nervous system. SLE can be generalized or partial and lead to status epilepticus. Aseptic meningitis, myelopathy, optic neuropathy, or other demyelinating disorders may also require urgent evaluation. The severity of cognitive impairment in SLE [46] patients varies. Formal neuropsychiatric examination is impaired in 21-67% of SLE patients. It is unclear whether this represents true encephalopathy, neurological damage, drug effects, depression, or other processes[47]. Headaches and mood disturbances are the most commonly reported neurological symptoms of SLE. Stroke and transient ischemic attack (TIA) may be associated with antiphospholipid antibody syndrome or vasculitis. Migraines may also be associated with antiphospholipid syndrome, although this is less clear [48].

Diagnosis: Diagnosis of SLE can be difficult and no single clinical feature or laboratory abnormalities can confirm the diagnosis of SLE. Instead, SLE is diagnosed based on a series of signs, symptoms, and appropriate laboratory tests. Imaging and histopathology also play an important role. The presence of antinuclear autoantibodies (ANA) is an immunological hallmark of SLE. In clinical settings, the ANA test is often used as part of initial research screening. A positive ANA test is a sensitive test found in 98% of SLE patients, but the presence of anti-DNA antibodies is a much more specific finding. Approximately 60% of SLE5 patients have anti-DNA antibodies [50].

Autoimmunity from Ayurveda perspective:

As per Ayurveda, autoimmune disorders are caused due to an unhealthy lifestyle and diet. Autoimmune diseases occur due to the harmful response of a self-immune system when the immune system starts attacking the body's tissue as a response against antigen (toxins) which can

be called *Ama*. The inflammatory responses that occur in autoimmune disorders are exaggerated by *Ama*. Antigen vitiated Dosha, altered immune response which further damages the tissues. Antigens (Ama) modulate signaling at cellular levels leading to incompatible autoimmune response that damages the tissues. Ankylosing spondylitis, crohn's disease, psoriasis, rheumatoid arthritis, ulcerative colitis and Lupus erythematosus etc. can be considered as autoimmune diseases that arise as response against Ama or antigens[51].

Ayurvedic perspective of Systemic Lupus Erythematosus [58,59,60,61]:

Under the heading Autoimmune disorders, Systemic lupus erythematosus comes under non -organ specific type where involvement of multiple organs can be seen. On a broad basis, this can be correlated with the Disease *'vatarakta'* in *Ayurvedic* context. *Ama* is one of the causative factors in the pathogenesis of Autoimmune diseases. Causes mentioned in Ayurved like *mithya Ahara-vihara chesta, Virudha Ashana* etc., causes *Agnimandya* (low digestive fire) which is unable to digest food. This *apakva* (uncooked) food will attain Amla rasa and ultimately results in Visha rupa Avastha which further leads to *Ama Visha*. When *Ama* (antigens) is associated with the different Dosha, Dhatu or Mala leads to different diseases. *Ama* associates Rasadi Dhatus result in Dhatu pradoshaja Vikaras. Likewise, *Ama* associated with Rakta Dhatu leads to *Rakta Pradoshaja vikaras*. *Vatarakta* vyadhi is one among them[51].

Pathogenesis: Acharya sushruta has explained that. Like *Kushta, Vatarakta* initially affects the *Twak, Mamsa* and later it affects the *Gambhira Dhatu*[53]. Due to the close similarity of its symptoms, this disease is compared to *Raktadhika Vatarakta*. Due to the indulgence in *Ahara* and *Vihara* that causes separate vitiation of Vata dosha and Rakta Dhatu. Vitiated Rakta dhatu creating aavaran (coating) over vata and thereby obstruction in the path of *vata* occurs. This leads to further vitiation of *Rakta dhatu* resulting in the manifestation of vatarakta. Vata is the main Dosha and *Dooshyas* are *Rasa, Rakta, Mamsa* and *Twak*[54].

SLE can be correlated with *Uttana* and *Gambhira Vatarakta* based on sthana involved in pathogenesis and *Raktadhika Vatarakta* based on predominant doshas involved which presents the similar symptoms as that of SLE[55]. Vatarakta is caused due to anyonya avarana of the *Vata* and *Rakta* which causes *shotha* (swelling), *Teevra shoola* (intense Pain) etc. symptoms in different parts of the body. As this is also involved in different systems of the body. By this it can be understood, as per the concept of *dhatu paak,* initially *Uttana Vatarakta* which further vitiates the *gambhira dhatu* leads to *Gambhira Vatarakta*. In *Uttana Vatarakta*, mainly *Twak* and *Mamsa* will be affected which can be presented with symptoms of *Kandu, Daha, Ruk, Toda, Spurana, Tamra Vivarnata* due to vitiated Rakta at the level of Twak and Mamsa which leads to Shotha (inflammation). In *Gambheera Vatarakta, Asthi, Sandhi, Majja* are affected which presents with symptoms of *Sandhi Shoola (severe* joint pain, *Sandhi Shotha (severe joint swelling and stiffness)* and *Tamra Vivarnata* (Reddish Discoloration) at the

affected joints and later it leads to *Vakrata of Sandhi* (permanent structural deformity). In Ayurveda, Vatarakta is explained as one of the examples of *Avarna* Vyadhi. In the context of Vatarakta, while explaining the Sadhya – asadhyata of the diseases, upadrava of the disease has been explained. This can be correlated to the multisystem involvement of SLE as a complication [56,57].

Conclusion:

Systemic lupus erythematosus is a chronic autoimmune disease of heterogeneous connective tissue. Systemic lupus erythematosus is an immune-mediated systemic disease associated with a variety of skin, renal, hematological and musculoskeletal abnormalities. Common symptoms are not specific. Genetic, environmental, sociodemographic, and cultural factors all contribute to differences in geographic/ethnic distribution and severity of clinical manifestations. SLE can be correlated with Uttana and Gambhira Vatarakta based on sthana involved in pathogenesis and Raktadhika Vatarakta based on predominant doshas involved which presents the similar symptoms as that of SLE. Further research over this less explored topic is expected.

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