



Review Article

Volume 12 Issue 5

Sept-Oct 2023

## SLE AN AYURVEDIC APPROACH

<sup>1</sup>Aiswarya K.\*, <sup>2</sup>Surej Subash, <sup>3</sup>Vinitha.C

<sup>1</sup>Final year P.G.Scholar, Department of Kayachikitsa, P.N.N.M. Ayurveda Medical College and Hospital Cheruthuruthy.

<sup>2</sup>Professor Department of Kayachikitsa, P.N.N.M. Ayurveda Medical College and Hospital Cheruthuruthy.

<sup>3</sup>Associate Professor Department of Kayachikitsa, P.N.N.M. Ayurveda Medical College and Hospital Cheruthuruthy

\*Corresponding Author's Email ID: aiswaryabams1993@gmail.com

### Abstract

Auto immunity is defined as the state in which the immune system react against the body's own normal components, producing disease or functional change. There are mainly two types of autoimmunity - Organ specific and Organ nonspecific type.

SLE is one of the organ nonspecific autoimmune disorder. Prevalence is about 20 to 200 per 100000 persons in India. Genetic, environmental, and immunological factors are responsible for the development of this disease. SLE is a disease of unknown etiology with a variety of presenting complaints. The pathology in SLE is related to deposit of immune complexes in various organs, which triggers complement and other mediators of inflammation. Symptoms may vary from person to person. It is a multisystem involving disease, affecting the internal organs like kidney, heart, liver...etc, leading to many complications and ultimately death. The general symptoms include fever, malaise, arthralgia, myalgia, headache, weight loss and loss of appetite. Treatment options depend upon the organ systems involved and severity of symptoms.

In *Ayurveda*, we can consider SLE as a *bahudoshaja vyadhi* mainly having *vidahitwa* and *dhatupaka*. Generally, the *vataraktha* line of management is adopted in this condition, when the symptoms of SLE manifest on the skin we can adopt *uthana* line of management, when it affects the multiple systems according to the severity we can adopt *gambhira* line of management. According to the *roga- rogi bala*, we can choose appropriate medications.

**Key words:** SLE, *Vataraktha*

## Introduction

Autoimmune diseases are the conditions in which the immune system mistakenly attacks one's own body. Autoimmune diseases are presented in two ways,

1. Organ specific autoimmunity.
2. Organ nonspecific autoimmunity

SLE is one among the organ nonspecific autoimmune disease. The first clear description of lupus erythematosus was given by Biett and was reported by his student Cazenave under the term erythema centrifugum in 1833. Lupus is the latin word for wolf. Cazenave discovered red rashes on the patient's face that looked like wolf bite.<sup>1</sup>

Systemic lupus erythematosus is systemic autoimmune disease with multisystemic involvement. The condition is characterised by varying clinical presentations, from mild mucocutaneous manifestations to multi organ and severe central nervous system involvement. Its prevalence is about 20 to 200 per 100000 in India. 90% of women are more affected by this condition, the ratio between the women and men are observed to be 9:1. Both genetic and nongenetic variables are responsible for this condition. Family members have an increased risk of developing SLE. There are mainly three forms of SLE, they are, systemic or disseminated form, discoid form and neonatal lupus.<sup>2</sup>

## Materials and methods

The non-genetic variables are UV radiations and some drugs. A postulated mechanism of this is that UV radiations causes apoptosis of host cells, leading to an increased burden of nuclear fragments. Patients receiving certain drugs including procainamide and hydralazine for more than 6 months develop ANA's with clinical features of SLE appearing in 15 to 20% of them. The general symptoms of SLE includes fever, malaise, arthralgia, myalgia, headache, loss of weight and loss of appetite. Fatigue is the most common symptom associated with SLE. Fever is another common nonspecific symptom of SLE.

Involvement of **musculoskeletal symptoms** are extremely common in patients with SLE, patients most often seek medical attention for joint pain with small joints of the hand and

wrist usually affected, it is one of the most common reason for the initial clinical presentation in patients with SLE. Arthralgia, arthritis, osteonecrosis and myopathy are the principal manifestations.<sup>3</sup>

**Dermatological manifestations** – Lupus was first described as a dermatologic condition. Cutaneous manifestations of SLE comprises of four diagnostic criteria. The first is malar rash, which is characterized by an erythematous rash over the cheeks and the nasal bridge. It lasts from days to weeks and is occasionally painful or pruritic. The second feature is photosensitivity. The third feature may be discoid rash, it also develops in sun exposed areas but are plaque like in character with follicular plugging and scarring. Alopecia is the fourth and often less specific cutaneous feature of SLE. Other cutaneous manifestations related to but not specific to SLE include Raynaud phenomenon, livedo reticularis, lupus profundus (panniculitis) bullous lesions, vasculitis purpura and urticaria. <sup>4</sup>

**Renal manifestations** – lupus nephritis is a common complication in SLE. 6 classes of lupus nephritis are mentioned. Other renal manifestations include thrombotic microangiopathy, vasculitis and atherosclerosis

Class I- Minimal mesangial lupus nephritis

Class II- Mesengial proliferative lupus nephritis

Class III- Focal lupus nephritis

Class IV- Diffuse segmental or diffuse global lupus nephritis

Class V- Membranous lupus nephritis

ClassVI-Advanced sclerosing lupus nephritis<sup>5</sup>

**Pulmonary manifestations-** Pleuritis is the most common pulmonary manifestation. Sometimes it is associated with pleural effusion. Other manifestations in pleura include exudative pleural effusions, acute lupus pneumonitis with bilateral pulmonary infiltrates, interstitial lung diseases, diffuse alveolar hemorrhage associated with capillaritis, pulmonary arterial hypertension, pulmonary embolism and shrinking lung syndrome.<sup>6</sup>

**Cardiovascular manifestations-** It may involve any layer of the heart, including pericardium myocardium, endocardium and even in the coronary arteries. Pericarditis is one of the most common presentation.<sup>7</sup>

**Gastrointestinal manifestations** – Any part of GIT may be involved in SLE. Oesophageal dysmotility, mesenteric vasculitis, lupus enteritis, peritonitis and ascities etc are the disease involved in this.<sup>8</sup>

**Neuropsychiatric manifestations-** Both central and peripheral nervous system may be involved in SLE with psychiatric manifestations.<sup>9</sup>

**Hematological and reticuloendothelial manifestations-** Anemia is present in 50% of patients with SLE. Other causes of anemia in SLE may include iron deficiency anemia, red blood cell aplasia and microangiopathic hemolytic anemia. Leucopenia secondary to neutropenia or lymphopenia is also very frequent and severe.<sup>10</sup>

## Discussion

While analyzing the symptoms of SLE we can see many of the conditions similar to our classic references, like *vataraktha*, *dooshivisha*, *dhatugatajwara lakshanas* etc.....

Here we can adopt *avasthanusari cikitsa*, ie, the treatment is adopted according to the condition of the patient. While analyzing the signs and symptoms of SLE we get similar type of clinical presentations in our classics. Flaring is one of the most important problem in SLE patients. So initially we can consider the *amatwa*

*Ushmanoalpabalatwena dhathumadyamapachitham*

*Dushtamamasayagam rasamama prachakshathe*

So initially we can adopt *amaja* line of management. A patient presenting with *srotorodha balabramsa gourava anilamoodatha*...so initially *ama pachana* is done. This condition is having similarity with *vataraktha*, there is a *misravarana* of *vata* and *raktha*. Many symptoms of SLE is having similarities with *rakthadhika vataraktha*, especially in cutaneous manifestations and muscular involvement we can adopted *uthana* line of *vataraktha* management like *seka*, *pradeha*, *avagaha* ....Ultimately in SLE, *dhatupaka* takesplace, ie, the

involvement of *saptha dhatus* there. While affecting the other organs like kidney liver spleen, *gambheera vataraktha cikitsa* can adopt<sup>11</sup>. *Dooshi visha* having similarities with the SLE, the main pathogenesis of this condition is the deposition of antigen antibody complexes with in the organs and tissues leading to the inflammation, so management of *dooshivisha* treatments, *vamana*, *virechana sodhanas* are helpful to this condition.

We can see similarity of symptoms in both SLE and *dhatugata jwara*. *Rasa dhatugatajwara lakshanas* like *Dainyam* (miserable feeling), *Sadanam* (malaise), *Arochakam* (loss of appetite) *Bahishtapam* (photosensitivity) *Angamarda* (myalgia) *Chardi* (Vomiting). *Raktha dhatugata jwara lakshanas* like *Rakthaushna*, *pidaka* (erythematous rashes) *Rakthashteevanam* (hemetemesis) *daha*, *raga*, *brama*. *Mamsa dhatugata jwara lakshanas* like *Glani* (malaise) *anthardaha*. *Medhodhatugata jwara*, *lakshans* like *Chardi* (vomiting) *glani* (malaise) *arochaka* (loss of appetite). *Asthidhatugata jwara lakshanas* like *vireka* (diarrhoea) *vamana* (chardi) *asthibheda* (myalgia/arthritis) *swasa*, *Majjagata dhatu jwara lakshans* like *anthardaha swasa kasa...* are seen in SLE. So *dhatugata jwara cikitsa* also adopted according to the presentation of patient.<sup>12</sup>

## Conclusion

SLE is one among the autoimmune disorder. While analyzing the signs and symptoms of this disease we can correlate it in many aspects, in our view it is a *bahudoshavastha vyadhi*, so according to the *roga bala* and *rogi bala* we can adopt appropriate treatment modalities. *Rasayana* have a great importance in it. According to the systemic involvement of SLE, after the main cikitsa procedures, we can choose the organ specific *rasayanas* to improve the immune system, and also there by to increase the *ojas*.

## References

1. Johnson AE, Gordon C, Palmer RG, Bacon PA, The prevalence and incidence of systemic lupus erythematosus in Birmingham, England: Relationship to ethnicity and country of birth, *Arthritis Rheum*. 38 (1995) 551–558. doi: 10.1002/art.1780380415. [\[PubMed\]](#) [\[CrossRef\]](#) [\[Google Scholar\]](#)

2. Krishnan E, Hubert HB, Ethnicity and mortality from systemic lupus erythematosus in the US, *Ann. Rheum. Dis* 65 (2006) 1500–1505. doi: 10.1136/ard.2005.040907. [[PMC free article](#)] [[PubMed](#)] [[CrossRef](#)] [[Google Scholar](#)]
3. Mukkera S, Mannem M, Chamarti K, Pillarisetty L, Vulasala SS, Alahari L, Ammu A, Mukkera A, Vadlapatla RK. Systemic Lupus Erythematosus-Associated Serositis Managed With Intravenous Belimumab: A Case Report. *Cureus*. 2022 Feb;14(2):e22639. [[PMC free article](#)] [[PubMed](#)]
4. Skudalski L, Shahriari N, Torre K, Santiago S, Bibb L, Kodomudi V, Grant-Kels JM, Lu J. Emerging therapeutics in the management of connective tissue disease. Part I. Lupus erythematosus and Sjögren syndrome. *J Am Acad Dermatol*. 2022 Jul;87(1):1-18. [[PubMed](#)]
5. Scheen M, Adedjouma A, Esteve E, Buob D, Abisror N, Planche V, Fain O, Boffa JJ, De Seigneux S, Mekinian A, Haidar F. Kidney disease in antiphospholipid antibody syndrome: Risk factors, pathophysiology and management. *Autoimmun Rev*. 2022 May;21(5):103072. [[PubMed](#)]
6. Scheen M, Adedjouma A, Esteve E, Buob D, Abisror N, Planche V, Fain O, Boffa JJ, De Seigneux S, Mekinian A, Haidar F. Kidney disease in antiphospholipid antibody syndrome: Risk factors, pathophysiology and management. *Autoimmun Rev*. 2022 May;21(5):103072. [[PubMed](#)]
7. Scheen M, Adedjouma A, Esteve E, Buob D, Abisror N, Planche V, Fain O, Boffa JJ, De Seigneux S, Mekinian A, Haidar F. Kidney disease in antiphospholipid antibody syndrome: Risk factors, pathophysiology and management. *Autoimmun Rev*. 2022 May;21(5):103072. [[PubMed](#)]
8. Aringer M, Costenbader K, Johnson SR. Assessing the EULAR/ACR classification criteria for patients with systemic lupus erythematosus. *Expert Rev Clin Immunol*. 2022 Feb;18(2):135-144. [[PubMed](#)]
9. Aringer M, Costenbader K, Johnson SR. Assessing the EULAR/ACR classification criteria for patients with systemic lupus erythematosus. *Expert Rev Clin Immunol*. 2022 Feb;18(2):135-144. [[PubMed](#)]

10. Sternhagen E, Bettendorf B, Lenert A, Lenert PS. The Role of Clinical Features and Serum Biomarkers in Identifying Patients with Incomplete Lupus Erythematosus at Higher Risk of Transitioning to Systemic Lupus Erythematosus: Current Perspectives. *J Inflamm Res.* 2022;15:1133-1145. [[PMC free article](#)] [[PubMed](#)]
11. Vaidya Jadhavji T.A., editor. *Charaka Samhita of Acharya Charaka, Chikitsa Sthana, Vatashonita chikitsaa, 29th chapter, verse 27, 35, and 45.* 2nd ed. Chaukambha Surabharathi Prakashan; Varanasi: 2005. p. 629. [[Google Scholar](#)]
12. Agnivesha, Charaka Samhita, Ayurveda- Dipika commentary by Chakrapanidutta, revised ed., Chikitsa Sthana (3:50 - 52), pg. 403, Chaukhambha Surbharati Prakashan, Varanasi, (2005)