

## Original Research Article

# Cutaneous manifestations of systemic lupus erythematosus

Nithya Gayathri Devi D.<sup>1</sup>, Saraswathy P.<sup>2\*</sup>, Kopika S.<sup>3</sup>

<sup>1</sup>Senior Assistant professor of Dermatology, <sup>2</sup>Assistant Professor of Dermatology, <sup>3</sup>Resident, Government Stanley Medical College, Chennai, Tamil Nadu, India

**Received:** 01 March 2018

**Revised:** 16 March 2018

**Accepted:** 17 March 2018

### \*Correspondence:

Dr. Sarawathy P.,

E-mail: [kingling@rediffmail.com](mailto:kingling@rediffmail.com)

**Copyright:** © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

### ABSTRACT

**Background:** SLE is a systemic autoimmune disorder associated with polyclonal activation and interplay of genetic, environmental and hormonal events. The aim of the study was to study the incidence of mucocutaneous spectrum, precipitating factors, HPE, immunofluorescence and lab data.

**Methods:** Prospective cohort study. 100 SLE patients attending MMC from August 2007 to September 2009 are included in study. Detailed history, investigations and 2 years follow up were done.

**Results:** ACLE lesions were seen in 50% patients. Sunlight was the most common precipitating factors [55%]. Mucocutaneous lesions are seen in 45%. Fever is the most common systemic symptom in 45.45%. Photosensitivity is seen in 98% of patients. DLE lesions are seen in 83.66% of patients. 98.5% of histopathology correlates with classical SLE.

**Conclusions:** Most of the results correlate with previous studies with few rare observations. Incidence of SLE is less compared to the Western part of world which may be due to the genetic and environmental factors. Photosensitivity being the most common symptom and sunlight being the most common exaggerating factor in our study may be due to the unprotected environmental exposure. Age, sex, ethnic factor with genetic predisposition are the main modifying factors of the various presentation of the disorder.

**Keywords:** Auto immune disorder, Immunofluorescence, Cutaneous manifestations

### INTRODUCTION

Early renowned physicians described "LUPUS" which is derived from the Latin word which means "WOLF" depicting its nature of destruction.<sup>1</sup> The ulcerated skin lesions typical of this disease signify this feature as it "bites, eats away and destroys". Lupus erythematosus (LE) was identified only as a cutaneous disease, until a century ago when emphasis was transferred from the integument to include visceral manifestations. At the beginning of this century blood vessels and connective tissues distributed throughout the body came to be implicated in the pathogenesis, which lead to the concept of "multisystem malady".<sup>2</sup> In 1942 Klimperer et al were

struck by the many morphological features that were common to diseases like scleroderma, rheumatoid arthritis, PAN, and classified them as collagenosis.<sup>3</sup> The discovery of auto antibodies to various cellular components of different tissue in these diseases has of late given place to the concept of "autoimmune diseases".

### Aim of the study

To study the incidence precipitating and exacerbating factors, relevant laboratory abnormalities, associated disorders, histopathological and immunofluorescence patterns of various spectrum of SLE.

## METHODS

### *Study population*

It is a prospective cohort study done at Government General Hospital, Chennai, Tamil Nadu, India, during the period August 2007 to September 2009.

100 cases of systemic lupus erythematosus who either fulfilled ARA criteria (or) ANA positivity were included in the study, from the patients attending the Skin and Rheumatology department, Govt. General Hospital.

A detailed history regarding the onset, progress, precipitating (or) exacerbating factors, recurrence, number, size, morphology, distribution and sequelae of the lesions was obtained. Symptoms related to cutaneous lesions and internal systems were noted in all cases. In all female patients detailed menstrual and obstetric history was taken to look for anti-phospholipid syndrome. Detailed laboratory investigations including biopsy were done.

Then depending on the above information conclusions and discussions were formulated. All these patients were followed up for 2 years and the patients were categorized according to their clinical features into the following groups.

### *Sample and diagnostic procedures*

#### *Chronic cutaneous lupus erythematosus (CCLE)*

It include (a) Discoid LE (DLE) characterised by well-defined erythematous plaques with thick adherent scales and keratotic plugging, peripheral hyperpigmentation and central hyper, hypo (or) depigmentation, atrophy, telangiectasia, and scarring. It was subdivided into localised type, when lesions were restricted to head and neck and disseminated type (DDLE) when lesions were present elsewhere also, and (b) Lupus erythematosus panniculitis, characterised by well defined, firm, non-tender, subcutaneous plaques with normal (or) indrawn overlying skin and with (or) without ulceration. Presence of DLE lesion elsewhere was taken as confirmatory evidence for the diagnosis.

#### *Subacute cutaneous lupus erythematosus (SCLE)*

It characterized by annular (or) polycyclic erythematous plaques with superficial scales (or) psoriasiform lesions without scarring.

#### *Acute cutaneous lupus erythematosus (ACLE)*

It characterised by acute erythema with edema and confined mostly to the malar area (or) other sun exposed areas. The lesions are sometimes generalised with morbilliform (or) exanthematous eruptions and are

characteristically associated with other cutaneous lesions and systemic manifestations.

The morphological description given above was taken as "classical form" for each specific group.

Biopsy of the skin lesions was done in all patients to confirm the clinical diagnosis of LE. The characteristic and diagnostic histopathological findings in LE are, 1) hydropic degeneration of the basal cells of the epidermis (or) the follicles, 2) patchy lymphohistiocytic infiltrate both independently and around the blood vessels and appendages, 3) degenerative changes in connective tissues like hyalinization, edema, mucin and fibrinoid change.

In addition to the above findings the presence of following histopathological features were taken as suggestive evidence for specific clinical type.

*DLE*- Hyperkeratosis, keratotic plugging both follicular and extrafollicular, epidermal atrophy, inflammatory infiltrate extending up to the deeper dermis and mucin deposits.

*SCLE*- Absence (or) minimal keratotic plugging, no epidermal atrophy, vesicular changes in active borders, focal hydropic degeneration of basal cells and sparse inflammatory infiltrate confined to the upper third of the dermis, dermal edema, extravasation of RBC's and dermal fibrinoid deposits.

*ACLE*- Increased degree of hydropic degeneration of basal cells, sparse dermal cellular infiltrate and upper dermal edema and rarely epidermal necrosis(Fig 8).

All case selected were examined clinically to rule out any systemic manifestations. Depending upon the systemic symptoms patients were investigated in detail pertaining to the system suspected to be involved.

CBC, platelet count, urine for albumin, sugar and deposits, RFT, LFT, CRP, VDRL, RF, CXR, and ECG were done to all patients. APL antibodies were done in all suspected patients. Patients with symptoms suggestive of renal problems and vasculitis were investigated for C3 and C4 levels. Renal biopsy was also done for few patients as suggested by nephrologist. Doppler was done for lower limb vessels for patients with gangrene and leg ulcer. Direct immunofluorescence and indirect immunofluorescence (IDIF) were done in few patients. With all the above methodologies and investigation, results were formulated and conclusions were drawn.

### *Statistical analysis*

The data analysis was performed using descriptive statistics including median, frequency and frequency percentage. Comparisons are made using chi square test

using standard equates. Results were reported with  $p \leq 0.05$  as the accept level of significance.

**RESULTS**

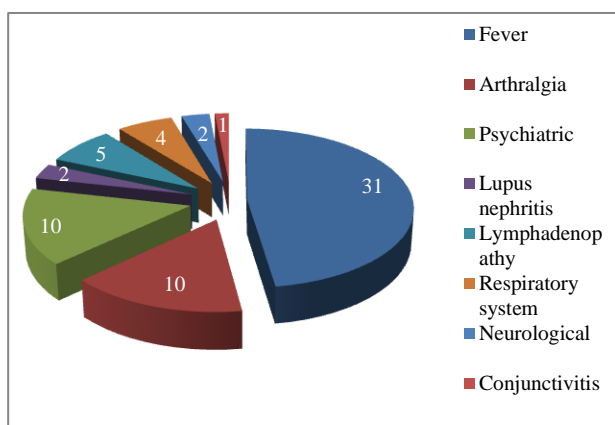
ACLE lesions were seen in 50% followed by CCLE and SCLE patients 46% and 4% respectively (Table 1). Sunlight was the most common precipitating factors [55%] followed by physical exertion and infections [11.11%].

Among the mucocutaneous lesions, 47.91% presented with photosensitivity followed by 26.08% and 21.73% presented with non-scarring alopecia (Table 3) and oral ulcer respectively. 45% of patients presented only with mucocutaneous problems initially, 40.47% of the patients primarily presented with systemic problems. 14.53% of patients presented initially with both systemic and mucocutaneous problems.

**Table 1: Initial clinical presentation.**

Total number of cases	LE specific skin lesions			LE non specific skin lesions	Both
	CACLE	SCLE	ACLE	30 (30.43%)	18 (17.39%)
	19 (19.11%)	13 (13.04%)	17 (17.39%)		

In patients with systemic problems as initial presentation 45.45% had fever and 35.71% had arthralgia (Figure 1). Photosensitivity was seen in almost all patients in SCLE spectrum [99.85%], 41.17% CCLE patients had photosensitivity.



**Figure 1: Associated systemic manifestations.**

**Table 2: Spectrum of LE specific skin lesions.**

LE specific skin lesions	Percentage (%)	
ACLE	Malar rash	68
	Butterfly rash	4
	Maculopapular rash	24
	Bullous lesions	4
SCLE	Annular	33.33
	Papulosquamous	66.66
CCLE	Localised	64.70
	Generalised	35.29
	Verrucous	5.88
	LEP	5.88

Among the CCLE patients 83.66% had classical DLE, 1.22% had verrucous DLE and 1.22% had follicular plugging in the ear and 1.22% had LEP lesions (Table 2). In DLE patients 60% ended up with depigmentation and

scarring. 40% with hyperpigmentation and 15.55% with scarring alopecia. In patients with SCLE 66% of them had papulosquamous lesions and 33% of them had annular scaly plaques both healed without scarring. In ACLE patients maculopapular rash, malar rash and butterfly rash were seen in 37%, 13.33% and 13.33% respectively.

**Table 3: Spectrum of LE nonspecific skin lesions.**

Lesions	Percentage (%)	
Diffuse non scarring alopecia	48.88	
Nail changes	22.22	
Mucosal lesions	22.22	
Icthyosis	8.88	
Vasculitis	Urticarial vasculitis	4.44
	Purpura	4.44
	Leg ulcer	2.22
	Gangrene	2.22
Acanthosis nigricans	4.44	
LE/LP overlap	2.22	
Anetoderma	2.22	

**Histopathology**

Majority of study patients showed features suggestive specific for LE.

In ACLE patients, other than the common features 56% of them had sparse inflammatory infiltrate, 16% had upper dermal edema.

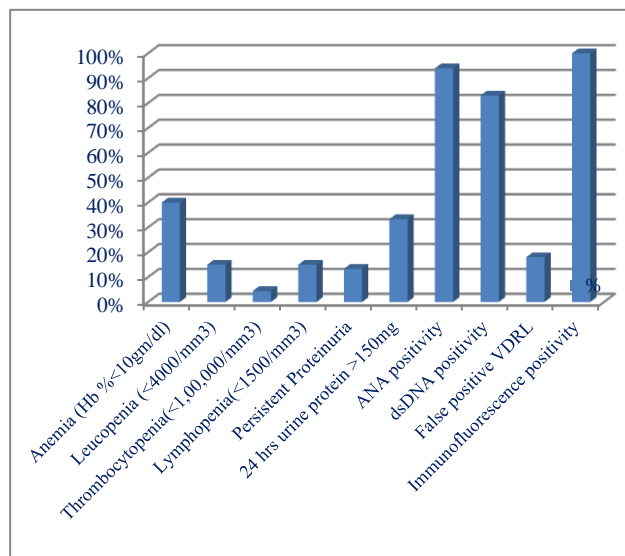
In patients with SCLE, focalliquefactive degeneration was most common [66.66%], followed by upper dermal infiltrate [66.66%] and upper dermal edema [33.33%].

In DLE patients, more than dermal edema, keratotic plugging [51%], colloid bodies [50%], dense and deep dermal infiltrate [51%], periappendageal infiltrate [48%], more melanophages [33%], perivascular infiltrate [29%] were observed.

Patients with urticarial vasculitis, LEP, LE/LP overlap all of them showed features classical of their own histopathological entity.

### Laboratory findings

In our study raised ESR, anemia and leucopenia was observed in 63.34%, 42%, and 15.55% patients respectively (Figure 2).



**Figure 2: Laboratory criteria of ARA in various spectrums of SLE.**

Abnormal 24 hrs urine protein excretion was seen in 33% and grade III lupus nephritis was observed in 50% in our study.

False positive VDRL was seen in 18% of patients and thrombocytopenia in 4.45%.

ANA was done for all the patients and was positive in 94% of the patients.

### DISCUSSION

In this study, among the total 52,369 new patients attending skin op GGH, Chennai, during the study period of August 2007 to September 2009, total number of patients with SLE was 100. The incidence of SLE in our study is 0.08% as compared to incidence of 0.01 to 0.12% in literature.<sup>1</sup> The incidence of 46.5%, 38.8%, 12.7%, and 2% of ACLE, CCLE, SCLE, LEP respectively, which is comparable with our study of 47.94%, 39.92%, 8.48% and 2.86% of ACLE, CCLE, SCLE, LEP respectively as shown in literature.<sup>1</sup>

Burning sensation and erythema were the most common symptoms among patients, which is 63.63% & 42.42% respectively. Photosensitivity was the most common symptom in SCLE patients and pain is the most common symptom in LEP patient as documented in literature.<sup>2</sup>

Photosensitivity was observed in 52% in study done PGI and 54.4% in study done in Iran, which is comparable with our study of 55%.<sup>4</sup>

In a Chinese study, 44.52% presented only with mucocutaneous symptoms as primary complaints which is consistent with our study of 45%.<sup>5</sup>

An Indian study done in Kerala observed primary presenting complaints as photosensitivity in 78% of his study patients, non scarring alopecia in 60%, malar rash in 57%, which is comparable with our study of 80%, 57.77% and 60% respectively.<sup>6</sup>

In a study done in China showed arthritis as primary presenting complaint in 44% while an Indian study showed it as 66% which is comparatively high than our study of 34%.<sup>5,6</sup> Fever was primary presenting complaint in 56.6% of patients in the literature which is high compared to our study of 45.44%.

Photosensitivity as a symptom is seen in 76% of ACLE patients, 62% of SCLE patients and 42% of CCLE patients as per literature.<sup>7</sup> In our study it was 88%, 100% and 43% of ACLE, SCLE and CCLE patients respectively.

Photosensitivity is the most common ARA skin criteria associated with other ARA criteria as given in the literature.<sup>7</sup> Photosensitivity is most commonly associated with 36.65% of hematological criteria and 21.5% of immunological criteria.

In 16 patients with DLE, 73.64% had lesions localised to the head and neck, 26.46% had disseminated type. This is compatible with an Indian study which observed 18.17% had DLE lesions over the scalp which is nearer to our study of 17.7%.<sup>8</sup>



**Figure 3: Annular polycyclic lesions of SCLE.**

In our study majority of ACLE patients had mucosal involvement followed by SCLE and ACLE as described in Iranic study.<sup>4</sup> A study done in India documented oral involvement in 64% in his study as compared to 62% observed in our study.<sup>8</sup> Among the mucosa, palate was

involved in 83.30% of patients, lips in 25% and gingiva in 3.57% (Figure 4).



**Figure 4: ACLE patient with butterfly rash, crusting of lips and conjunctivitis.**

Morphology and sequelae of lesions were classical in the majority of the patients, in all the three spectrum as described in literature.<sup>7</sup> In DLE patients majority of them had plaque like lesions which ended with scarring as described in literature. Few of the patients also had verrucous lesion, follicular plugging lesion in the ear which is also described in literature.<sup>7</sup> In our study two third of SLE patients had papulosquamous lesions and one third had annular lesions (Figure 3), both these types of lesions did not end in scarring as described in the literature.<sup>9</sup>



**Figure 5: Squamous cell carcinoma in a case of LE/LP overlap.**

In our study of ACLE patients, classical lesion as described in literature of ACLE patients like facial erythema, butterfly rash (sparing nasolabial fold) (Figure 4), generalized exanthematous rashes were described.<sup>9</sup> LE/LP overlap turning in to SCC (Figure 5) is seen in one patient, bullous lesion in one patient and LEP (Figure 6) in one patient all of them had their morphology similar as described in literature.<sup>7,9</sup>

As per Gilliam classification of specific and non-specific lesions, specific skin lesions are comparatively common than non-specific skin lesions in our study.

In Kerala study, malar rash was seen in 28%, butterfly rash was seen in 26% of patients and maculopapular rash was seen in 20%.<sup>6</sup> In our study malar rash, butterfly rash and maculopapular rash were seen in 37%, 35%, 18% respectively. Though only few presented with LE specific skin lesions initially, in due course of follow up, majority of them developed LE specific skin lesions.



**Figure 6: Lupus erythematosus panniculitis over abdomen.**

Madras study showed facial erythema in 80% which is observed as 82% in our study.<sup>10</sup> Out of 25 patients with ACLE 24% had butterfly rash, which is consistent with literature.<sup>9</sup> Literature documents malar rash in 80% of patients which is observed as 76.52% in our study. An Japanese study observed bullous SLE in 10% of their patients which is high compared to our study of 2.22%.<sup>11</sup>

Various review studies showed diffuse non scarring alopecia in 86% of patients, which comparable to our study patients with 85%.<sup>12</sup> It was observed that the severity of hair loss was proportional to the disease activity, as also observed by the above same study. Nail changes documented in our study included ragged cuticle in 42%, paronychia in 25%, nail dystrophy in 16%, melanonychia in 6% and beau's lines in 11%. In our study among the above findings ragged cuticle was seen in most of the patients.

Non-specific mucosal lesions are seen in 26% of patients in literature, but in our study it was 22.22%.<sup>9</sup> As per literature vasculitis presented as urticaria in 10% of patients, as purpura in 8%, as leg ulcer in 10%, and as gangrene (Figure 7) in 10% of patients, but in our study it was 4.44%, 4.44%, 2.22% and 2.22% respectively which is very low.<sup>9</sup> Among the 2 patients with urticarial vasculitis 1 patient had renal involvement and low complement levels which states that the urticaria may be due to immune complex mediation. Association of hypocomplementemia was documented with lupus

nephritis and urticaria in literature.<sup>7,9</sup> Patients with leg ulcer, was also associated with urticarial vasculitis. LE/LP overlap was seen in 1 patient, who turned into SCC later, which is a rare documentation in literature.<sup>9</sup>



**Figure 7: Gangrene of foot in a case of ACLE patient.**

By literature observations fever and arthralgia in 92.67% and 22.4% respectively which is comparable with our study of 92.42% and 22.44%.<sup>7,9</sup> As per studies done in Kerala and RadhaMadhavan Madras, lupus nephritis is observed in 35% as compared to 33.33% in our study.<sup>6,10</sup> RadhaMadhavan studies showed renal symptoms as initial complaints in 7.4% but in our study it was 4.04%.<sup>10</sup>

Respiratory problems were seen 26% in Nazarinia Iranian study which is comparatively higher than our study of 8.88%.<sup>4</sup> Radhamadavan observed 25% of neuropsychiatric manifestations in their patients which is nearer to our observation of 26.52%.<sup>10</sup> Among 12 patients with neuropsychiatric manifestations 4.32% had seizures and 40% had psychiatric problems like depression, anxiety neurosis and obsessive compulsive neurosis. Lymphadenopathy was documented as 50% in literature but in our study it was 11.11%.<sup>7,9</sup>

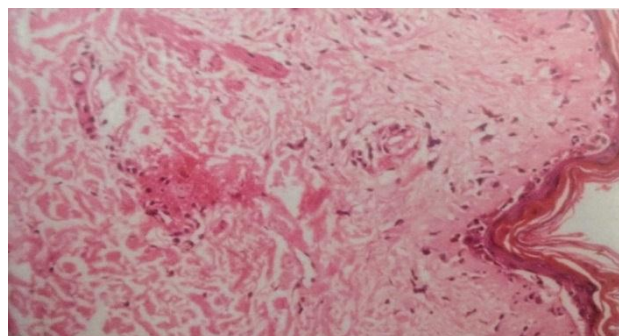
### **HPE**

Classical common histopathological features documented in literature were same in our study.<sup>13</sup> Other uncommon literature findings like atrophic epidermis, vesicular changes in active borders, extravasation of RBCs and fibrinoid deposits were not encountered in our study.

Characteristic HPE (Figure 8) skin changes of LE specific skin lesion as in literature like hyperkeratosis liquefaction degeneration of basal cells periappendageal infiltrate, perivascular infiltrate is seen in majority of patients.<sup>7,9</sup> Varying mosaicism of these features were seen in 3 major spectrum of LE specific skin disease.<sup>13</sup>

It is described in literature that, follicular plugging, keratotic plugging, extensive basal cell degeneration, dense pigment incontinence, patchy inflammatory infiltrate around and independent of appendages and dense deep dermal infiltrate is seen more commonly in DLE than in SCLE or ACLE.<sup>9,13</sup> The above finding is consistent with our study like extensive basal cell

degeneration in 100%, follicular plugging is seen in 74% of patients, keratotic plugging in 65%, dense pigment incontinence in 82%, dense deep dermal infiltrate in 70% and periappendageal infiltrate in 68% of patients.



**Figure 8: Histopathology of ACLE- showing subepidermal cleft and predominantly upper dermal infiltrate.**

Focal liquefaction degeneration of basal cells, dermal edema and sparse inflammatory infiltrate in upper dermis (as against the deep dermal infiltrate of DLE lesions) which are all common documentation in SCLE patients in literature, were also documented in our study. Other uncommon literature findings like atrophic epidermis, vesicular changes in active borders, extravasation of RBC's and fibrinoid deposits were not encountered in our study.<sup>9,13</sup>

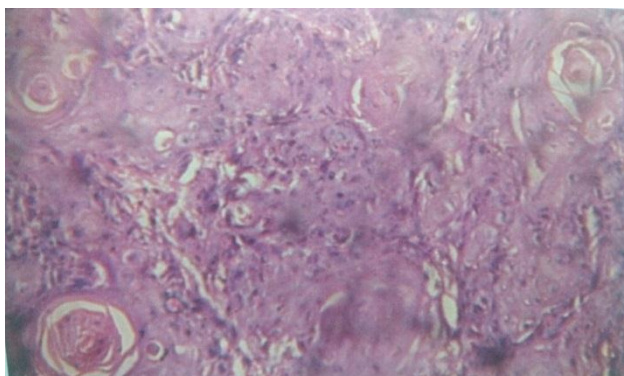
As per literature reports hyperkeratosis is less striking in ACLE than in DLE and SCLE, this finding is consistent with our study.<sup>13</sup> Upper dermal sparse inflammatory cell, and upper dermal edema, which is a common finding documented in literature, was frequently encountered in our study. Histopathological feature of bullous SLE is consistent with the literature. Severe forms of ACLE may have epidermal necrosis and the clinical lesion may resemble TEN. The above finding, which was a rare entity, has not been encountered in our study.

In patients with urticarial vasculitis, histopathological examination showed inflammatory infiltrate predominantly within and around small blood vessels mainly of neutrophils and also leukocytoclastosis. A case of LEP showed minimal atrophy of epidermis, inflammatory infiltrate cell collection in dermis and septilobular lymphohistiocytic infiltrate in subcutis. In patient with LE/LP overlap, hypertrophic DLE lesions histopathologically showed features of lichen planus, but ulcerative DLE lesions showed features of SCC like, individual cell keratinisation and keratin pearls (Figure 9). All the above findings documented in our study were also seen in literature.<sup>13</sup>

### **Laboratory findings**

Paul study showed raised ESR in 65.43%, anemia in 42% and leucopenia in 13% in his patients, which is observed

as 63.34%, 42%, and 15.55% respectively in our study.<sup>6</sup> In Indian study 26.67% had abnormal 24 hrs urine protein excretion which is comparable to 33% in our study.<sup>8</sup> Grade III lupus nephritis was observed in 49.7% of Nazarinia study as compared to our study of 50%.<sup>4</sup>



**Figure 9: Squamous cell carcinoma with horn pearls which occurred over a DLE lesion.**

False positive VDRL was seen in 23% as per literature but in our study it was 18%. Thrombocytopenia was seen in 12.22% Yokohari studies, which is 4.45% in our study.<sup>11</sup>

ANA was done for all the patients. Paul and Nazarinia studies showed ANA positivity in of 93% and 94% respectively, which is almost correlating with our study of 9%.<sup>4,6</sup>

ANA positivity in literature in various spectrum like DLE, SCLE and CCLE are 30%-40%, 60% and 85% respectively.<sup>7,9</sup> But in our study it was 70%, 100% and 100% in CCLE, SCLE and ACLE respectively.

## CONCLUSION

The incidence of SLE varies with the western literature and an increase in incidence shows there is environmental and genetic factors' role. The female preponderance shows that the hormonal influence is the key factor. Photosensitivity being the common symptom and sunlight being the common precipitating factor makes us conclude that there is environmental effect like increased ultraviolet light reaching the atmosphere due to ozone depletion. There is also association with other autoimmune disorders in few patients which states that autoimmune trigger may enhance the possibility. Hence suspected disease overlap with other disorders should be investigated in detail. ACLE patients have more common acute systemic involvement; hence these patients should be thoroughly examined clinically and investigated to reduce the morbidity and mortality. Though most of the SLE patients present initially with dermatological manifestations, it is highly necessary for even the

asymptomatic patients to have periodic screening for other organs involved to reduce the morbidity and mortality.

*Funding: No funding sources*

*Conflict of interest: None declared*

*Ethical approval: The study was approved by the institutional ethics committee*

## REFERENCES

1. Talbott JH. Historical background of discoid and systemic lupus erythematosus in Lupus erythematosus. In: Dubois EL, Ed. New York and London: McGraw Hill Book Company; 1966: 1-9.
2. Walter JB, Israel MS. The collagen disease in General Pathology. Walter JB, Israel MS, editors. 5<sup>th</sup> edition. Edinburgh, London and New York: Churchill, Livingston publications; 1979: 205.
3. Harvy MA, Shulman LE, Tumulty PC, Conley CL, Schoenrich EH. Systemic lupus erythematosus: Review of literature and clinical analysis of 138 case. *Medicine*. 1954;2(33):291-437.
4. Nazarinia MA, Ghaffarparand F, Shamsdin A, Karimi AA, Abbasi N, Amiri A. Systemic lupus erythematosus in the Fars Province of Iran. *Lupus*. 2008;17(3):221-7.
5. Feng PH, Boey ML. Systemic lupus erythematosus in Chinese The Singapore Experience. *Rheumatol Int*. 1982;2:151-4.
6. Paul BJ, Fassaludeen M, Nandakumar, Razia MV. Clinical profile of systemic lupus erythematosus in northern Kerala. *J Indian Rheumatol Assoc*. 2003;11:94-7.
7. Griffiths C, Barker J, Bleiker T, Chalmers R, Daniel Creamer. *Rook's textbook of Dermatology*. Lupus erythematosus. 9<sup>th</sup> edition. 2016: 51.1-51.39
8. Malaviya AN, Chandrasekaran AN, Kumar A, Shamar PN. Systemic lupus erythematosus in India. *Lupus*. 1997;6:690-700.
9. Goldsmith LA, Katz SI, Barnara A, Amy G. *Fitzpatrick dermatology in general medicine, Disorders of connective tissue*. 8th edition. 2006.
10. Madhavan R. SLE – The Madras Experience. *J Assoc Phys India*. 1988;36:481-4.
11. Yokohari R, Tsunematsu T. Application to Japanese patients, of the 1982 American Rheumatism Association revised criteria for the classification of systemic lupus erythematosus. *Arthritis Rheum*. 1985;28:693-8.
12. Masi AT, Kaslow RA. Sex effects in SLE – a clue to pathogenesis. *Arthritis Rheum*. 1978;21:480.
13. Elder DE. *Lever's Histopathology of the Skin*. In: Elenitsas R, Johnson BL, Murphy GF, editors. 10th edition. Chapter 10. Lippincott Williams and Wilkins; 2006.

**Cite this article as:** Nithya Gayathri Devi D, Saraswathy P, Kopika S. Cutaneous manifestations of systemic lupus erythematosus. *Int J Res Dermatol* 2018;4:142-8.