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Association of Discoid Lupus with other Clinical Manifestations among Patients with Systemic Lupus Erythematosus

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Abstract

Background—Cutaneous discoid lupus (DLE) among SLE patients may be associated with less severe disease, with low frequency of nephritis and end-stage renal disease (ESRD).

Objective—To investigate associations between confirmed DLE and other SLE manifestations, adjusting for confounders.

Methods—We identified patients with rheumatologist confirmation, according to ACR SLE classification criteria 1997, >2 visits, >3 months of follow-up, and documented year of SLE diagnosis. DLE was confirmed by dermatologist, supported by histopathology and images. SLE manifestations, medications and serologies were collected. Multivariable-adjusted logistic regression analyses tested for associations between DLE and each of the ACR SLE criteria, and ESRD.

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Results—A total of 1,043 SLE patients, (117 with DLE and 926 without DLE), were included in the study. After multivariable adjustment, DLE in SLE was significantly associated with photosensitivity (OR 1.63), leukopenia (OR 1.55) and anti-Smith antibodies (OR 2.41). DLE was significantly associated with reduced risks of arthritis (OR 0.49) and pleuritis (OR 0.56). We found no significant associations between DLE and nephritis or ESRD.

Limitations—Cross-sectional data collection with risk of data not captured from visits outside system.

Conclusions—In our SLE cohort, DLE was confirmed by a dermatologist and we adjusted for possible confounding by medication use, in particular hydroxychloroquine. We found increased risks of photosensitivity, leukopenia and anti-Smith antibodies and decreased risks of pleuritis and arthritis in SLE patients with DLE. DLE was not related to anti-dsDNA antibodies, lupus nephritis, or ESRD. These findings have implications for prognosis among SLE patients.

Keywords

Systemic lupus erythematosus; discoid lupus erythematosus; cutaneous lupus erythematosus; prognosis; epidemiology

INTRODUCTION

Systemic lupus erythematosus (SLE) is a heterogeneous autoimmune disease of unknown etiology. It has been proposed that different clinical subsets of SLE exist, each associated with variable manifestations of the disease [1–3]. Several researchers have observed that chronic cutaneous lupus of the discoid variant (‘discoid lupus or DLE’) occurs infrequently among patients with more severe organ involvement, in particular lupus nephritis and end-stage renal disease (ESRD) from nephritis, and seems to impart a better long-term prognosis [4–9]. DLE, a scarring, potentially disfiguring form of cutaneous lupus, is of particular interest to the clinician evaluating a patient with SLE, as it is one of the most outward clinical signs of disease and could provide immediate insight into the clinical prognosis of the patient at the bedside. Furthermore, differing SLE manifestations between patients with and without DLE would suggest different underlying pathophysiologies of SLE subtypes and be the basis for future mechanistic study.

In this study, we evaluate the associations between dermatologist-confirmed DLE among patients with SLE and other common clinical and serologic SLE manifestations.

METHODS

Study Population

The Brigham and Women’s Hospital (BWH) Lupus Center is staffed by 7 SLE expert rheumatologists and serves over 800 SLE patients annually. The BWH Lupus Registry, contains data from 5,030 individuals seen for potential SLE since the 1960s. Medical records have been reviewed by rheumatologists expert in the treatment of SLE, for demographic data, date of first symptoms, date of diagnosis, all ACR criteria, and serologies.

Inclusion Criteria

From the Registry, we identified subjects who fulfilled the following criteria: (1) definite SLE diagnosis per rheumatologist / SLE expert case review, (2) 1997 ACR classification criteria for SLE [10], (3) a documented year of SLE diagnosis and (4) >2 visits and >3 months of follow-up between January 1, 1970 and April, 30, 2011.

Data collection

We collected SLE manifestations, medication and serologic data from review of electronic medical records. Electronic medical record data have been available since October 1, 1989. From the electronic medical records, we collected the following data for all subjects: age at SLE diagnosis, date of SLE diagnosis, self-reported race/ethnicity (Caucasian, African American, Asian, Hispanic, other), sex, duration (months) of follow-up at BWH, number of Lupus Center visits, all ACR criteria for classification of SLE , discoid lupus (ever), SLE-specific serologies (anti-Ro, anti-La, Anti-smith, anti-RNP, anti-dsDNA, ANA initial pattern, anticardiolipin IgM and IgG, lupus anticoagulant), clinical laboratories (thrombocytopenia ever, defined as platelet count < 100,000, anemia, ever, defined as hematocrit < 24, leukopenia, ever, defined as white blood cell count <3,000,) medications (ever use and number of prescriptions for the following medications: steroids (ever/never) [prednisone, prednisolone, medrol, solumedrol], hydroxycholorquine (ever/never), immunosuppressives (azathioprine, cyclophosphamide, methotrexate, mycophenolate mofetil, systemic corticosteroids – ever/never). All patients had testing for anti-dsDNA antibodies in our study, performed by ELISA in our hospital immunology lab.

These data were augmented by individual review of the medical records, in particular for those with dates of diagnoses prior to 1989 to: 1) recover missing data (including above demographic, serologic and medication data); 2) obtain details of diagnosis and treatment. The presence of DLE was confirmed by a board-certified dermatologist (JFM) with review of dermatology / multispecialty notes supported by pathology and digital images, where applicable. DLE is a clinical diagnosis that is further supported by pathology findings. Criteria for confirmation of DLE included a specific diagnosis of ‘discoid’ lupus from a specialist dermatologist AND support from one or more of the following: (1) a clinical description consistent with DLE [elements including follicular plugging, dyspigmentation, atrophy, scar formation, scarring-alopecia, telangiectasia, erythema, scale - *with emphasis on chronic scarring changes*], (2) histopathologic results consistent with DLE in the medical records, and/or (3) photographs in the medical records confirming DLE lesions. All aspects of this project were approved by the Partners’ Healthcare Human Subjects IRB.

Statistical Analyses

We employed Fisher’s exact tests for categorical variables (race/ethnicity), wilcoxon rank sum tests for continuous variables and chi-square tests for medication use among SLE subjects with and without DLE. In univariable, followed by multivariable, logistic regression analyses, we modeled the odds of DLE associated *individually* with each of the ACR SLE criteria, as well as ESRD. Each individual models was adjusted for age at diagnosis, sex, race/ethnicity, disease duration and *ever* use of the following medications individually and in combination: steroids (ever/never), hydroxycholorquine (ever/never), immunosuppressives (azathioprine, cyclophosphamide, methotrexate, mycophenolate mofetil, systemic corticosteroids – ever/never).

Suspected confounders were assessed between the primary predictor of interest and the outcome (DLE) as a > 10% change in the risk estimate with inclusion of the covariate; problematic collinearity diagnostics such as tolerance and variance inflation factor review in the Belsley-Kuh-Welsch method were employed [11]. Each of the ACR criteria and ESRD was re-evaluated in models containing (1) all *ever-use* medications, (2) manual subtraction of individual medications by level of significance in the model and (3) individually (azathioprine, cyclophosphamide, hydroxychloroquine, methotrexate, mycophenolate mofetil, systemic corticosteroids). Wald 95% confidence intervals were calculated for odds ratios. All analyses performed using SAS software version 9.2 (SAS Institute, Cary, North Carolina, USA).

RESULTS

Of 1,043 SLE patients who met inclusion criteria, 92% were female and 51% White; 100% were ANA positive, 66% were anti-dsDNA positive. Mean age at diagnosis was 32 years (± 13) and mean duration of follow-up was 10 years (± 6.5). One hundred and seventeen patients were confirmed to have DLE.

Sociodemographic features and medication usage of SLE patients evaluated in subgroups as those with (n=117) and without DLE are shown in Table 1 along with medication use between the two groups. A statistically significant difference existed between the race/ethnicity of SLE patients with DLE and without DLE (p=0.02). The number of ACR criteria was higher among SLE patients with DLE as compared to those without DLE (p<0.01). Age at diagnosis, SLE disease duration, sex and medication use was not significantly different between groups. There was, however, a non-significant trend towards greater hydroxychloroquine use in SLE patients with DLE compared to those without.

In individual multivariable logistic regression models [adjusting for age at diagnosis, sex, race/ethnicity, disease duration, medication use], among patients with SLE, DLE was significantly associated with the presence of anti-Smith antibodies (OR 2.41, p<0.01), photosensitivity (OR 1.63, p=0.02) and leukopenia (OR 1.55, p=0.04) (Table 2). DLE was inversely associated with both arthritis (OR 0.49, p<0.01) and pleuritis (OR 0.56, p=0.01). We found no significant associations between DLE and malar rash, oral ulcers, pericarditis, proteinuria, casts, seizure, psychosis, anemia, lymphopenia, thrombocytopenia, anti-dsDNA, antiphospholipid antibodies, nephritis or ESRD. No significant associations between DLE and WHO class III-IV nephritis (n=97; DLE+class III/IV nephritis n=26) were found (OR 0.54, p=0.21). Regression models controlling for use of medications individually, or all medications combined in the same model, did not yield significantly different associations (results shown in Table 2).

COMMENT

It was first reported thirty years ago by Gilliam and Prystowsky that patients with SLE and DLE had less frequent and less severe systemic organ involvement [7, 8]. Several other authors, including Callen, have observed that in the subgroup of SLE with active discoid lesions, patients tended to have a more benign disease course [4, 5]. These studies tended to be of relatively small numbers of patients and observational in nature, with data often gathered in the setting of a dermatology clinic, which may have biased toward a group of subjects with less severe systemic disease. It has been noted, in particular, that patients with SLE and DLE had a low prevalence of severe renal disease [5, 6]. Based on these earlier observational data, many clinicians have offered prognostic information to the SLE patient who presents with discoid lesions as part of their SLE clinical phenotype.

More recently, Santiago-Casas et al reported the clinical manifestations and damage accrual among patients with SLE and DLE in the ‘PROFILE’ multiethnic lupus cohort [9]. This group determined that SLE patients with DLE were more likely to have malar rash, photosensitivity, oral ulcers, leukopenia, vasculitis and seizures, while less likely to have arthritis, ESRD and immunologic abnormalities than SLE patients without a history of DLE. They did not however find an association between DLE and nephritis. Cases of DLE in the PROFILE study were not confirmed or validated by an expert dermatologist, and that study did not assess effects of individual and combinations of medications upon the outcomes of interest.

Describing a ‘pure’ subset of DLE patients in these studies is crucial, as other cutaneous lupus-specific and non-specific skin disease may be associated with different SLE

phenotypic subsets of disease. The historic reports of less severe organ involvement pertained specifically to DLE patients seen by expert dermatologists who largely defined this disease entity [4, 5, 7, 8, 12]. Of note, Vasquez, Chong and colleagues compared DLE-only, 'borderline'-DLE/SLE and DLE/SLE patients with regard to several clinical and serologic features [13]. Interestingly, the borderline DLE/SLE and DLE-only patients in their study had low levels of anti-Smith positivity while DLE/SLE patients had relatively elevated anti-Smith levels, complementing our findings of increased anti-Smith positivity among SLE patients with DLE (compared to SLE patients without DLE). Overall, their study concluded that borderline DLE/SLE patients and DLE-only patients appeared more similar to each other in serology, lesion distribution and treatment history than did the DLE/SLE patients.

SLE is clearly a heterogeneous disease but it remains unclear whether SLE subtypes are actually different diseases. Past studies have attempted to distinguish specific subtypes of SLE using clinical observation and description or various statistical clustering techniques [1–3]. Being able to phenotype SLE subsets would have implications for an individual's disease monitoring, offer patients and physicians prognostic and survival information. No specific means of calculating SLE patient survival based on presenting features yet exists. In a few past studies, associations, such as that of anti-dsDNA antibodies with lupus nephritis, and 'mucocutaneous manifestations and arthritis' with a low incidence of 'serositis and hematologic disease', have been reported [2, 14]. Our finding that there is no difference in anti-dsDNA between the groups is interesting and not expected given the historic belief that patients with DLE have a lower incidence of severe manifestations, including renal disease and associated serologies such as anti-dsDNA. A multicenter study of 513 Danish SLE patients evaluated disease manifestations and attempted to define clinical subsets [1]. The rate of nephritis among patients meeting ACR Criteria for the Classification of SLE was 45% with a mean duration of follow-up of 8.2 years from diagnosis and 12.8 years from first symptom. This group identified three clinical 'clusters': 1) predominantly discoid disease with notable absence of malar rash and nephritis, 2) predominant nephritis, serositis and lymphopenia, and 3) malar rash and photosensitivity. Work by To and colleagues in a Chinese SLE population supports the concept that clinical manifestations and severity of SLE cluster into three groups: 1) mucocutaneous/arthritis-predominant with low nephritis incidence, 2) nephritis/hematologic-predominant with low mucocutaneous, and 3) heterogeneous (consisting of all manifestations in no clear cluster) [2]. While these studies have attempted to predict clinical subsets and organ damage with some success, they have not been able to provide practical clinical prognostic information for patients and clinicians.

In this large cohort of SLE patients, we have found an increased frequency of photosensitivity, leukopenia and anti-Smith antibodies among SLE patients with DLE and an inverse association of DLE with both pleuritis and arthritis, after adusting for multiple covariates including medication exposures, individually and as a group. We did *not* observe the inverse associations of DLE with anti-dsDNA antibodies, lupus nephritis, or ESRD that have been noted in other studies. This finding will no doubt have an impact on the information we provide to patients presenting with features of DLE as part of their systemic disease.

Limitations of our study include the cross-sectional and partially retrospective nature of data collection with its inherent risk of missing data. Furthermore, our study is only able to determine the *associations* between DLE and other SLE manifestations and cannot temporally relate these clinical features or imply a causal relationship. Regression models were performed as independent tests, and we are aware of the possibility of issues surrounding multiple testing and that future studies may be performed to reproduce our individual findings.

In summary, our findings could have important implications for prognosis among patients with DLE and possibly for different underlying pathophysiologies of SLE subtypes.

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Abbreviations and Acronyms

ACR	American College of Rheumatology
ANA	Anti-nuclear antibodies
anti-dsDNA	Anti-double stranded DNA antibodies
DLE	Discoid Lupus Erythematosus
ESRD	End-Stage Renal Disease
IRB	Institutional Review Board
SLE	Systemic Lupus Erythematosus

Capsule Summary

- Prior studies suggest that DLE among SLE patients is a marker for less severe disease, often offered as reassurance.
- We did *not* observe any associations (either positive or negative) with DLE and severe lupus manifestations (i.e. renal or neurologic).
- These findings have important implications for counseling our SLE patients with DLE regarding prognosis.

Table 1

Sociodemographic Features of SLE patients with and without Discoid Lupus.

Feature	SLE without Discoid Lupus n=926 (90%)	SLE with Discoid Lupus n=117 (10%)	p-value*
Female (%)	847 (92)	111 (95)	0.28
Race/Ethnicity (%)			
Caucasian	480 (52)	56 (48)	**0.02**
African American	130 (14)	31 (27)	
Hispanic	41 (4)	6 (5)	
Asian	83 (9)	8 (7)	
Other	10 (1)	1 (1)	
Missing race/ethnicity	182 (19)	15 (12)	0.09
Age at Diagnosis in Years, mean (SD)	32.6 (13.5)	32.0 (12.6)	0.91
SLE Duration in Years, mean (SD)	18.4 (10.6)	18.4 (10.5)	0.90
Number of ACR criteria for SLE, mean (SD)	**5.2 (1.2)**	**5.6 (1.4)**	**<0.01**
Medications	**n (%)**	**n (%)**	
Hydroxychloroquine	739 (80)	103 (88)	0.05
Mycophenylate	178 (20)	24 (20)	0.71
Corticosteroids (systemic)	693 (75)	88 (75)	1.00
Methotrexate	101 (11)	18 (15)	0.16
Cyclophosphamide	101 (11)	15 (13)	0.53
Azathioprine	204 (22)	30 (26)	0.41
Rituximab	26 (3)	1 (1)	0.35
Leflunomide	22 (2)	2 (2)	1.00

* Fisher's exact tests for categorical variables (race/ethnicity). Wilcoxon rank sum tests for continuous variables and chi-square tests for medications.

Table 2

Associations between Discoid Lupus and other ACR Criteria for SLE as well as End-Stage Renal Disease

SLE Manifestation (n=positive finding out of 1043)	SLE without DLE (n=926) n (%)	SLE with DLE (n=117) n (%)	Unadjusted OR (95% CI)	Adjusted OR* (95% CI)	Adjusted OR** (95% CI)
Anti-Smith	201 (21.7)	45 (38.5)	2.25 (1.50–3.38)	**2.27 (1.50–3.45)**	**2.41 (1.58– 3.69)**
Photosensitivity	374 (40.4)	60 (51.3)	1.55 (1.06–2.28)	**1.71 (1.15–2.55)**	**1.63 (1.09– 2.44)**
Leukopenia	301 (32.5)	50 (42.7)	1.55 (1.05–2.29)	**1.50 (1.01–2.24)**	**1.55 (1.03– 2.32)**
Pleuritis	349 (37.7)	31 (26.5)	0.59 (0.39–0.92)	**0.56 (0.36–0.88)**	**0.56 (0.36–0.87)**
Arthritis	738 (79.7)	79 (67.5)	0.53 (0.35–0.80)	**0.51 (0.33–0.79)**	**0.49 (0.31–0.76)**
Lupus Nephritis	281 (30.3)	38 (32.5)	1.10 (0.73–1.66)	1.09 (0.71–1.68)	1.33 (0.83–2.14)
Pericarditis	112 (12.1)	10 (8.6)	0.68 (0.35–1.34)	0.68 (0.34–1.36)	0.68 (0.34–1.36)
Proteinuria	256 (27.7)	27 (23.1)	0.78 (0.50–1.23)	0.70 (0.43–1.13)	0.77 (0.47–1.27)
Casts	117 (12.6)	9 (7.7)	0.56 (0.28–1.17)	0.53 (0.26–1.09)	0.57 (0.27–1.20)
End-Stage Renal Disease	48 (5.1)	7 (6.0)	1.16 (0.51–2.64)	0.96 (0.41– 2.22)	1.24 (0.50–3.05)
Oral ulcers	240 (25.9)	37 (31.6)	1.32 (0.87–2.00)	1.35 (0.88–2.07)	1.32 (0.86–2.03)
Malar Rash	406 (43.8)	46 (39.3)	0.82 (0.56–1.23)	0.86 (0.57–1.30)	0.88 (0.58–1.32)
Seizure	100 (10.8)	14 (12)	1.12 (0.61–2.03)	1.14 (0.62–2.09)	1.20 (0.65–2.21)
Psychosis	16 (1.7)	3 (2.6)	1.50 (0.43–5.21)	1.45 (0.41–5.14)	1.50 (0.42–5.38)
Anemia	181 (19.5)	26 (22.2)	1.17 (0.74–1.87)	1.12 (0.69–1.80)	1.15 (0.71–1.86)
Lymphopenia	340 (36.7)	51 (43.6)	1.33 (0.90–1.96)	1.32 (0.88–1.97)	1.38 (0.91–2.08)
Thrombocytopenia	110 (11.9)	18 (15.4)	1.35 (0.79–2.31)	1.45 (0.83–2.54)	1.54 (0.87–2.71)
Anti-dsDNA	610 (65.9)	83 (70.9)	1.27 (0.83–1.93)	1.25 (0.81–1.93)	1.33 (0.86–2.07)
Antiphospholipid antibodies	225 (24.3)	24 (20.5)	0.80 (0.50–1.29)	0.85 (0.52–1.37)	0.87 (0.54–1.43)

OR= odds ratio

* **OR**= Multivariable logistic regression analyses modeling the odds ratio of DLE associated with each SLE manifestation or laboratory finding *individually*, adjusted for age at diagnosis, sex, race/ethnicity, disease duration

** **OR**= Multivariable logistic regression analyses modeling the odds ratio of DLE associated with each SLE manifestation or laboratory finding *individually*, adjusted for age at diagnosis, sex, race/ethnicity, disease duration, ever medication use: steroids (ever/never), hydroxycholorquine (ever/never), immunosuppressives (azathioprine, cyclophosphamide, methotrexate, mycophenolate mofetil, systemic corticosteroids – ever/never)