Al-Azhar International Medical Journal

Volume 5 | Issue 3 Article 7

2024
Section: Internal Medicine

Nail Fold Capilloroscopy Versus Serum interleukin 17 in Rheumatoid Arthritis and Systemic Lupus Erythematosus Patients and their Correlation with Disease Activity in Upper Egypt

Mohamed I. AbdelKareem
Rheumatology and Rehabilitation department, Faculty of Medicine - Al-Azhar University (Assiut)

Mohamed M. Rayan
Rheumatology and Rehabilitation department, Faculty of Medicine - Al-Azhar University (Assiut)

Ashraf I. Sayed
Rheumatology and Rehabilitation department, Faculty of Medicine - Al-Azhar University (Assiut), ashraf.ismail128@gmail.com

How to Cite This Article

AbdelKareem, Mohamed I.; Rayan, Mohamed M.; and Sayed, Ashraf I. (2024) "Nail Fold Capilloroscopy Versus Serum interleukin 17 in Rheumatoid Arthritis and Systemic Lupus Erythematosus Patients and their Correlation with Disease Activity in Upper Egypt," *Al-Azhar International Medical Journal*: Vol. 5: Iss. 3, Article 7.
DOI: https://doi.org/10.58675/2682-339X.2321

ORIGINAL ARTICLE

Nail Fold Capilloroscopy Versus Serum Interleukin 17 in Rheumatoid Arthritis and Systemic Lupus Erythematosus Patients and Their Correlation with Disease Activity in Upper Egypt

Ashraf Ismail Abdelhamed Sayed*, Mohamed Ismail Mohamed AbdelKareem, Mohamed Moneer Mohamed Rayan

Department of Rheumatology and Rehabilitation, Faculty of Medicine, Al-Azhar University, Assiut, Egypt

Abstract

Background: **Nailfold capillaroscopy (NFC) represents the best method to analyze microvascular abnormalities among connective tissue diseases (CTDs) since capillaroscopic changes of the nailfold have been well-established in many CTDs.**

Aim: **To investigate serum interleukin 17 (IL-17) and Nailfold microcirculation in Rheumatoid arthritis (RA) and systemic lupus erythematosus (SLE) patients and to study their correlation with disease activity in upper Egypt.**

Patients and methods: **A total of 50 patients with RA and 30 patients with SLE. In addition to, 30 healthy patients as control group.**

Results: **Patients with either SLE or RA had significantly higher IL-17 in comparison to the control group. Patients with SLE had a significantly higher frequency of Bushy capillaries elongated capillaries, enlarged capillaries, subpapillary plexus, disorganized capillaries, hemorrhage, and avascular area in comparison to the control group. Patients with RA had a significantly higher frequency of bushy capillaries while patients with SLE had significantly higher frequency of elongated capillaries, disorganized capillaries, hemorrhage and avascular area.**

Conclusion: **SLE or RA patients had significantly higher IL-17 in comparison to the control group. NFC, patients with RA had a significantly higher frequency of bushy capillaries while patients with SLE had a significantly higher frequency of elongated capillaries, disorganized capillaries, hemorrhage and avascular area.**

Keywords: Nailfold capillaroscopy, Rheumatoid arthritis, Systemic lupus erythematosus

1. Introduction

Rheumatoid joint torment (RA) is a provocative, safe mediated disorder with a normality of 0.5−1% in made countries.[1]

Th17 cells explicitly produce the imprint cytokines like interleukin 17 (IL-17), IL-21 and IL-22, and have been displayed to expect a fundamental part for the tenacious searing response and resulting tissue hurt in rheumatoid arthritis (RA) affected joints.[2]

Principal systemic lupus erythematosus (SLE) is a tenacious insusceptible framework disease depicted by formation of autoantibodies and safe complex improvement that lead to multi-organ red hot cells entrance and consequently tissue hurt. The pathogenesis of SLE incorporates incitation of a surprising safe structure including the natural resistant cells like dendritic cells (DCs) as well as flexible invulnerable cells including B and Insusceptible framework microorganisms with various cytokine dysregulations.[3]

Nailfold capillaroscopy (NFC) is an easy imaging system and the best strategy for morphological assessment of healthy vessels in the nailfold district.

Accepted 5 November 2023.
Available online 23 May 2024

* Corresponding author. MB.B.CH Faculty of Medicine, Al-Azhar University (Assiut), Master Degree of Rheumatology and Rehabilitation, Demonstrator of Rheumatology and Rehabilitation, Al-Azhar University (Assiut).

https://doi.org/10.58675/2682-339X.2321

It has been used for exploring microvascular anomalies in rheumatologic afflictions, including scleroderma and SLE. The relationships between illness movement and capillaroscopic changes might demonstrate their convenience in deciding sickness seriousness.[4]

Hence, this study aims to examine nailfold microcirculation in this population, assess morphological and structural changes quantitatively and qualitatively, and recognize serum IL-17 in their sera.

2. Patients and methods

This study was carried out on 50 RA patients diagnosed, 30 SLE), and 30 volunteers age and sex matched as a control group of those attended the outpatient clinic and inpatient of Rheumatology and Rehabilitation Department of Al-Azhar university hospital (Assiut) who was given an informed consent to participate in this study. Mature under 18, Other connective tissue sicknesses, and prescriptions: eosinophilic fasciitis, reflex thoughtful dystrophy, morphea, direct scleroderma, diabetic cherioarthropathy, Burger's illness and other vasculitis, diabetes mellitus, and smoking were undeniably precluded. All patients were presented to the going with: full history taking, individual history, history of present disease with examination of the going with fights of joint aftereffects, past history: of drugs, surgeries, sensitivities, blood bondings, and family ancestry: similar conditions, Rheumatic disease. Disease development score for 28 joints (DAS-28) in RA patients, which is a really resolved document containing number of sensitive joints, number of broadened joints, ESR or C reactive protein (CRP), and patient overall assessment. The DAS-28 territory is from 0 to 9.4. Ailment activity examination was performed using the SLE contamination activity record (SLEDAI). The most outrageous score in this system is 105, and we considered extension in score greater than 3 to 4 concentrations as a delicate/moderate flare and scores greater than 12 centers as serious flare. Lab assessments included; Liver chemicals, complete, ESR, CRP, serum creatinine, blood urea, rheumatoid variable (RF), serum hostile to cyclic citrullinated peptide, against dsDNA immune response, supplement C3 and C4, IL 17, complete urinalysis, egg whites/creatinine proportion, and lipid profile Radiograph evaluation was Plain x-pillar on two hands (postero-front view). Capillaroscopy appraisal was applied to the second to fifth fingers (excepting the thumbs) of two hands. Verbal and formed consent was obtained from individuals who were welcome to partake in the audit. The data's privacy was guaranteed. SPSS was used to collect and analyze the data, mean ± SD and looked at by understudy t test and ANOVA test followed by post-hoc examination while ostensible information was communicated in the type of recurrence (rate) and analyzed by Chi^2-test. Level of certainty was kept at 95% and subsequently, P esteem was viewed as critical if less than 0.05.

3. Results

Different studied groups had insignificant differences as regard baseline data ($P > 0.05$) with exception of duration of disease was significantly longer among those patients with RA in comparison to those with SLE (Table 1). Both groups had insignificant differences as regard baseline laboratory with exception of patients with RA had significantly higher hemoglobin level (12.78 ± 0.78 vs. 10.38 ± 1.72 (g/dl); $P < 0.001$), complement C3 [2.59 ± 0.32 vs. 0.66 ± 0.64 (mg/dl); $P < 0.001$] and complement C4 (3.46 ± 0.14 vs. 0.30 ± 0.16 (mg/dl); $P < 0.001$) while patients with SLE had significantly higher serum creatinine (Table 2). Patients with RA

Table 1. Baseline data of studied patients.

	RA group ($n = 50$) [n (%)]	SLE group ($n = 30$) [n (%)]	Control group ($n = 30$) [n (%)]	P value
Age (year)	47.05 ± 12.84	49.27 ± 11.39	49.20 ± 5.57	0.11
Disease duration	8.06 ± 4.55	3.36 ± 2.51		**<0.001**
BMI (kg/m^2)	27.13 ± 7.12	28.77 ± 5.70	27.01 ± 6.87	0.28
Sex				0.40
Male	9 (18)	5 (16.7)	6 (20)	
Female	41 (82)	25 (83.3)	24 (80)	
Marital status				0.19
Single	5 (10)	0	0	
Married	45 (90)	30 (100)	30 (100%)	
Occupation				0.45
Housewife	42 (84)	25 (83.3)	24 (80)	
Employee	8 (16)	5 (16.7)	6 (20)	
Family history	6 (12)	7 (24.1)	5 (16.7)	0.50

Table 2. Baseline laboratory data of the studied groups of patients.

	RA ($n = 50$)	SLE ($n = 30$)	P value
Hemoglobin (g/dl)	12.78 ± 0.78	10.38 ± 1.72	**<0.001**
Leucocytes (10^3/ul)	5.73 ± 2.79	5.45 ± 1.14	0.67
Platelets (10^3/ul)	283.27 ± 92.11	279.50 ± 46.95	0.67
Creatinine (mg/dl)	0.79 ± 0.22	1.87 ± 0.66	**<0.001**
Urea (mg/dl)	8.90 ± 3.44	36.98 ± 8.98	**0.01**
Complement 3 (mg/dl)	2.59 ± 0.32	0.66 ± 0.64	**<0.001**
Complement 4 (mg/dl)	3.46 ± 0.14	0.30 ± 0.16	**<0.001**
CRP (mg/dl)	30.61 ± 12.22	24.05 ± 1.67	0.30
ESR (ml/h)	75.33 ± 33.88	78.95 ± 2.78	0.08
Albumin (mg/dl)	31.30 ± 4.41	38.10 ± 3.35	0.38
AST (U/l)	46.49 ± 13.67	45.65 ± 13.87	0.43
ALT (U/l)	36.24 ± 10.78	31.75 ± 12.30	0.30

had significantly higher IL-17 in comparison to those with SLE ($P < 0.001$) and the control group ($P < 0.001$). Also, patients with SLE had significantly higher IL-17 in comparison to the control group ($P = 0.01$) (Table 3). In patients with SLE, IL-17 had significant positive correlations with CRP ($r = 0.60$, $P < 0.001$), ESR ($r = 0.56$, $P = 0.01$), and SLEDAI

Table 3. Serum interleukin-17 among the studied groups.

	Interleukin-17 (pg/ml)
Groups	
RA group	158.42 ± 21.40
SLE group	75.75 ± 3.27
Control group	41.58 ± 6.57
Significance	
P 1	**<0.001**
P 2	**<0.001**
P 3	**0.01**

Bold value indicates <0.001 was statistically highly significant differences.

Table 4. Correlation of interleukin-17 with other variables in patients' groups.

	RA group	SLE group
Age (years)	0.09 (0.39)	0.05 (0.66)
Body mass index (kg/m^2)	−0.02 (0.98)	−0.06 (0.57)
Duration of disease (years)	0.08 (0.48)	−0.08 (0.48)
Hemoglobin (g/dl)	0.03 (0.78)	0.19 (0.08)
Leucocytes (10^3/ul)	0.13 (0.24)	−0.03 (0.78)
Platelets (10^3/ul)	0.03 (0.12)	−0.02 (0.80)
Creatinine (mg/dl)	−0.08 (0.45)	0.14 (0.20)
Urea (mg/dl)	0.02 (0.98)	0.16 (0.14)
Complement 3 (mg/dl)	−0.01 (0.90)	**−0.34 (0.03)**
Complement 4 (mg/dl)	0.05 (0.63)	**−0.40 (0.02)**
CRP (mg/dl)	**0.76 (<0.001)**	**0.60 (<0.001)**
ESR (ml/h)	**0.70 (<0.001)**	**0.56 (0.01)**
Albumin (mg/dl)	0.12 (0.27)	0.06 (0.55)
AST (U/l)	0.04 (0.70)	0.07 (0.51)
ALT (U/l)	0.10 (0.37)	−0.03 (0.76)
SLEDAI		**0.67 (<0.001)**
Disease activity score 28	**0.70 (<0.001)**	

Bold value indicates <0.001 was statistically highly significant differences.

Table 5. Serum interleukin-17 in patients with rheumatoid arthritis based on severity.

	Interleukin-17 (pg/ml)
Severity of RA	
Low/moderate activity ($n = 15$)	141.40 ± 3.15
High activity ($n = 35$)	165.70 ± 21.76
P value	**<0.001**

Bold value indicates <0.001 was statistically highly significant differences.

Table 6. Serum interleukin-17 in patients with systemic lupus erythematosus based on severity.

	Interleukin-17 (pg/ml)
Severity of SLE	
Mild to moderate activity ($n = 13$)	72.66 ± 1.43
Severe activity ($n = 17$)	78.11 ± 2.04
P value	**<0.001**

Bold value indicates <0.001 was statistically highly significant differences.

($r = 0.67$, $P < 0.001$) but it has a negative correlation with complement 3 ($r = -0.34$, $P = 0.03$) and complement 4 ($r = -40$, $P = 0.02$). All other correlations were insignificant ($P > 0.05$) (Tables 4–6). Both groups had insignificant differences as regard to density ($P = 0.09$). The control group had significantly higher capillary density (<0.001) in comparison to RA group. Control group had significantly higher capillary density ($P = 0.01$) in comparison to SLE group (Table 7). Patients with high activity had significantly higher frequency of elongated capillaries, enlarged capillaries, subpapillary plexus, hemorrhage and avascular area in comparison to those with low/moderate activity (Table 8). Patients with severe activity had significantly higher frequency of tortuous capillaries, elongated capillaries, disorganized capillaries, hemorrhage and avascular area in comparison to those with mild/moderate activity. Also, patients with severe activity had significantly lower capillary density in comparison to those with mild/moderate activity (3.01 ± 0.56 vs. 5.09 ± 1.23 (mm); $P = 0.02$) (Table 9, Figures 1-3).

4. Discussion

NFC is the technique for decision for breaking down microvascular irregularities in connective tissue illnesses (CTE) in light of the fact that slim changes in the nailfold are deep rooted in numerous CTEs. NFC is very helpful in recognizing different nail overlay irregularities as it is the highest quality level for distinguishing microvascular changes in the nail fold.[5]

In the current review, we meant to explore interleukin 17 and microcirculation in blood serum of

Table 7. Nail fold capilloroscopy findings in the studied groups.

	RA ($n = 50$) [n (%)]	SLE ($n = 30$) [n (%)]	Control group ($n = 30$) [n (%)]	*P*1	*P*2	*P*3
Tortous capillaries	33 (66)	20 (66.7)	16 (53.3)	0.45	0.09	0.34
Bushy capillaries	28 (56)	13 (43.3)	2 (6.7)	**0.03**	**<0.001**	**<0.001**
Elongated capillaries	16 (32)	18 (60)	3 (10)	**0.01**	**<0.001**	**<0.001**
Enlarged capillaries	14 (28)	13 (43.3)	5 (16.7)	0.45	0.07	**0.01**
Subpapillary plexus	18 (36)	12 (40)	3 (10)	0.21	0.54	**<0.001**
Disorganized capillaries	21 (4%)	19 (63.%)	3 (10)	**<0.001**	**<0.001**	**<0.001**
Haemorrhage	12 (24)	16 (53.3)	4 (13.3)	**<0.001**	**0.04**	**<0.001**
Avascular area	14 (28)	14 (46.7)	3 (10)	**<0.001**	**<0.001**	**<0.001**
Density (mm)	3.09 ± 0.67	4.72 ± 0.26	6.07 ± 0.73	0.09	**<0.001**	**0.01**

Bold value indicates <0.001 was statistically highly significant differences.

Table 8. Nail fold capilloroscopy findings in patients with rheumatoid arthritis based on severity.

	Disease activity score 28		*P* value
	High activity ($n = 35$) [n (%)]	Low/moderate activity ($n = 15$) [n (%)]	
Tortous capillaries	25 (71.4)	8 (53.3)	0.18
Bushy capillaries	21 (60)	7 (46.7)	0.28
Elongated capillaries	13 (37.1)	3 (20)	**0.03**
Enlarged capillaries	12 (34.3)	2 (13.3)	**<0.001**
Subpapillary plexus	16 (42.9)	3 (20)	**0.03**
Disorganized capillaries	14 (40)	7 (46.7)	0.44
Haemorrhage	12 (34.3)	0	**<0.001**
Avascular area	12 (34.3)	2 (13.3)	**<0.001**
Density (mm)	3.09 ± 0.59	3.08 ± 0.86	0.94

Bold value indicates <0.001 was statistically highly significant differences.

Table 9. Nail fold capilloroscopy findings in systemic lupus erythematosus group based on severity.

	SELDAI		*P* value
	Severe activity ($n = 17$) [n (%)]	Mild to moderate activity ($n = 13$) [n (%)]	
Tortous capillaries	17 (100)	3 (23.1)	**<0.001**
Bushy capillaries	6 (35.3)	7 (53.8)	0.26
Elongated capillaries	17 (100)	1 (7.8)	**<0.001**
Enlarged capillaries	7 (41.2)	6 (46.2)	0.53
Subpapillary plexus	8 (47.1)	4 (30.8)	0.30
Disorganized capillaries	15 (88.2)	4 (30.8)	**<0.001**
Haemorrhage	12 (70.6)	4 (30.8)	**0.03**
Avascular area	14 (82.35)	0	**<0.001**
Density (mm)	3.01 ± 0.56	5.09 ± 1.23	**0.02**

Bold value indicates <0.001 was statistically highly significant differences.

patients with rheumatoid joint pain and lupus erythematosus and examine their relationship with sickness action in Upper Egypt. A sum of 50 patients with rheumatoid joint pain and 30 patients with SLE took part in the review. The fundamental finding of the ongoing review was that patients with SLE or rheumatoid joint pain (RA) had altogether higher interleukin 17 (IL-17) levels than the benchmark group. Patients with rheumatoid joint inflammation had essentially higher IL-17 levels than those with SLE ($P < 0.001$) and controls ($P < 0.001$). Besides, SLE patients had essentially higher interleukin-17 levels than controls ($P = 0.01$).

Steady with the ongoing review, Shen *et al.*[6] led a meta-investigation with a sum of twenty correlations between SLE populaces and sound controls. The joined outcomes showed that SLE patients had expanded degrees of coursing IL-17.

Moreover, in the ongoing concentrate in SLE patients, IL-17 showed huge positive connections with CRP ($r = 0.60$, $P < 0.001$), ESR ($r = 0.56$, $P = 0.01$), and the SLEDAI ($r = 0.67$, $P < 0.001$).)) be that as it may, has a negative connection with supplement 3 ($r = -0.34$, $P = 0.03$) and supplement 4 ($r = -40$, $P = 0.02$). Any remaining relationships were irrelevant ($P > 0.05$).

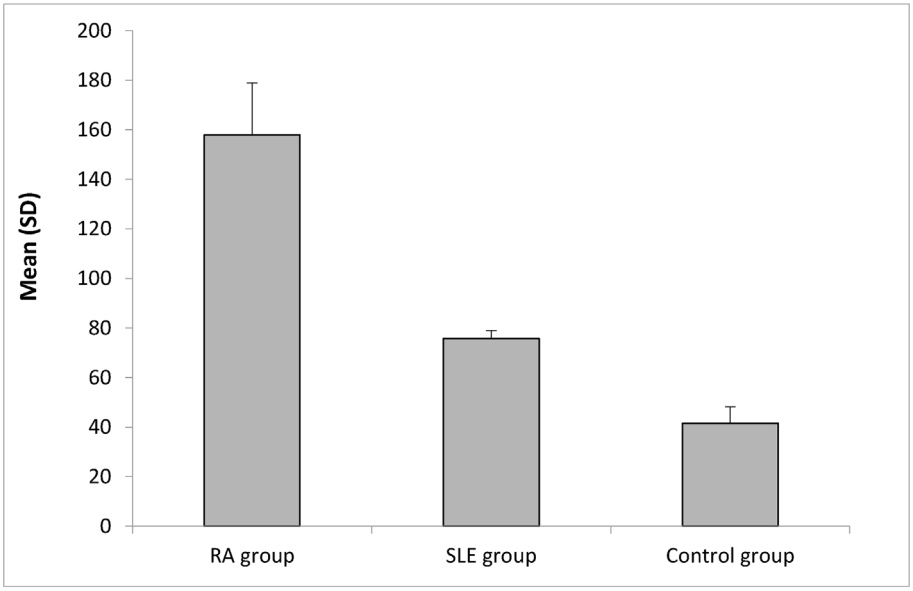

Figure 1. Mean level of interleukin-17 in the studied groups.

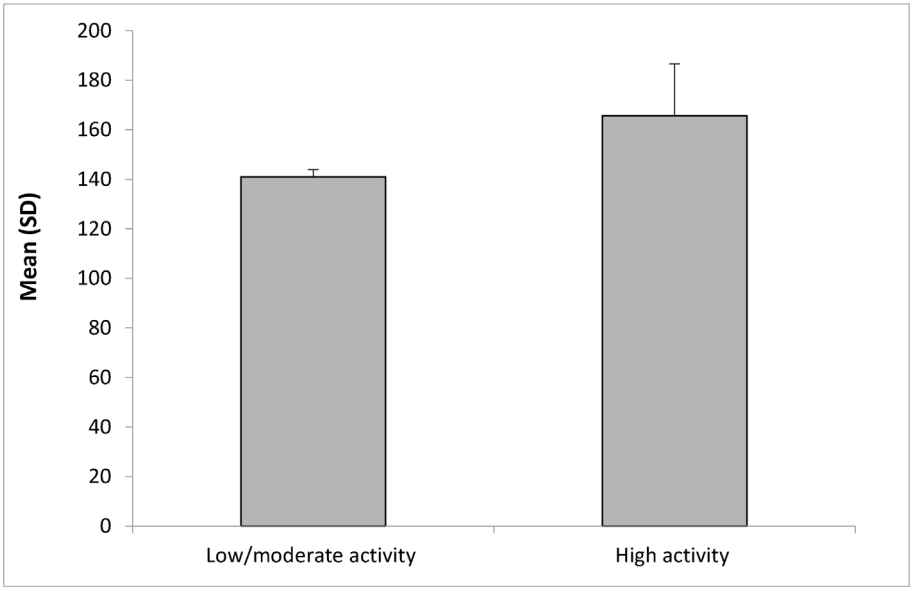

Figure 2. Mean level of interleukin-17 in patients with rheumatoid arthritis based on severity.

One distribution revealed a relationship among SLEDAI and serum IL-17 Doreau *et al.*,[7] while another distribution detailed an affiliation just in patients without renal sickness Wong *et al.*[8]

In this review, patients with serious action in the SLE bunch had essentially higher interleukin-17 levels than patients with gentle/moderate action (78.11 ± 2.04 versus 72.66 ± 1.43 (pg/ml); $P < 0.001$). Reliable to these discoveries, Zhou *et al.*[9] proposed that many examinations have related coursing levels of IL-17 with sickness movement, supporting the significant job of IL-17 in the pathogenesis of SLE.

In this review, we likewise found that patients with rheumatoid joint pain had essentially higher IL-17 levels than the benchmark group. Notwithstanding, covers IL-17 levels are conflicting: a few late examinations have shown that plasma IL-17 levels in patients with rheumatoid joint pain do not contrast from those in solid controls. Zayat *et al.*,[10] Vuklic *et al.*,[11] Wang *et al.*[12] While different examinations have detailed higher serum levels of IL-17 in patients with rheumatoid joint pain, Van Roon *et al.*,[13] and Taylor *et al.*[14]

Additionally, steady with these discoveries in regards to illness action, Al-Saadani *et al.*,[15] detailed

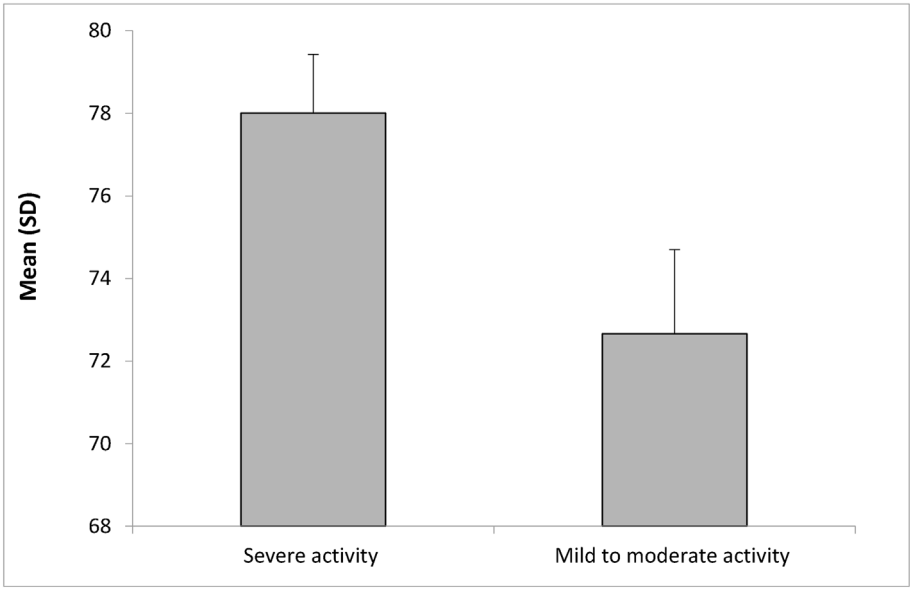

Figure 3. Mean level of interleukin-17 in patients with systemic lupus erythematosus based on severity.

that serum IL-17 levels were fundamentally connected with sickness action utilizing DAS-28. DAS-28 is a measurably determined record that incorporates delicate joint count, enlarged joint count, ESR, and by and large sickness movement. Al-Ghatani[16] showed the significant job of serum IL-17 in the pathogenesis of the horrendous and fiery example normal for rheumatoid joint pain.

Our discoveries that IL-17 levels are expanded in patients with SLE and RA support the job of IL-17 in the pathophysiology of SLE and RA. Given the conceivable relationship between IL-17 and immune system sicknesses, polymorphisms in IL-17 qualities that might impact IL-17 articulation have been explored as potential reasons for immune system illnesses by Jin *et al.*,[17] and Li and Bai.[18]

Concerning the relationship between Drove movement and NFC, our outcomes were predictable with those of Liu *et al.*,[19] appearance expanded illness action surveyed by SLEDAI, Foundational Lupus Movement Scales, and European Agreement Action Scales in SLE patients with NFC changes.

Then again, in the investigation of Ali *et al.*,[9] led on patients with rheumatoid joint pain, there was no critical connection between movement boundaries.

4.1. Conclusion

SLE or RA patients had significantly higher IL-17 in comparison to the control group. NFC, patients with RA had significantly higher frequency of bushy capillaries while patients with SLE had a significantly higher frequency of elongated capillaries, disorganized capillaries, hemorrhage and avascular area.

Conflicts of interest

There are no conflicts of interest.

References

1. Cutolo M, Paolino S, Smith V. Nailfold capillaroscopy in rheumatology: ready for the daily use but with care in terminology. *J Clin Med*. 2019;38:2293–2297.
2. Hemdan N, Birkenmeier G, Wichmann G, et al. Interleukin-17-producing T helper cells in autoimmunity. *Autoimmun Rev*. 2010;9:785–792.
3. López-Benjume B, Rodríguez-Pintó I, Amigo M, et al. Eculizumab use in catastrophic antiphospholipid syndrome (CAPS): descriptive analysis from the 'CAPS Registry'. *Autoimmun Rev*. 2022;21:103055.
4. Lambova SN, Müller-Ladner U. Capillaroscopic pattern in systemic lupus erythematosus and undifferentiated connective tissue disease: what we still have to learn? *Rheumatol Int*. 2013;33:689–695.
5. Chanprapaph K, Fakprapai W, Limtong P, et al. Nailfold capillaroscopy with USB digital microscopy in connective tissue diseases: a comparative study of 245 patients and healthy controls. *Front Med*. 2021;8:52–59.
6. Shen H-H, Fan Y, Wang Y, et al. Elevated circulating interleukin-17 levels in patients with systemic lupus erythematosus: a meta-analysis. *Immunol Invest*. 2020;49:662–675.
7. Doreau A, Belot A, Bastid J, et al. Interleukin 17 acts in synergy with B cell-activating factor to influence B cell biology and the pathophysiology of systemic lupus erythematosus. *Nat Immunol*. 2009;10:778–785.
8. Wong C, Lit L, Tam L, et al. Hyperproduction of IL-23 and IL-17 in patients with systemic lupus erythematosus: implications for Th17-mediated inflammation in auto-immunity. *Clin Immunol*. 2008;127:385–393.
9. Ali AM, Hamza SM, Aboud F, et al. Ailfold capillaroscopic changes in Egyptian patients with psoriatic arthritis in

comparison to rheumatoid arthritis. *Egypt Rheumatol.* 2019;41: 303–307.
10. Zayat AS, Ellegaard K, Conaghan P, et al. The specificity of ultrasound-detected bone erosions for rheumatoid arthritis. *Ann Rheum Dis*. 2015;74:897–903.
11. Vukelic M, Li Y, Kyttaris V د. Novel treatments in lupus. *Front Immunol*. 2018;9:2658–2675.
12. Wang Y, Zhao R, Gu Z, Dong C, Guo G, Li L. Effects of glucocorticoids on osteoporosis in rheumatoid arthritis: a systematic review and meta-analysis. *Osteoporos Int*. 2020;8: 1–9.
13. Van Roon AM, Huisman CC, Van Roon A, et al. Abnormal nailfold capillaroscopy is common in patients with connective tissue disease and associated with abnormal pulmonary function tests. *J Rheumatol*. 2019;46:1109–1116.
14. Taylor TC, Li Y, Li D, et al. Arid5a mediates an IL-17-dependent pathway that drives autoimmunity but not anti-fungal host defense. *bioRxiv*. 2022;8:75–79.
15. Al-Saadany HM, Hussein MS, Gaber R, et al. Th-17 cells and serum IL-17 in rheumatoid arthritis patients: correlation with disease activity and severity. *Egypt Rheumatol*. 2016;38: 1–7.
16. Al-Gahtani SN. A review of systemic lupus erythematosus (SLE): symptoms, risk factors, treatment, and health related quality of life issues. *Open J Rheumatol Autoimmune Dis*. 2021; 11:115–143.
17. Jin L, Bai R, Zhou J, et al. Association of serum T cell immunoglobulin domain and Mucin-3 and interleukin-17 with systemic lupus erythematosus. *Med Sci Monit Basic Res*. 2018;24:168–176.
18. Lee YH, Bae S. Associations between circulating IL-17 levels and rheumatoid arthritis and between IL-17 gene polymorphisms and disease susceptibility: a meta-analysis. *Postgrad Med J*. 2017;93:465–471.
19. Liu X, Sun S, Liu D. IL-17D: a less studied cytokine of IL-17 family. *Int Arch Allergy Immunol*. 2020;181:618–623.