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ORIGINAL ARTICLE

Impact of Pulse Steroid on the Level of IL-17 in Active Lupus Nephritis

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Abstract

Background: ***Lupus nephritis (LN) is one of the most severe conditions associated with systemic lupus erythematosus (SLE). Interleukin-17 is one of the main inflammatory cytokines linked to LN, but the relationship is unclear.***

Aim of the work: ***Examine the correlation between the histopathological findings, therapy response, and IL-17 serum level in cases with active LN and following pulse therapy.***

Patients and methods: ***The current research divided 60 subjects into active LN, inactive LN, and control groups. Every individual underwent comprehensive history taking, a comprehensive physical examination, an evaluation of disease activity using the SLE disease activity index (SLEDAI), and clinical tests (including serum IL-17 levels). After the induction therapy, cases with active LN were followed up to determine the fate after treatment and to determine changes in the laboratory and urinary tested parameters.***

Results: ***Compared to the inactive LN and control groups, the active LN group had statistically significantly higher IL-17 levels. The IL-17 level in the active LN showed a statistically significant decrease after treatment compared to before treatment.***

Conclusion: ***In cases of SLE, a higher serum IL-17 level may help predict the level of LN activity. Active LN patients' serum IL-17 levels are predictive of treatment efficacy.***

Keywords: SLE; LN; IL-17; Remission; Steroids

1. Introduction

Systemic lupus erythematosus (SLE) is an autoimmune disease that affects several systems. Up to 70% of children and 30–60% of adults with SLE develop lupus nephritis (LN) during the disease period .[1]

The pathogenesis of LN is a complicated process. The development of immune complexes and the generation of autoantibodies are the distinguishing features of the pathogenesis, even though the exact mechanisms are still unclear .[2] In addition, a large number of cytokines are involved .[3]

The CD4+ T helper cell subtype Th17 has shown hyperactivation. It is involved in neuropsychiatric, cutaneous, and LN SLE manifestations, so it is linked to disease activity and death .[4]

Th17 cells secrete IL-17, a pleiotropic proinflammatory cytokine. It is associated with psoriasis, rheumatoid arthritis (RA), IBD, SLE, and systemic sclerosis .[5]

The goal of the current investigation was to study the IL-17 serum level in active LN cases and follow pulse therapy in relation to histopathological findings and therapeutic response.

2. Patients and methods

Egypt's Al-Azhar University Hospital's internal medicine department conducted this six-month prospective observational and analytical study.

Out of 60 participants, group 1 had 20 active LN cases, group 2 had 20 inactive LN cases, and group 3 had 20 healthy controls.

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The study included both male and female adults who had (LN) more than 18 months ago and whose biopsy results confirmed the diagnosis[6]

The following features led to the exclusion of the cases: end-stage renal disease (ESRD), pregnant and lactating females, cases with bleeding disorders, and cases receiving biologic-blocking antibodies.

Return serum creatinine and urine protein to normal (≤ 0.5 g/day) indicated complete remission (CR) .[7] Partial remission (PR) was defined as either a 10% decrease in creatinine clearance or ≥ 50% decrease in urinary protein: creatinine ratio to < 1.0 (if baseline ratio ≤ 3.0) or < 3.0 (if baseline ratio > 0.3). [8]

This research is conducted in compliance with Helsinki Standards as revised in 2013 .[9] The study was carried out after receiving approval from the Al-Azhar University Faculty of Medicine's local ethics committee and signed informed consent from all participating individuals.

The following procedures were applied to the cases: a clinical examination, a history-taking session that included information about the patient's demographics, the illness's history, details about the induction therapy, follow-up (remission duration and time to achieve remission), renal flare-ups, infections and non-infection complications requiring hospital admission, and related co-morbidities.

Every case in the study was given a Systemic Lupus Erythematosus Disease Activity Index (SLEDAI), with cases deemed inactive if their score was less than 4, moderate disease activity if it was between 4 and 8, severe disease activity if it was between 9 and 12, and inactive if it was greater than 12 .[10]

CBC, CRP, serum creatinine, erythrocyte sedimentation rate (ESR), liver functions (AST-ALT), serum anti-nuclear antibody (ANA), and albumin creatinine ratio were among the laboratory tests performed.

The Human Interleukin 17 ELISA kit, produced by Sun Red Biological Technology Co., Ltd. (cat #: 201-12- 0143, Sun Red biotechnology company, Shanghai, China), measured IL-17 serum levels.

Laboratory tests (including the serum level of IL-17) and urine analysis were performed again in the cases with active LN after treatment was completed.

1. Statistical evaluation

The data was coded, processed, and analyzed using the Statistical Package for Social Sciences (SPSS) 26 for Windows® (IBM, SPSS Inc, Chicago, IL, USA). The number (frequency) and percentage of qualitative data were displayed. The Monte-Carlo test, also known as the Chi-Square test, made group comparisons easier. The Kolmogorov-Smirnov test examined the normality of quantitative data. The data was displayed as either the median and range or the median ± SD.

Data not distributed normally was tested using the Mann-Whitney U-test. The independent samples t-test (students' t-test) was used to compare two groups with normally distributed quantitative variables.

If the quantitative variables were normally distributed, three or more groups were compared using one-way analysis of variance (one-way ANOVA). If the data were abnormally distributed, the Kruskal-Wallis test was employed.

If the data did not follow a normal distribution, the Wilcoxon-Signed rank test was employed. In contrast, two dependent groups with normally distributed quantitative variables were compared using the paired samples t-test.

When comparing two dependent groups with categorical data, McNamara's and the marginal homogeneity tests were employed (2x2 tables and more than 2x2 tables, respectively).

The Youden index J was used to determine the ideal cutoff value of IL-17 to distinguish between different groups. P values under 0.05 are significant for all tests.

3. Results

Sixty subjects were divided into three groups for the current study: twenty cases of active LN were included in group 1, twenty cases of inactive LN were included in group 2, and twenty healthy control subjects were included in group 3.

The onset and duration of SLE were not statistically different between active and inactive LN cases. The age of onset of LN in cases with active LN was 23 years with range between 16 and 48 years, the duration of LN ranged between 1 and 5 years. The median SLEDAI was 12 with range between 4 and 12. Table 1.

Table 1. Comparison of the demographic and clinical data between the study groups

VARIABLES	GROUP 1 (ACTIVE LN) (N=20)	GROUP 2 (INACTIVE LN) (N=20)	GROUP 3 (CONTROL GROUP) (N=20)	TEST OF SIGNIFICANCE	MULTIPLE COMPARISONS
AGE (YEARS) [MEAN ± SD]	29 ± 9.34	30.40 ± 9.98	31.65 ±10.34	F = 0.359 P = 0.700	P_1=0.896 P_2 = 0.676 P_3= 0.916
GENDER [N (%)]				MC = 1.600	P_1= 1
MALE	4 (20%)	4 (20%)	7 (35%)	P= 0.439	P_2 = 0.762

FEMALE	16 (80%)	16 (80%)	13 (65%)		P_3= 0.762
			FAMILY HISTORY [N (%)]	MC = 4.444	P_1=0.460
NEGATIVE	18 (90%)	16 (80%)	20 (100%)	P= 0.108	P_2 = 0.434
POSITIVE	2 (10%)	4 (20%)	0 (0%)		P_3= 0.218
AGE OF ONSET OF SLE (YEARS) [MEDIAN (RANGE)]	21 (16-45)	23 (17 – 47)		z = - 0.928 P = 0.640	
DURATION OF SLE (YEARS) [MEDIAN (RANGE)]	4 (1-6)	3 (1-19)		z = - 1.642 P = 0.210	
AGE OF ONSET OF LN (YEARS) [MEDIAN (RANGE)]	23 (16-48)				
DURATION OF LN (YEARS) [MEDIAN (RANGE)]	3 (1-5)				
SLEDAI [MEDIAN (RANGE)]	12 (4-12)				

X^2: PEARSON CHI-SQUARE TEST MC: MONTE-CARLO TEST
F: ONE-WAY ANOVA TEST Z: MANN WHITNEY U-TEST
P: GENERAL INTERGROUP SIGNIFICANCE
P1: COMPARISON BETWEEN GROUP 1 (ACTIVE LN) AND GROUP 2 (INACTIVE LN)
P2: COMPARISON BETWEEN GROUP 1 (ACTIVE LN) AND GROUP 3 (CONTROL GROUP)
P3: COMPARISON BETWEEN GROUP 2 (INACTIVE LN) AND GROUP 3 (CONTROL GROUP)
*: STATISTICALLY SIGNIFICANT (P≤ 0.05)

The control group's haemoglobin level was significantly higher than that of the active LN group and the inactive LN group. The levels of ALT and AST were significantly higher in the active LN group compared to the control group (Table 2).

Both the active LN and inactive LN groups had significantly higher creatinine and ACR levels than the control group. The active LN group had statistically significantly lower C3 and C4 than the control and inactive LN groups (Table II).

The C-reactive protein level was significantly lower in the control group compared to the active LN group and the inactive LN group. The active LN group had significantly higher urea levels and ESRs compared to the control and inactive LN groups. The ESR and urea levels of the inactive LN cases were also significantly higher than the control group's (Table 2).

Table 2 shows that the median (range) of IL-17 level was 246 pg/ml (88 - 412), 57 105 pg/ml (11 - 230) and 36 pg/ml (4.6 – 100.3) in the active LN, inactive LN and control group respectively. As compared to the inactive LN and control groups, the active LN group's median IL-17 was statistically significantly higher. Additionally, compared to the control group, the median IL-17 level was statistically significantly higher in the inactive LN cases (Table 2).

Table 2. Comparison of the basal laboratory data in the study groups

VARIABLES	GROUP 1 (ACTIVE LN) (N=20)	GROUP 2 (INACTIVE LN) (N=20)	GROUP 3 (CONTROL GROUP) (N=20)	SIGNIFICANCE TEST	INTERGROUP SIGNIFICANCE
HEMOGLOBIN (GM/DL) [MEAN ± SD]	9.66 ± 2.07	9.96 ± 1.29	11.60 ± 1.56	F = 8.511 P = 0.001*	P_1= 0.710 P_2 = 0.001* P_3= 0.008*
PLTS(10^3/ML) [MEAN ± SD]	215 (135 – 560)	241 (148 – 420)	239 (157 – 422)	KW = 0.833 P = 0.659	P_1= 0.913 P_2 = 0.990 P_3= 0.925
WBCS (10^3/ML) [MEAN ± SD]	7.45 (3.8 – 19)	6.55 (4.4 – 14.6)	6.05 (4.6 – 10.3)	KW = 0.437 P = 0.804	P_1=0.836 P_2 = 0.264 P_3 = 0.576
ALT (U/L) [MEAN ± SD]	33 (16 – 80)	27 (10 – 69)	20 (10 – 40)	KW = 7.535 P =0.023 *	P_1 = 0.416 P_2= 0.024* P_3= 0.329
AST (U/L) [MEAN ± SD]	36 (10 – 77)	30 (11 – 53)	23 (11 – 42)	KW = 6.831 P =0.033 *	P_1 = 0.649 P_2= 0.021* P_3= 0.155
CREATININE (MG/DL) [MEAN ± SD]	2.3 (0.9 – 4.9)	1.05 (0.8 – 2.8)	0.8 (0.4 – 1)	KW= 38.637 P <0.001 *	P_1<0.001* P_2 <0.001* P_3= 0.151
UREA (MG/DL) [MEAN ± SD]	87 (22 – 146)	44 (29 – 100)	24 (14 – 40)	KW= 36.992 P <0.001 *	P_1<0.001* P_2 <0.001* P_3= 0.014*
C3 (MG/DL) [MEAN ± SD]	25 (2 – 71)	92 (45 – 188)	117 (88 – 133)	KW= 38.267 P <0.001 *	P_1<0.001* P_2 <0.001* P_3= 0.518
C4 (MG/DL) [MEAN ± SD]	8 (2 – 18)	23 (9 – 50)	23 (18 – 36)	KW = 36.937 P <0.001 *	P_1<0.001* P_2 <0.001* P_3= 0.109
CRP (MG/DL) [MEAN ± SD]	7 (0- 117)	7.5 (0- 106)	1 (0- 12)	KW = 13.001 P =0.002 *	P_1 = 0.692 P_2= 0.020*

					P_3= 0.031*
ESR (MG/DL) [MEAN ± SD]	39 (23- 79)	29 (11- 58)	18 (3- 40)	KW = 23.111 P <0.001 *	P_1 = 0.021* P_2<0.001* P_3= 0.009*
ACR (MG/DL) [MEAN ± SD]	2683 (1079 – 5062)	410 (79 – 2047)	23 (6 – 864)	KW = 42.499 P <0.001 *	P_1<0.001* P_2 <0.001* P_3= 0.196
IL-17 (PG/ML) [MEDIAN (RANGE)]	246 (88 - 412)	105 (11 - 230)	36 (4.6 – 100.3)	KW= 36.278 P <0.001 *	P_1<0.001* P_2<0.001* P_3= 0.004*
ANA [N (%)]				MC = 39.231	P_1= 1
NEGATIVE	3 (15%)	3 (15%)	20 (100%)	P< 0.001*	P_2 < 0.001*
POSITIVE	17 (75%)	17 (75%)	0 (0%)		P_3< 0.001*
			ANTI-DSDNA [N (%)]	MC = 23.384	P_1=0.246
NEGATIVE	8 (40%)	6 (30%)	20 (100%)	P< 0.001*	P_2 < 0.001*
POSITIVE	12 (60%)	14 (70%)	0 (0%)		P_3< 0.001*

MC: MONTE-CARLO TEST F: ONE-WAY ANOVA TEST KW: KRUSKAL WALLIS TEST
P: GENERAL INTERGROUP SIGNIFICANCE
P1: COMPARISON BETWEEN GROUP 1 (ACTIVE LN) AND GROUP 2 (INACTIVE LN)
P2: COMPARISON BETWEEN GROUP 1 (ACTIVE LN) AND GROUP 3 (CONTROL GROUP)
P3: COMPARISON BETWEEN GROUP 2 (INACTIVE LN) AND GROUP 3 (CONTROL GROUP)
**: STATISTICALLY SIGNIFICANT (P≤ 0.05)*

As related to other groups, cases with active LN had a statistically higher prevalence of protein, granular casts, and RBCs in their urine analysis (Table 3).

Table 3. Comparison of urine analysis between the study groups

VARIABLES	GROUP 1 (ACTIVE LN) (N=20)	GROUP 2 (INACTIVE LN) (N=20)	GROUP 3 (CONTROL GROUP) (N=20)	TEST OF SIGNIFICANCE	MULTIPLE COMPARISONS
PROTEIN [N (%)]				MC = 39.995	P_1= 0.002*
NEGATIVE	0 (0%)	13 (65%)	16 (80%)	P< 0.001*	P_2 < 0.001*
+	3 (15%)	4 (20%)	4 (20%)		P_3= 0.025*
++	9 (45%)	2 (10%)	0 (0%)		
+++	8 (40%)	1 (5%)	0 (0%)		
			GRANULAR CAST [N (%)]	MC = 23.384	P_1< 0.001*
NEGATIVE	8 (40%)	19 (95%)	20 (100%)	P< 0.001*	P_2 < 0.001*
+	5 (25%)	1 (5%)	0 (0%)		P_3= 0.128
++	3 (15%)	0 (0%)	0 (0%)		
+++	4 (20%)	0 (0%)	0 (0%)		
			HYALINE CAST [N (%)]	MC = 8.571	P_1= 0.068
NEGATIVE	16 (8%)	20 (100%)	20 (100%)	P= 0.073	P_2 = 0.068
+	1 (5%)	0 (0%)	0 (0%)		P_3= 1
++	3 (15%)	0 (0%)	0 (0%)		
RBCS [N (%)]				χ^2 = 6.941	P_1= 0.003*
NEGATIVE	5 (25%)	13 (65%)	11 (55%)	P= 0.031*	P_2 = 0.034*
POSITIVE	15 (75%)	7 (35%)	9 (45%)		P_3= 0.186

X^2: PEARSON CHI-SQUARE TEST MC: MONTECARLO TEST
P: GENERAL INTERGROUP SIGNIFICANCE
P1: COMPARISON BETWEEN GROUP 1 (ACTIVE LN) AND GROUP 2 (INACTIVE LN)
P2: COMPARISON BETWEEN GROUP 1 (ACTIVE LN) AND GROUP 3 (CONTROL GROUP)
P3: COMPARISON BETWEEN GROUP 2 (INACTIVE LN) AND GROUP 3 (CONTROL GROUP)
*: STATISTICALLY SIGNIFICANT (P≤ 0.05)

In cases with active LN, with follow up, 1 case died, 2 cases showed no remission while 17 cases (85%) showed remission (Data not shown).

> 137.6 pg/ml was the optimal cutoff point for IL-17 to distinguish between Active LN and Inactive LN, with high sensitivity (80%) and moderate specificity (70%) (Table 4) (Figure 1). The best cutoff point of IL-17 to differentiate between (Inactive LN group) and Control group was > 33 ng/ml, with high sensitivity (85%) and specificity (75%) (Table 4) (Figure 2).

Table 4. Predictive value of IL-17 to differentiate between (Active LN group) and (Inactive LN group)/ Group 2 (Inactive LN) and Control group

	GROUP 1 (ACTIVE LN) AND GROUP 2 (INACTIVE LN)	GROUP 2 (INACTIVE LN) AND CONTROL GROUP
AUC	0.828	0.855
CUT OFF POINT	> 137.6	> 48.75
SENSITIVITY	80%	85 %
SPECIFICITY	70%	75 %
PPV	78%	80 %
NPV	72%	75 %
ACCURACY	76%	85 %
P	< 0.001*	< 0.001*

AUC: AREA UNDER CURVE, PPV: POSITIVE PREDICTIVE VALUE, NPV: NEGATIVE PREDICTIVE VALUE

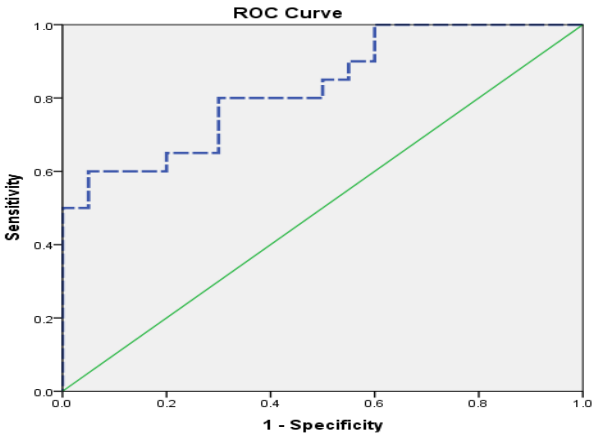

Figure 1. ROC curve of IL-17 to differentiate between Group 1 (Active LN) and Group 2 (Inactive LN)

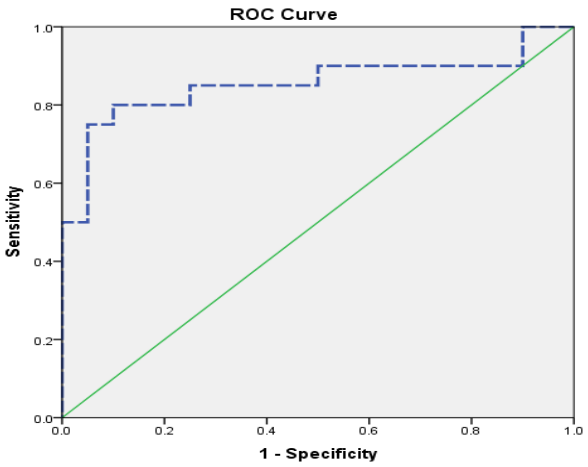

Figure 2. ROC curve of IL-17 to differentiate between Group 2 (Inactive LN) and Control group

Serum creatinine, ESR, and ACR decreased significantly after treatment compared to basal values. The C3 and C4 levels showed a statistically significant increase. (Table 5).

IL-17 level showed a statistically significant decrease after treatment (P= 0.001). The prevalence of positive ANA and Anti-dsDNA before and after treatment did not differ statistically significantly in the cases of active LN (Table 5).

Table 5. Comparison of the laboratory data before and after treatment in the active LN group

ARIABLES	BASAL (N=20)	AFTER TREATMENT (N=19)	SIGNIFICANCE TEST
HEMOGLOBIN (GM/DL) [MEAN ± SD]	9.66 ± 2.07	9.88 ± 1.78	t = - 0.818 P = 0.424
PLTS(10^3/ML) [MEAN ± SD]	215 (135 – 560)	267 (142 – 400)	Z = - 0.121 P = 0.904
WBCS (10^3/ML) [MEAN ± SD]	7.45 (3.8 – 19)	8.7 (4.4 – 14)	Z = 0.010 P = 0.984
ALT (U/L) [MEAN ± SD]	33 (16 – 80)	35 (20 – 65)	Z = - 0.486 P =0.327
AST (U/L) [MEAN ± SD]	36 (10 – 77)	35 (10 – 75)	Z = - 0.040 P =0.968
CREATININE (MG/DL) [MEAN ± SD]	2.3 (0.9 – 4.9)	1.95 (0.8 – 4.8)	Z= -2.236 P = 0.020 *
UREA (MG/DL) [MEAN ± SD]	87 (22 – 146)	55 (35 – 175)	Z= -1.650 P = 0.099
C3 (MG/DL) [MEAN ± SD]	25 (2 – 71)	84 (19 – 165)	Z= - 2.984 P = 0.003 *
C4 (MG/DL) [MEAN ± SD]	8 (2 – 18)	19 (3 – 31)	Z = - 3.823 P <0.001 *
CRP (MG/DL) [MEAN ± SD]	7 (0- 117)	4 (0.5- 75)	Z = -0.161 P =0.872
ESR (MG/DL) [MEAN ± SD]	39 (23- 79)	15 (9- 30)	Z = -3.661 P <0.001 *
ACR (MG/DL) [MEAN ± SD]	2683 (1079 – 5062)	1556 (633 – 3520)	Z = -3.847 P <0.001 *
IL-17 (PG/ML) [MEDIAN (RANGE)]	246 (88 - 412)	121.1 (33.9 – 333.7)	z = - 3.300 P = 0.001 *
ANA [N (%)]			MCN = 0.845
NEGATIVE	3 (15%)	3 (15.8%)	P= 0.625
POSITIVE	17 (75%)	16 (84.2%)	
		ANTI-DSDNA [N (%)]	MCN = 1.163
NEGATIVE	8 (40%)	8 (42.1%)	P= 0.246
POSITIVE	12 (60%)	11 (57.9%)	

T: PAIRED SAMPLE T-TEST Z: WILCOXON-SIGNED RANK TEST
MCN: MCNAMAR'S TEST *: STATISTICALLY SIGNIFICANT (P≤ 0.05)

In the cases of active lupus nephritis, there was a statistically significant improvement in the protein and RBCs found by urine analysis after treatment as compared to the value before treatment (Table 6).

Table 6. Comparison of urine analysis before and after treatment in the active LN group

VARIABLES	BASAL (N=20)	AFTER TREATME (N=19)
PROTEIN [N (%)]		
NEGATIVE	0 (0%)	0 (0%)
+	3 (15%)	13 (68.4%)
++	9 (45%)	5 (26.3%)
+++	8 (40%)	1 (5.3%)
		GRANUL
NEGATIVE	8 (40%)	9 (47.4%)
+	5 (25%)	5 (26.3%)
++	3 (15%)	4 (21.1%)
+++	4 (20%)	1 (5.3%)
		HYALI
NEGATIVE	16 (80%)	16 (84.2%)
+	1 (5%)	1 (5.3%)
++	3 (15%)	1 (5.3%)
+++	0 (0%)	1 (5.3%)
RBCS [N (%)]		
NEGATIVE	5 (25%)	11 (57.9%)
POSITIVE	15 (75%)	8 (42.1%)

MCN: MCNAMAR'S TEST MH: MARGINAL HOMOGENEITY TEST
*: STATISTICALLY SIGNIFICANT (P≤ 0.05)

4. Discussion

In this research, 60 subjects were divided into 20 active LN cases, 20 inactive LN cases, and 20 healthy control subjects.

In the current study, median IL-17 levels were significantly higher in the active LN group than in the inactive LN and control groups. Inactive LN patients also had a significantly higher median IL-17 level than the control group.

According to current findings, Dedong and his colleagues showed a significant difference in the levels between the groups (P < 0.001). The active group had higher levels of IL-17 as related to the inactive and healthy control groups (P < 0.001). Between the inactive group and the healthy control group, IL-17 did not differ significantly (P=0.059) .[11]

Zickert et al. (2015) demonstrated that the cases' baseline IL-17 level was 97.42 (3.30-381.6) pg/ml, which was statistically significantly higher than the control group's level of 3.30 (3.30-62.66) pg/ml. [12]

Additionally, this was consistent with the findings of the Abdel Galil et al. study, which showed that IL-17 levels were significantly higher in SLE patients than in normal subjects (p < 0.001). There was a significant difference in IL-17 levels between the active and inactive patient groups (P < 0.001). [13]

Protein in urine is strongly associated with elevated serum IL-17 levels .[14,15] A positive correlation exists between its baseline concentration and the severity of proteinuria .[16]

There is a positive correlation between the nephritis activity index and serum IL-17 (and TWEAK) levels .[17] The histological activity index, cellular crescent, and endocapillary proliferation positively correlated with circulating .[18]

However, this study's results were inconsistent with those of a prior investigation that found no appreciable difference in IL-17 serum levels between SLE cases with and without nephritis .[19] Additionally, Vincent et al. did not discover any association between baseline serum IL-17 levels and the existence or nonexistence of renal disease during their subjects' long-term follow-up .[20]

With high sensitivity (80%) and moderate specificity, the optimal cutoff point of IL-17 in the current study to distinguish between Group 1 (Active LN) and Group 2 (Inactive LN) was > 137.6 pg/ml (70 percent). The optimal IL-17 cutoff point, with high sensitivity (85%) and specificity, to distinguish between Group 2 (Inactive LN) and the Control group was > 33 ng/ml (75 percent).

The ROC curve analysis conducted by Abdel Galil et al. determined that IL-17 had an optimal cutoff of 19.7 pg/ml for disease activity indicators, with a sensitivity of 93.3%, specificity of 92.9%, positive predictive value (PPV) of 90.3%, and an area under the curve (AUC) of 0.95 (with a 95% confidence interval of 0.90-1). [13]

DeJong et al. found that IL-17 had an area under the curve (AUC) of 0.91 when used as a biomarker to predict LN activity (SLEDAI > 9). (P < 0.001). [11]

In the cases with active LN, with follow-up, 1 case died, 2 cases showed no remission, and 17 cases (85%) showed remission.

Consistent with the present investigation, Dedong et al. demonstrated that thirty-seven active LN cases took up the treatment and underwent a six-month follow-up. In the active group, 19 cases had a CR, 13 had a PR, and 5 had an NR following therapy .[11]

Response evaluation in LN is challenging, and despite significant efforts, no widely recognized response criteria are currently available. Proteinuria is the most crucial outcome for response in most LN studies; however, the term "remission" should only be applied in light of renal biopsy results .[21]

In the current research, the median (range) IL-17 level in the active LN before treatment was 246 pg/ml (88 - 412), which showed a statistically significant decrease after treatment to 121.1 pg/ml (33.9 – 333.7) (P= 0.001).

This agreed with Zickert et al. (2015), who included fifty-two cases with active LN. The research showed that the IL-17 level in cases at baseline was 97.42 (3.30-381.6) pg/ml, which revealed a statistically significant decrease after treatment [47.98 (3.30-824.5) pg/ml] .[12]

However, a different study revealed that although serum levels of IL-17A and IL-21 decreased during induction therapy, the non-remission group's levels of these cytokines remained higher than the remission group's .[16]

The present study's limitations include a small sample size and the fact that cases were recruited from a single center.

4. Conclusion

In cases of SLE, a higher serum IL-17 level could be used as a predictive factor for the presence of LN activity. In cases of active LN, serum IL-17 is a good predictor of how well the patient will respond to treatment.

Disclosure

The authors have no financial interest to declare in relation to the content of this article.

Authorship

All authors have a substantial contribution to the article

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Conflicts of interest

There are no conflicts of interest.

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