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The Association of Metabolic Syndrome and Its Components with Lupus Nephritis in Systemic Lupus Erythematosus Patients

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Abstract

Background: Metabolic syndrome (Mets) is common in people with Systemic Lupus Erythematosus (SLE), with an incidence that varies from 3.3 percent to 45.2 percent. The purpose of this work was to compare the incidence of Mets in a group of individuals with SLE with that of healthy controls and to examine any potential connotation between Mets and lupus nephritis (LN).

Methods: Mets occurred in 34.2% of SLE patients compared with controls. SLE cases had higher rates of hypertension, diabetes, and dyslipidaemia. LN patients demonstrated elevated cholesterol, triglycerides, and SLEDAI scores. Mets was strongly associated with LN, increased steroid use, and higher disease activity. Age, BMI, central obesity, hypertension, and LN were key predictors.

Results: Metabolic syndrome was more prevalent in SLE patients (34.2%) than in controls. Compared to controls, the SLE group had higher rates of hypertension, diabetes, and elevated blood pressure, but lower body weight, triglycerides, and HDL levels. Within SLE cases, LN patients showed higher cholesterol, triglycerides, and SLEDAI scores than non-LN patients. The presence of Mets was strongly associated with LN, higher steroid doses, and increased disease activity. Significant predictors of Mets in SLE included age, BMI, central obesity, hypertension, SLEDAI score, LN, and steroid use

Conclusions: Mets is common in SLE, particularly with LN, and correlates with disease activity and cardiovascular risk.

Keywords: Metabolic Syndrome, Lupus Nephritis, Systemic Lupus Erythematosus, body mass index, high density lipoproteins.

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Introduction:

An immunological, persistent, and diverse illness known as SLE can arise for no apparent reason. [1, 2]. The prevalence of SLE and the associated morbidity and mortality rates are higher among some racial and ethnic groups. [3] SLE frequently leads to the onset of irreversible organ damage. [4]

One common consequence of SLE is LN, an immunological complex glomerulonephritis .[5] Although LN is not always the first sign of SLE, it is among the most severe symptoms that develop as the disease progresses. Restoring kidney function and avoiding potentially fatal consequences are both made possible by prompt identification and treatment of LN . [6]

Several studies have shown that coronary artery disease and cerebrovascular disorders are leading causes of death and morbidity for SLE patients. patients with SLE had a 3.04-fold increased risk of myocardial infarction and a 1.96-fold increased risk of stroke in contrast to the whole population. [7] Combinations of traditional risk factors and those linked to SLE increase the probability of CVD. An autoimmune response promotes the activity and advancement of SLE, which in turn increases the risk of CVD, as well as endothelial dysfunction, early atherogenesis, and cardiac disease. [8]

Furthermore, conventional risk factors for CVD in SLE patients include advancing age, hypertension, dyslipidaemia, diabetes mellitus, menopause, and smoking, much as in the general population. [8]

The stakes for death and disability are higher when CVD is considered a risk factor for Mets. Mets accelerates insulin resistance, oxidative damage, and the risk of CVD. [9]

Hypertriglyceridemia, insulin resistance, low HDL, arterial hypertension, and visceral obesity are the criteria for considering the Mets.[10] Type II diabetes mellitus and cardiovascular diseases are strongly predicted by Mets in the general population. [11]

Several meta-analyses and longitudinal studies have found that vascular mortality, cardiovascular events, cerebrovascular events, and subclinical atherosclerosis are all more likely in people with Mets. Particularly in the elderly, however, there is ongoing dispute over whether the syndrome or its individual components provide a better prognostic value for mortality . [9]

The prevalence of Mets in patients with SLE varies from 3.3 percent to 45.2 percent, which is approximately double the general population rate. Since inflammation and insulin resistance are important components of visceral obesity, the Mets may have a pivotal role in the connection between SLE-related chronic inflammation and accelerated atherosclerosis. [12]

Few studies discussed the association between Mets and SLE, to our knowledge this study is the first shedding light on the relation between Mets and LN. We aimed to study the prevalence of Mets in a cohort of Egyptian SLE patients versus healthy controls and to explore the relation between Mets and LN.

Patients and Methods:

This retrospective case-control research was performed on 161 participants diagnosed as SLE in accordance with 2019 ACR/EULAR classification criteria[13] aged more than 18 year and classified as 92 individuals with LN based on results of renal biopsy & 69 individuals without nephritis as well as 60 healthy controls with matched age and sex . The study was conducted from July 2023 to July 2024.

The Ethical Committee of Sohag University Hospitals gave their clearance before the study could begin. For this study, we made sure to get patients' written, informed consent.

Individuals who were under the age of eighteen years old, individuals who were unable or unable to provide written informed consent, individuals who had autoimmune diseases other than SLE, patients who had renal issues that were unrelated to LN, pregnant individuals, and patients who had malignancies were all excluded from the research.

All individuals were subjected to: Full medical history (Demographic data, age, marital status, sex, occupation, duration of the disease and residence). History of all possible SLE manifestations and complications, Therapeutic history, general and rheumatological examination.

Assessment of the Disease activity in the patients was done using the SLEDAI:

The scale comprises 24 "weighted" attributes categorized into 9 domains (organ systems). The final score consists of the aggregate of all weighted assigned scores. [13]

The SLEDAI scores have been utilized to establish activity categories: no activity (SLEDAI = 0), moderate activity (SLEDAI = 6-10), mild

activity (SLEDAI = 1-5), high activity (SLEDAI = 11-19), as well as very high activity (SLEDAI >20). The maximum total score of SLEDAI is 105. [14]

Data of renal biopsy was collected from individuals' previous medical reports before the study.

Laboratory investigations: Routine investigations including, complete blood picture, (anemic or not, presence of thrombocytopenia, leucopoenia), ESR by Westergren method and, liver functions, Kidney functions, urine analysis, protein /creatinine ratio (p/c ratio).

Renal biopsy:

Indirect immunofluorescence, light microscopy, and electron microscopy were used to examine renal specimens. The International Society of Nephrology/Renal Pathology Society (ISN/RPS) used the following criteria to categorize renal histopathology:

- Class I: Minimal mesangial LN
- Class II: Mesangial proliferative LN
- Class III: Focal LN (chronic & active, sclerosing & proliferative)
- Class IV: Diffuse LN (proliferative & sclerosing, active & chronic, segmental & global)
- Class V: Membranous LN
- Class VI: Advanced sclerosis LN. [15]

ANA by IF.

Anti-dsDNA was done by ELISA.

C3 and C4 proteins by nephelometry.

Evaluation of metabolic parameters:

Clinical history was obtained from all participants, including personal and familial history of diabetes mellitus, hypertension, dyslipidaemia, and CVD.

Anthropometric measurements were conducted as follows:

- Waist circumference was measured when the participant lay flat on their back, legs parallel to the floor, and iliac crest level during normal expiration. Men with waist circumferences over 102 centimetres and women over 88 centimetres were considered obese . [16]
- BMI is determined by dividing weight in kilograms by height in meters squared. According to WHO, BMI classifications include underweight (<18.5 kg/m²), overweight (25-29.9 kg/m²), normal (18.5-24.9 kg/m²), and obese (≥30 kg/m²). [17] The seated person's resting arterial blood pressure was taken twice after five minutes. Systolic blood pressure readings of 140 mm Hg or above, or the use of blood pressure medication, were diagnostic of hypertension. [18]

To minimize bias, all assessments were performed under standardized conditions. Fasting venous blood samples were collected for biochemical analysis of the following metabolic parameters:

- Fasting glucose: values ≥126 mg/dL were considered indicative of diabetes; 100–125 mg/dL as impaired fasting glucose. [19,20]
- Total cholesterol: levels ≥200 mg/dL were considered elevated.
- Elevated levels of LDL-C were defined as values of 130 mg/dL or more, whereas optimum levels were defined as below 100 mg/dL.
- HDL-C levels under 40 mg/dL in men as well as 50 mg/dL in women were deemed to be below average.
- Triglycerides: levels ≥150 mg/dL were considered elevated. [21]

In 2005, the National Cholesterol Education Program Adult Treatment Panel III (2005 NCEP/ATP III) [22] stated The metabolic syndrome is characterized by the presence of three out of the five criteria listed below: central obesity (waist circumference ≥102 cm in men and ≥88 cm in women), arterial hypertension (blood pressure ≥130/85 mmHg or the use of antihypertensive medications), low HDL cholesterol (<40 mg/dL in men and <50 mg/dL in women), hypertriglyceridemia (triglycerides ≥150 mg/dL or the use of lipid-lowering medications), and glucose intolerance or diabetes (fasting glucose ≥100 mg/dL or the use of hypoglycaemic agents). We collected BP, anthropometric data (height, weight, BMI, waist circumference), sociodemographic data (gender, age, smoking status), and laboratory data (total cholesterol, HDL cholesterol, fasting blood glucose, triglycerides) from a control group.

Statistical analysis:

The data was examined with the use of SPSS version 26.0, which is an IBM product for the social sciences. Quantitative variables included in this group included means and standard deviations (SD), which were compared using paired Student's t-tests. We used the chi-square

test to compare the percentage-based qualitative variables and the fisher exact test to compare the frequencies-based qualitative variables. If the data was not parametric, we applied the Fisher exact test instead of the usual chi-square test. We utilized the Pearson Chi-square test to compare percentages of qualitative variables. The Mann-Whitney test was used for non-parametric variables in order to compare groups. If the p-value was below 0.05, we deemed all comparisons statistically significant. Three levels of significance were used to accept the significance of differences: the following values are significant: * <0.05, ** <0.01, and *** <0.001.

Results:

This study included 161 patients diagnosed as SLE according to 2019 ACR/EULAR classification criteria of SLE, as well as 60 age and sex matched normal volunteers as control group. The patients were classified according to the presence of LN into 2 groups, 92 patients with LN and 69 patients without LN.

The mean age of the SLE cases was 35.89±10.38 years and that of controls was 33.64±5.11. Females represented 143 (88.82%) of the cases and 52 (86.67%) of the control group. Males represented 18 (11.18%) of the cases and 8 (13.33%) of the control group. Among lupus patients the most common clinical manifestations were Mucocutaneus manifestations present in (96.27%) of the individuals in the form of oral ulcers present in (75.15%) , Photosensitivity in about (65.83%) and discoid lupus lesions in about (11.80%), followed by musculoskeletal manifestations (92.54%), constitutional manifestations (78.8%), haematological manifestations (62.73%), pulmonary manifestations (19.25%), neuropsychiatry manifestations (14.90%),and the least presented manifestations were cardiac manifestations (7.45%) as shown in **table 1**.

On comparing SLE patients and controls as regards anthropometric measures and Mets components. Both groups showed a highly significant difference in terms of Mets, hypertension, central obesity, and waist circumference (p <0.001), as well as a highly significant difference in terms of BMI, HDL cholesterol, systolic blood pressure, and diastolic blood pressure. Diabetes mellitus incidence (p=0.002, p=0.002, p=0.001, p=0.001, and p=0.004). In terms of weight, triglycerides, and HDL (<40 mg/dL in males or <50 in women), there was a significant distinction between the two groups (P=0.01, P=0.04, and P=0.01, correspondingly) as shown in **table 2**.

The occurrence of high systolic and diastolic blood pressure was significantly different between the two groups when comparing SLE patients with and without LN (p<0.001, p=0.004, and p=0.001). Compared to the other group, LN persons had significantly higher cholesterol levels and a significantly higher prevalence of Mets (p=0.001 and p=0.008, individually). Additionally, there was a significant difference between the two groups in terms of the presence of Mets components, specifically triglycerides (p=0.01) as shown in **table 3**.

In terms of SLEDAI score and laboratory results, comparing SLE individuals with and without LN exists. LN patients had significantly higher blood creatinine levels and a significantly higher SLEDAI score (p=0.002 and p=0.004, individually). When looking at AST, there was a highly significant difference among the two groups (p=0.004). The presence of albumin in urine (p<0.001) and the p/c ratio (p<0.001), which were both greater in LN patients, showed a highly significant statistical variance between the two groups. Patients with nephritis were more likely to have casts in their urine (p=0.002), consumed C3 (p=0.008), and had RBCS in their urine (p=0.04), all of which were significantly different between the two groups. regarding ANA(IF), ANA profile including Anti ds.DNA and anti-histone showing statistically significant difference between both groups (p=0.02, p=0.008) respectively as shown in **table 4**.

On comparing patients with Mets with those without Mets there was a significant difference between the two groups in terms of Mets components (p<0.001), which include obesity, weight, BMI, central obesity, hypertension, hypertension, fasting blood sugar, HDL (<50 in women or <40 mg/dL in men), diabetes, cholesterol, triglycerides (≥150), and low-density lipoprotein (LDL). In addition, the two groups differed significantly in terms of waist circumference (p=0.002) and height (p=0.02) as shown in **table 5**.

Univariate logistic regression analysis of the factors that influence the occurrence of Mets in SLE individuals revealed that the most significant

factors were age, BMI, central obesity, hypertension, SLEDAI score, LN, and steroid dose (mg) (p =0.02, p<0.001, p=0.02, p=0.002, p<0.001), correspondingly as shown in **table 6**.

Table 1: Demographic and clinical characteristics of SLE cases and controls:

Variable	SLE patients N=161	Controls N=60	P value
Age/ years			
Mean ± SD	35.89±10.38	33.64±5.11	0.0519
Median (range)	34 (19:65)	30 (21:43)	
Gender	143 (88.82%)	52 (86.67%)	0.66
Female	18 (11.18%)	8 (13.33%)	
Male			
Smoking	6 (3.73%)	6 (10.00%)	0.09
Mucocutaneus manifestations			
Malar rash	97 (60.24%)		
Photosensitivity	106 (65.83%)		
DLE	19 (11.80%)		
Alopecia	87 (54.03%)		
Oral ulcer	121 (75.15%)		
cutaneous manifestations	155 (96.27%)		
constitutional manifestations	127 (78.8%)		
Musculoskeletal manifestations	149 (92.54%)		
neuropsychiatry manifestations	24 (14.90%)		
hematological manifestations	101 (62.73%)		
cardiac manifestations	12 (7.45%)		
pulmonary manifestations	31 (19.25%)		

DLE: discoid lupus erythematosus t test Mann Whitney test chi square Fischer exact test

Table 2 Anthropometric measures of Mets and it's components in SLE cases and controls:

Variable	SLE patients N=161	Controls N=60	P value
Weight (Kg)			
Mean ± SD	75.59±15.03	81.28±17.25	0.01 *
Median (range)	75 (35:120)	80 (55:150)	
Height (cm)			
Mean ± SD	162.97±8.53	160.9±9.45	0.12
Median (range)	162 (130:188)	160 (140:178)	
BMI			
Mean ± SD	28.82±6.27	30.84±6.84	0.002 **
Median (range)	27.8 (14.8:48.8)	31.23 (19.4:49)	
Waist circumference (cm)			
Mean ± SD	72.83±13.81	88.28±21.22	<0.001 ***
Median (range)	72 (39:102)	88 (54:160)	
Systolic blood pressure (mm Hg)			
Mean ± SD	124.47±15.81	117±10.86	0.001 **
Median (range)	120 (100:170)	120 (90:150)	
Diastolic blood pressure (mm Hg)			
Mean ± SD	81.37±10.32	77.41±7.65	0.001**
Median (range)	80 (60:110)	80 (60:90)	
Fasting blood sugar (mg/dL)			
Mean ± SD	92.14±31.24	86.32±10.34	0.44
Median (range)	83 (60:300)	87.5 (60:113)	
Cholesterol (mg/dL)			
Mean ± SD	164.01±56.08	168.8±31.22	0.27
Median (range)	160 (45:402)	166 (105:240)	
Triglyceride (mg/dL)			
Mean ± SD	114.43±69.13	120.25±35.34	0.04*
Median (range)	90 (30:613)	120 (41:220)	
HDL (mg/dL)			
Mean ± SD	42.22±9.09	47.23±11.13	0.002**
Median (range)	40 (21:75)	45.5 (30:94)	
LDL (mg/dL)			
Mean ± SD	88.16±26.34	96.26±21.78	0.06
Median (range)	90 (10:130)	91.7 (45:135)	
Central obesity	30 (18.63%)	28 (46.67%)	<0.001 ***
Hypertension	52 (36.02%)	5 (8.33%)	<0.001***
DM	34 (21.12%)	3 (5.00%)	0.004**
Increased Triglycerides	50 (31.06%)	12 (20.00%)	0.10
Decreased HDL	119 (73.91%)	34 (56.67%)	0.01*
Mets	55 (34.16%)	4 (6.67%)	<0.001***

*Statistically significant at P value<0.05. **Statistically significant at P value<0.01.
*** Statistically significant at P value<0.001 test Mann Whitney test chi square Fischer exact test

Table 3 Comparison between SLE cases with and without LN regarding anthropometric measures and Mets components:

Variable	SLE		P value
	No LN, N=69	LN, N=92	
Weight (Kg)			
Mean ± SD	73.88±16.24	76.87±14.02	0.21
Median (range)	72 (35:120)	75.5 (47:112)	
Height (cm)			
Mean ± SD	163.03±8.79	162.92±8.38	0.94
Median (range)	163 (130:177)	161 (145:188)	
BMI			
Mean ± SD	28.18±6.64	29.3±5.96	0.26
Median (range)	27 (14.8:48.6)	28 (18.3:48.8)	
Waist circumference (cm)			
Mean ± SD	73.20±13.34	72.54±14.21	0.59
Median (range)	73 (40:100)	70 (39:102)	
Diastolic blood pressure (mm Hg)			
Mean ± SD	77.83±9.79	84.02±9.95	0.001**
Median (range)	80 (60:100)	80 (70:110)	
Systolic blood pressure (mm Hg)			
Mean ± SD	119.49±14.15	128.61±16.03	0.004**
Median (range)	120 (100:160)	130 (100:170)	
Fasting blood sugar (mg/dL)			
Mean ± SD	92.87±36.42	91.59±26.90	0.59
Median (range)	84 (60:300)	83 (67:250)	
Cholesterol (mg/dL)			
Mean ± SD	150.28±50.50	174.32±58.08	0.008**
Median (range)	150 (45:274)	170 (70:402)	
Triglyceride (mg/dL)			
Mean ± SD	101.13±56.91	124.41±75.81	0.01*
Median (range)	80 (30:416)	110 (30:613)	
HDL (mg/dL)			
Mean ± SD	41.86±8.73	42.49±9.40	0.65
Median (range)	40 (24:61)	41 (21:75)	
LDL (mg/dL)			
Mean ± SD	83.67±27.27	91.53±25.25	0.052
Median (range)	80 (10:130)	99 (15:130)	
Central obesity	10 (14.49%)	20 (21.74%)	0.24
Hypertension	14 (20.29%)	44 (47.83)	<0.001***
DM	15 (21.74%)	19 (20.65%)	0.87
Increased Triglyceride	14 (20.29%)	36 (39.13%)	0.01*
Decreased HDL	49 (71.01%)	70 (76.09%)	0.47
Mets	14 (20.29%)	41 (44.57%)	0.001**

t test Mann Whitney test chi square Fischer exact test

*Statistically significant at P value<0.05.

**Statistically significant at P value<0.01.

*** Statistically significant at P value<0.001

Table 4. Comparison between SLE cases with and without LN as regard SLEDAI score and Laboratory findings:

Variable	SLE		P value
	No LN N=69	LN N=92	
SELDI			
Mean ± SD	6.04±5.77	10.64±7.97	0.002**
Median (range)	4 (0:24)	10 (0:34)	
WBCs (10^3/ µL)			
Mean ± SD	6.33±3.14	6.59±3.10	0.52
Median (range)	5.6 (1.01:17.7)	6.6 (1.28:17.2)	
HB (gm/dL)			
Mean ± SD	11.04±1.96	10.70±1.89	0.27
Median (range)	11 (6.4:16.3)	10.6 (5.7:14.9)	
MCV			
Mean ± SD	81.06±7.90	80.23±10.99	0.60
Median (range)	80.8 (62:100)	81.2 (8:96)	
Platelets (10^3/ µL)			
Mean ± SD	240.35±103.85	266.63±105.29	0.20
Median (range)	255 (3.9:541)	265.5 (18:577)	
ESR			
Mean ± SD	55.18±36.84	60.35±35.34	0.23
Median (range)	45 (8:155)	50 (5:150)	
S. creatinine (mg/dL)			
Mean ± SD	0.75±0.41	1.07±0.72	0.004**
Median (range)	0.7 (0.3:3.4)	0.9 (0.3:4.2)	
AST (IU/L)			
Mean ± SD	26.29±17.89	20.30±11.01	0.004**
Median (range)	21 (9:102)	18 (2:79)	
ALT (IU/L)			
Mean ± SD	24.54±22.93	17.70±11.75	0.06
Median (range)	18 (6:127)	15 (7:91)	
P/C ratio			
Mean ± SD	0.22±0.41	2.48±3.12	<0.001***
Median (range)	0.12 (0:3.2)	1.3 (0.02:13.6)	
Cast	9 (13.04%)	32 (34.78%)	0.002**
Albumin	33 (47.82%)	70 (76.09%)	<0.001***
Pus	29 (42.03%)	45 (48.91%)	0.39
RBCs	16 (23.19%)	35 (38.04%)	0.045*
Glucose			
Yes	4 (5.80%)	3 (3.26%)	0.46
C3			
Mean ± SD	106.22±36.14	93.53±30.35	0.008**
Median (range)	109 (0:168)	89.5 (23:150)	
C3 group			
Consumed	11 (15.94%)	33 (35.87%)	0.005**
C4			
Mean ± SD	25.13±13.57	21.21±10.64	0.07
Median (range)	22 (0:81)	19 (4.5:58.8)	
C4 group			
Consumed	6 (8.70%)	16 (17.39%)	0.11
ANA (IF)			
Negative	6 (8.70%)	4 (4.35%)	0.33
Positive	63 (91.30%)	88 (95.65%)	
ANA profile			
Negative	20 (28.99%)	25 (27.17%)	0.80
Positive	49 (71.01%)	67 (72.83%)	
Anti ds DNA			
Negative	41 (59.42%)	40 (43.96%)	0.02*
Positive	22 (31.88%)	48 (52.75%)	
Equi	6 (8.70%)	3 (3.30%)	

t test Mann Whitney test chi square Fischer exact test

*Statistically significant at P value<0.05.

**Statistically significant at P value<0.01.

*** Statistically significant at P value<0.001

Table 5 comparison between SLE individuals with & without Mets regarding anthropometric measures and Mets components:

Variable	No Mets N=106	Mets N=55	P value
Weight (Kg)			
Mean ± SD	71.05±12.28	84.35±16.05	<0.001***
Median (range)	71 (35:100)	85 (45:120)	
Height (cm)			
Mean ± SD	164.13±9.32	160.73±6.24	0.02*
Median (range)	163 (130:188)	160 (149:174)	
BMI			
Mean ± SD	26.57±4.40	33.15±7.08	<0.001***
Median (range)	26.7 (14.8:42)	31.6 (19.5:48.8)	
Waist circumference (cm)			
Mean ± SD	69.48±11.81	79.27±15.14	0.002**
Median (range)	70 (39:102)	88 (49:100)	
Systolic blood pressure (mm Hg)			
Mean ± SD	117.5±11.51	137.91±14.26	<0.001***
Median (range)	120 (100:170)	140 (100:160)	
Diastolic blood pressure (mm Hg)			
Mean ± SD	77.12±8.61	89.55±8.24	<0.001***
Median (range)	80 (60:110)	90 (60:100)	
Fasting blood sugar (mg/dL)			
Mean ± SD	83.70±14.72	108.4±45.40	<0.001***
Median (range)	81 (60:170)	91 (70:300)	
Cholesterol (mg/dL)			
Mean ± SD	146.56±52.32	197.66±47.37	<0.001***
Median (range)	149 (45:402)	200 (100:366)	
Triglyceride (mg/dL)			
Mean ± SD	89.71±60.03	162.09±60.33	<0.001***
Median (range)	80 (30:613)	159 (59:416)	
HDL (mg/dL)			
Mean ± SD	83.38±9.12	39.98±8.69	0.03*
Median (range)	42 (22:75)	40 (21:66)	
LDL (mg/dL)			
Mean ± SD	81.10±26.04	101.76±21.25	<0.001***
Median (range)	80 (10:130)	110 (30:130)	
Central obesity	3 (2.83%)	27 (49.09%)	<0.001***
Hypertension	12 (11.32%)	46 (83.64%)	<0.001***
DM	9 (8.49%)	25 (45.45%)	<0.001***
Increased Triglyceride	7 (6.60%)	43 (78.18%)	<0.001***
Decreased HDL	68 (64.15%)	51 (92.73%)	<0.001***

t test Mann Whitney test chi square Fischer exact test

*Statistically significant at P value<0.05.

**Statistically significant at P value<0.01.

*** Statistically significant at P value<0.001

Table 6. Univariate logistic regression analysis of factors affecting occurrence of Mets in SLE patients:

Variable	Univariate analysis	
	Odds ratio (95% CI)	P value
Age/ years	1.04 (1.01:1.07)	0.02*
BMI	1.24 (1.14:1.34)	<0.001***
Central obesity vs. no	33.11 (9.36:117.16)	<0.001***
Hypertension vs. no	40.04 (15.74:101.82)	<0.001***
DM vs. no	8.98 (3.78:21.33)	<0.001***
SLEDAI score	1.05 (1.01:1.10)	0.02*
LN vs. no	3.15 (1.52:6.46)	0.002
Abnormal C3 vs. normal	1.95 (0.96:3.98)	0.07
Abnormal C4 vs. normal	1.74 (0.70:4.33)	0.23
Steroid dose (mg)	1.06 (1.03:1.09)	<0.001***
Duration of steroid (months)	0.98 (0.94:1.02)	0.23

Discussion

Metabolic syndrome occurs 3.3% to 45.2% more frequently in those with SLE in contrast to the general population. Visceral adiposity is a result of inflammation and insulin resistance, two important processes that may be mediated by Mets in SLE. [12]

Hypertension and low HDL are the most frequent components; high triglycerides and high blood pressure show the strongest associations with SLE. [23,24] Chronic inflammation and adipokine imbalance (e.g., leptin↑, adiponectin↓ in some studies) contribute to insulin resistance, dyslipidaemia, and atherogenesis in SLE.[26] Higher disease activity, renal involvement, and accrued damage are associated with persistent Mets phenotypes in SLE. [25]

Glucocorticoids promote Mets features (gluconeogenesis/IR, weight gain, dyslipidaemia); conversely, hydroxychloroquine (HCQ) is recommended in SLE in part for favourable cardiometabolic effects and CV risk reduction. [23,27]

EULAR cardiovascular-risk guidance and 2023 SLE management updates emphasize systematic CV risk assessment, lifestyle measures (weight, BP, lipids, glucose), steroid minimization, and background HCQ unless contraindicated. [27,28,29]

According to our findings metabolic syndrome was present more frequently in SLE patients than in healthy controls. The SLE group had a significantly higher incidence of hypertension and diabetes mellitus in contrast to the control group. Conversely, WC, HDL levels and BMI were significantly lower in the SLE group than in the controls.

Supporting our results, **Bhat et al. [30]** showed that the systolic and diastolic blood pressures of the SLE group were significantly higher than those of the control group. In accordance with our findings, **Sabio et al. [31]** found there was a significant increase in hypertension, systolic blood pressure and diastolic blood pressure in SLE group than controls group.

In the same line, **Liu et al. [36]** illustrated in their study that 24.4% of SLE individuals were currently taking medications to treat HTN.

In this study There was statistically significant decrease in weight, cholesterol, triglycerides and HDL in SLE group than control group. In accord with our findings, **Quevedo-Abeledo et al [32]** illustrated that triglycerides and LDL: HDL ratio was significantly lower in individuals with SLE in contrast to controls. while, **Mobini et al. [33] and Sabio et al.[31]** demonstrated that there was no significant variance in triglycerides and HDL between SLE group and controls group. In contrast, **Bhat et al. [30] and Sinicato et al. [34]** demonstrated that In the SLE group, total cholesterol and triglyceride levels were significantly elevated compared to the control group. The control group exhibited no statistically significant difference in weight contrasted with the experimental group. The observed disparity can be ascribed to disparities in the research location and sample size.

There was a very high significant increase in occurrence of hypertension including SBP and DBP in LN individuals than those without nephritis.

In our study SLE cases with LN showed a significantly higher prevalence of Mets, higher cholesterol levels and elevated triglyceride levels contrasted with those without LN. Supporting our results, **Rabrenović et al.** [37] revealed that Mets was related to impaired kidney function. Also, **Mirghani et al.** [38] demonstrated that cases with LN had higher total cholesterol and triglycerides.

Complying with our results, **Hammam et al.** [39] noted that the Mets group exhibited significantly elevated LN activity. Also, **Sinicato et al.** [34] **and Medeiros et al.** [40] revealed that nephritis was significantly increased in SLE group with Mets than those without Mets.

Conversely, **Demir et al.** [41], **Bhat et al.** [30], **Telles et al.** [42] **and Sabio et al.** [31] showed that there was not a significant distinction in renal involvement between the SLE group with and without Mets.

SLE individuals with LN demonstrated significantly higher SLEDAI scores and serum creatinine levels in contrast to those without LN. Supporting our results, **Rabrenović et al.**[37] noted that The LN group's creatinine and SLEDAI scores were significantly higher than the control group's.

SLE cases with LN exhibited a markedly significant increase in urinary albumin levels and the P/C ratio contrasted with those without LN. In accordance with our results, **Rabrenović et al.** [37] found that In the LN group, the urine p/c ratio was significantly higher than in the control group. The LN group, in contrast to the control group, had much lower serum albumin levels.

In comparison to instances without nephritis, SLE patients with LN exhibited a marked rise in the prevalence of urinary casts and a higher consumption of C3 levels. In agreement with our results, **Rabrenović et al.** [37] reported that In comparison to the control group, the LN group exhibited a significantly higher C3 consumption.

There was a significant increase in Anti ds. DNA & anti histone antibodies in LN cases in contrast to those without nephritis. Consistent with our results, **Rabrenović et al.** [37] revealed that Anti-ds DNA antibody was significantly higher in LN group in contrast to control group.

SLE cases with Mets exhibited a highly statistically significant increase in WC, SBP and DBP, hypertension, fasting blood glucose, diabetes, cholesterol, LDL, and triglycerides in contrast to SLE cases without Mets. Conversely, HDL levels were significantly lower in SLE cases with Mets than in those without. Supporting our study, **Telles et al.** [42] noted that Waist circumference, fasting blood sugar, systolic and diastolic blood pressure, and triglyceride levels were all substantially higher in the SLE group with Mets in contrast to the SLE group without Mets. On the other hand, in contrast to the non-Mets group, the SLE group with Mets had much higher HDL values.

In the univariate logistic regression study of factors influencing the occurrence of Mets in cases with SLE, we identified age, BMI, central obesity, DM, hypertension, SLEDAI score, LN, and steroid dosage as the most significant predictors.

Kang et al. [43] revealed that a significant association between newly developed LN and obesity was found utilizing univariate logistic regression. In accordance with our findings, **Parker et al.** [25] found that Mets was related to renal lupus and higher oral dosages of corticosteroids. Complying with our study, **Medeiros et al.** [40] noted that Age, nephritis, and prednisone dosage seemed to have an enhanced effect in their univariate study of Mets in SLE cases. **Telles et al.** [42] reported that Significant associations were seen between Mets features and nephrotic proteinuria throughout lupus follow-up, a higher modified SLEDAI-2k, and an older age upon diagnosis of SLE.

Conclusions:

Individuals with SLE have a higher frequency of Mets in contrast to the control group, and it is more frequently linked to LN. Age, BMI, central obesity, hypertension, SLEDAI, LN and steroid dose are predictors for Mets in SLE cases.

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