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Study of Cardiovascular Diseases Risk in Systemic Lupus Erythematosus Can Be Comparable with Type 2 Diabetes Mellitus

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Authors' contributions

This work was carried out in collaboration among all authors. Author RMA designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Author AAE performed the statistical analysis and managed the analyses of the study, author MHA examined the patients with Doppler ultrasound and author ATE managed the literature searches. All authors read and approved the final manuscript.

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ABSTRACT

Background: Systemic lupus erythematosus (SLE) is a complex autoimmune disease. Cardiovascular manifestations are common in SLE, which may have a wide range of severity and one of the major causes of morbidity and mortality. Diabetes mellitus (DM) is one of the most common metabolic diseases. One of the major risk factor for cardiovascular diseases is DM, which is the most common cause of death among diabetic patients.
Aim of the Work: evaluation of subclinical atherosclerosis as a predictor for CVD in patients with SLE and DM.
Methods: 50 SLE patients, 50 T2DM patients, 50 diabetic SLE patients and 50 healthy controlled subjects were enrolled in this study. They were undergone to Doppler examination of the extra-cranial portion of the carotid and femoral arteries measuring the intima-media thickness.

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Result: In SLE the subclinical atherosclerosis was present in 22% of patients and was significantly associated with older age (p<0,001), high blood pressure (p=0,023), overweight (p=0,004), proteinuria (p<0,001), total cholesterol (p=0,004), active disease (p=0,007), high SLEDAI score (p<0,001) and long duration of SLE (p<0,001); whereas in diabetic patients the subclinical atherosclerosis was documented in 24% and was significantly associated with older age (p<0,001), overweight (p<0,001), proteinuria (p<0,001), low hemoglobin (p=0,041), total cholesterol (p=0,001), LDL (p<0,001), uncontrolled diabetes (p=0,005) and long duration of diabetes (p=0,001) but in SLE diabetic patients the subclinical atherosclerosis was documented in 44% of patients.
Conclusion: Subclinical atherosclerosis is frequent in patients with SLE and increases with increased disease activity. Subclinical atherosclerosis in SLE diabetic patients was significantly more than that in SLE or diabetic patients.

Keywords: Cardiovascular diseases; systemic lupus erythematosus; type 2 diabetes mellitus.

1. INTRODUCTION

Systemic lupus erythematosus (SLE) is a complex autoimmune disease whose incidence varies significantly by sex, race, ethnicity, and socioeconomic status [1]. The etiology of SLE is still unknown. It can influence any organ in the body, particularly the skin, joints, kidneys and cardiovascular system [2]. Cardiovascular manifestations are common in SLE patients, which may have a wide range of severity [3]. All the anatomical structures of the heart can be affected in SLE provoking speeding-up atherosclerotic diseases, endocarditis, myocarditis and pericarditis which are the most common cardiac manifestations of SLE [4]. One of the major causes of morbidity and mortality in systemic autoimmune diseases such as SLE is atherosclerotic diseases [5], as the risk of coronary artery disease (CAD) is 4-8 times higher than in normal persons and incidence of myocardial infarction (MI) is increased up to 50 folds in SLE patients [6]. Diabetes Mellitus (DM) is one of the most common metabolic diseases, which occur due to defect in insulin secretion, insulin action or both leading to hyperglycemia. Hyperglycemia causes disturbances of carbohydrate, fat, and protein metabolism leading to various organs damage, dysfunction, and failure on the long run [7]. One of the major risk factor for Cardiovascular Diseases (CVD) is DM, which is the most common cause of death among diabetic adult patients [8]. Identification of subclinical atherosclerosis by vascular ultrasound examination, in high cardiovascular risk groups can predict future CVD events [9].

The objective of this study was to determine prevalence of subclinical CVD through the presence of carotid or femoral plaque and their associated risk factors in patients with SLE and type 2 diabetes mellitus (T2DM) in comparison with healthy controls.

2. MATERIALS AND METHODS

This study was cross sectional survey study, which was performed at the departments of internal medicine and radiology, Tanta University Hospitals (a tertiary hospital), in the period from August 2017 to August 2018, on a total of 200 patients who were subdivided to four mutually exclusive groups; all patients were consecutively selected from our outpatient clinics.

- Group I: included 50 patients with SLE without demonstrated CVD.
- Group II: included 50 patients with T2DM without demonstrated CVD.
- Group III: included 50 patients with SLE and T2DM without demonstrated CVD.
- Control group: included 50 demographically comparable non-SLE and non-diabetic subjects without demonstrated CVD.

Inclusion criteria were SLE patients without DM and without history of CVD or peripheral vascular disease. Type 2 Diabetic patients without SLE or other rheumatic diseases and without history of CVD or peripheral vascular disease. Patients of group III have SLE and T2DM without other rheumatic diseases and without history of CVD.

Exclusion criteria were Type I DM, gestational diabetes and monogenic diabetes syndromes (MODY),other types of rheumatic diseases other than SLE. Clinical atherosclerotic CVD for all subjects (clinical atherosclerotic CVD was defined as a history of myocardial infarction, stable or unstable angina, coronary or other arterial revascularization, stroke, transient

ischemic attack, and peripheral arterial disease of atherosclerotic origin).

2.1 All Cases Were Subjected to the Following

Thorough history taking, complete clinical examination, laboratory investigations including complete blood count, Erythrocyte Sedimentation Rate (ESR), C-reactive protein (CRP), blood glucose level (fasting blood glucose level (FBG) and 2 hours postprandial blood glucose level (2hPPBG)) and Haemoglbin A1c (HbA1c) for diabetic patients, Blood urea, serum creatinine, lipid profile (cholesterol, triglyceride and Low Density Lipoprotein (LDL)), and immunological tests (antinuclear and anti-DNA antibodies, C3 and C4 levels) for SLE patients.

Doppler ultrasound of carotid and femoral arteries: The Doppler examination was performed by the same operator to eliminate the inter-operator variability; the patients and controls were scanned using alinion ecube 5 quick guide with 11.5 MHz transducer. Participants were examined while lying supine in a dimly lit, quiet room. Participants were asked to avoid vasoactive medications, caffeine and cigarette smoking prior to the examination.

- Intima media thickness (IMT) is measured: in left and right common carotid arteries, carotid bulbs, internal carotid arteries and Left and right common femoral arteries.
- According to current sonographic criteria we refer to normal "IMT" when complex IMT is ≤ 0.9 mm, IMT > 0.9 mm were considered indicative of thickened intima and IMT value > 1.3 mm indicative of atherosclerotic plaque.
- All exams carried out by a single specialist physician, and all images were taken.

2.2 Statistical Analysis

All collected data were organized, tabulated and statistically analyzed using the IBM SPSS, version 23 statistic software. For quantitative data, the median with interquartile range (IQR) were calculated for abnormally distributed data and the mean with standard deviation (SD) were calculated for normally distributed data. Qualitative data were reported as frequency and percentage. Mann–Whitney U test was used for two group comparisons for quantitative data if abnormally distributed while unpaired t-test was used if normally distributed. Chi-Square test was performed to conduct group comparisons for categorical data.

3. RESULTS

According to our SLE patients, there was not any significant proportion difference across the sex distribution; however SLE patients with subclinical atherosclerosis were significantly older than those without atherosclerosis with a significant level of <0,001 as showed in Table 1.

Our study revealed that hypertension, increased body mass index (BMI), proteinuria, hypercholesterolemia and increased LDL cholesterol, all have a significant increase in subclinical atherosclerosis in SLE patients (p value, 0.023, 0.004, 0.000, 0.004 and 0.006 respectively). And also it revealed that SLE activity, increased SLEDAI (Systemic Lupus Erythematosus Disease Activity Index) score and increased disease duration, all have significant increased proportion of subclinical atherosclerosis in SLE patients (p value, 0.007, <0,001 and <0,001 respectively) as showed in Table 1.

According to T2DM patients, there was not any significant proportion difference across the sex distribution; however DM patients with subclinical atherosclerosis were significantly older than those without atherosclerosis with a p-value of <0,001 as showed in Table 2.

Our study revealed that only hypertension, increased BMI, proteinuria, hypercholesterolemia and increased LDL cholesterol, all have a significant increase in subclinical atherosclerosis in T2DM patients, (p-value, 0.007, <0,001, <0,001, 0.001 and <0,001 respectively) also revealed that uncontrolled DM, increased disease duration, high levels of FBG, 2hPPBG and HbA1c all have significant increased proportion of subclinical atherosclerosis in DM patients (p-value, 0.005, 0.001, 0.007, 0.005 and 0.001 respectively) as showed in Table 2.

According to SLE diabetic patients, there was not any significant proportion difference across the sex distribution; however SLE diabetic patients with subclinical atherosclerosis were significantly older than those without atherosclerosis with p-value 0.001 as showed in Table 3.

Our study showed that only hypertension, increased BMI, serum creatinine, proteinuria, serum albumin, hypercholesterolemia and

increased LDL, all have a significant increase in subclinical atherosclerosis in SLE diabetic patients, (p-value, <0,001, <0.001, 0.002, <0.001, 0.033, <0,001 and <0,001 respectively). Also uncontrolled DM, increased DM duration, high levels of FBG, 2hPPBG and HbA1c, SLE activity and increased SLE duration all have significant increased proportion of subclinical atherosclerosis in SLE diabetic patients (p-value, 0.005, <0.001, 0.006, 0.001, 0.001, <0.001and 0.002 respectively) as showed in Table 3.

According to all studied groups, the proportion of subclinical atherosclerosis was significantly more in all studied populations (the SLE, diabetic and SLE diabetic groups) in comparison to the control one with percentage of 23%, 25%, 46% and 6% respectively.

Also our study showed that the proportion of subclinical atherosclerosis in Group III is significantly more than that in both groups I and II as showed in Fig. 1.

4. DISCUSSION

SLE is an autoimmune disease that has important effects on life expectancy and is known to be associated with an increased risk of cardiovascular diseases. CVD in SLE are very common but in most of cases they are not severe [10].

DM is a disease of abnormal carbohydrate metabolism that is considered to be an independent CVD risk factor. On the other hand, although insulin resistance and hyperglycemia are very important in progression of CVD in diabetic patients, there are several ways to leading of CVD, as low HDL, small and dense LDL, chronic and low grade inflammation, endothelial dysfunction, more aggregated platelets, more diffuse coronary atherosclerosis, and so on. So the develop and severity of CVD in diabetics does not has a simple explanation [11].

The results of our study showed that 22% of our SLE patients (group I) showed subclinical atherosclerosis; this finding is in agreement with Berti, et al. [12] who proved that SLE patient have an increased cardiovascular risk detected by the carotid IMT [12] and Henrot, et al. [13].

SLE patients with subclinical atherosclerosis were significantly older than those without atherosclerosis. These findings are in agreement with Tselios, et al. [14] who found that CVD incidence is higher in old age SLE patient and more common in males than females due to the protective effect of female hormones [14].

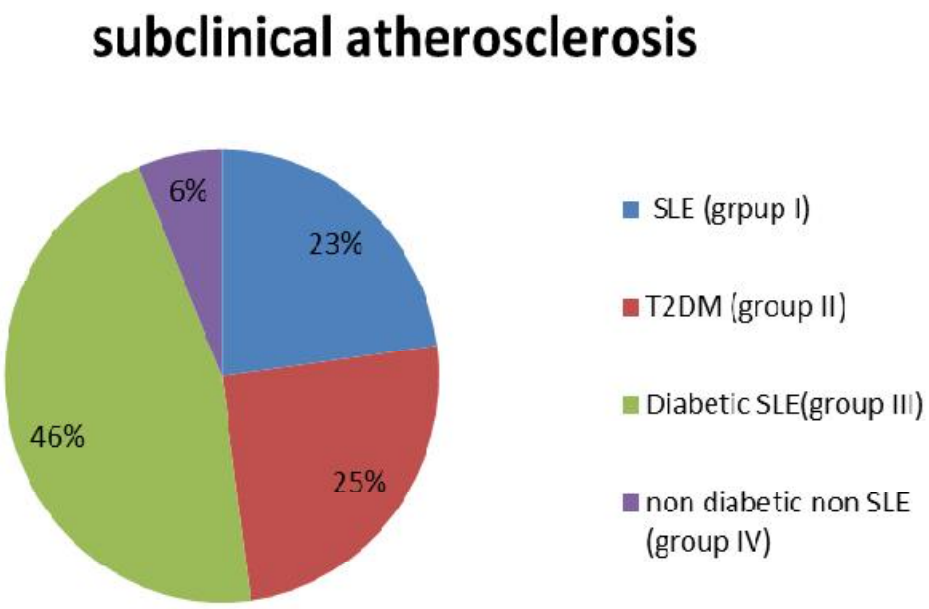

Fig. 1. Prevalence of Subclinical atherosclerosis across studied groups

Table 1. Demographic, clinical and laboratory characteristics of the patients with systemic lupus erythematous

Patient criteria			Subclinical atherosclerosis			Test of significance
			Present	Absent	Total	*P value*
			n=11 (22%)	n=39 (78%)	n=50 (100%)	
Sex	Female	Count (%)	8(72.7%)	31(79.5%)	39 (78.0%)	0.633
	Male	Count (%)	3(27.3%)	8 (20.5%)	11 (22.0%)	
Age (years)	Median (IQR)		50 (18)	42 (5)	42.5(7)	<0,001*
Hypertension	Yes	Count (%)	6(54.5%)	7 (17.9%)	13 (26.0%)	0.023*
	No	Count (%)	5(45.5%)	22(82.1%)	27 (74.0%)	
C-reactive protein (CRP)(mg/dL)	Positive	Count (%)	2(18.2%)	11(28.2%)	13 (26.0%)	0.503
	Negative	Count (%)	9(81.8%)	28(71.8%)	37 (74.0%)	
body mass index(BMI)	Median (IQR)		27(3)	25(2)	25(2)	0.004*
Serum creatinine(mg/dL)	Median (IQR)		1.4(0.4)	1.3(0.3)	1.35(0.3)	0.169
Proteinuria (mg/24h)	Median (IQR)		336(110)	158(138)	212(165)	<0,001*
Hemoglobin (gm/dL)	Median (IQR)		11(3)	11.6(3)	11.4(3)	0.419
Platelet (10^3/mm3)	Median (IQR)		154(193)	214(105)	212(146)	0.336
ALT (IU/L)	Median (IQR)		15(53)	18(9)	18(10)	1.000
AST (IU/L)	Median (IQR)		22(20)	19(8)	19(10)	0.698
Serum albumin (g/dL)	Median (IQR)		3.4(0.5)	3.6(0.6)	3.5(0.6)	0.283
Prothrombin time (seconds)	Median (IQR)		12.7(1)	12.7(1)	12.7(1)	0.471
Total cholesterol (mg/dL)	Median (IQR)		235(64)	182(61)	183(68)	0.004*
LDL (mg/dL)	Median (IQR)		106(27)	89(20)	92.5(24)	0.006*
Triglycerides (mg/dL)	Median (IQR)		153(70)	93(118)	102(113)	0.214
ESR 1st hour (mm/h)	Median (IQR)		24(27)	24(14)	25.5(17)	0.127
ESR 2nd hour (mm/h)	Median (IQR)		72(55)	48(27)	55(38)	0.132
SLE activity (using SLEDAI score)	Active	Count (%)	9(81.8%)	14(35.9%)	23 (46.0%)	0.007*
	Not active	Count (%)	2(18.2%)	25(64.1%)	27 (54.0%)	
SLEDAI score	Median (IQR)		12(5)	5(1)	5(3)	<0,001*
Disease duration	Mean (SD)		10(4)	5(2)	5(3)	<0,001*

**Significant as P value <0.05, ALT: Alanine aminotransferase, AST: Aspartate aminotransferase*

Table 2. Demographic, clinical and laboratory characteristics of the patients with T2DM

Patient clinical and laboratory criteria			Subclinical atherosclerosis			p value.
			Present	Absent	Total	
			n=12 (24%)	n=38 (76%)	n=50 (100%)	
Sex	Female	Count (%)	5(41.7%)	18(47.4%)	23(46.0%)	1.000
	Male	Count (%)	7(58.3%)	20(52.6%)	27(54.0%)	
Age (years)	Median (IQR)		56(10)	48.5(7)	51(9)	0.001*
Hypertension	Yes	Count (%)	9(75.0%)	11(28.9%)	20(40.0%)	0.007
	No	Count (%)	3(25.0%)	27(71.1%)	40(60.0%)	
Body mass index (BMI)	Median (IQR)		31(13)	26.36(2)	27(4)	<0,001*
Serum creatinine(mg/dL)	Median (IQR)		1.65(1.2)	1.4(0.3)	1.45(0.)	0.072
Proteinuria (mg/24h)	Median (IQR)		446.5(300)	205.6(135)	219.7(183)	<0,001*
Hemoglobin (gm/dL)	Median (IQR)		10.8(1)	11.8(2)	11(2)	0.041
Platelet (10^3/mm3)	Median (IQR)		255(160)	215(98)	215(95)	0.334
ALT (IU/L)	Median (IQR)		34.5(26)	44.5(10)	42(10)	0.173
AST (IU/L)	Median (IQR)		37(18)	38.5(8)	38(10)	0.8
Serum albumin (g/dL)	Median (IQR)		3.5(0.1)	3.55(0.5)	3.5(0.4)	0.076
Prothrombin time (second)	Median (IQR)		12.17(2)	12.6(1)	12.6(1)	0.460
Total cholesterol (mg/dL)	Median (IQR)		244(141)	182(52)	189.5(66)	0.001*
LDL (mg/dL)	Median (IQR)		120(48)	89(20)	98(28)	<0,001*
Triglycerides (mg/dL)	Median (IQR)		161.5(64)	136.5(111)	154(63)	0.453
DM control	Uncontrolled	Count (%)	10(83.3%)	14(36.8%)	24(48%)	0.005*
	Controlled	Count (%)	2(16.7%)	24(63.2%)	26(52%)	
DM duration (years)	Mean (SD)		10(4)	6.5(4)	8(4)	0.001*
FBG (mg/dL)	Median (IQR)		205(96)	147.5(37)	154(63)	0.007*
2hPPBG (mg/dL)	Median (IQR)		353.5(179)	241.5(66)	253.5(133)	0.005*
HbA1c (%)	Median (IQR)		10.75(4)	6.5(4)	8(4)	0.001*

**Significant as P value <0.05*

Table 3. Demographic, clinical and laboratory characteristics of the SLE diabetic patients

Patient Demographic, clinical and laboratory criteria		Subclinical atherosclerosis			p value.
		Present	Absent	Total	
		n=22 (44%)	n=28 (56%)	n=50 (100%)	
Sex	Female Count (%)	15(68.2%)	24(85.7%)	39(78.0%)	0.178
	Male Count (%)	7(31.8%)	4(14.3%)	11(22.0%)	
Age (years)	Median (IQR)	64(13)	41.5(5)	42.5(7)	0.001*
Hypertension	Yes Count (%)	15(68.2%)	3(10.7%)	18(36.0%)	<0,001*
	No Count (%)	7(31.8%)	25(89.3%)	32(64.0%)	
C-reactive protein (CRP) (mg/dL)	PositiveCount (%)	6(27.3%)	7(25.0%)	13(26.0%)	0.554
	NegativeCount (%)	16(72.7%)	21(75.0%)	37(74.0%)	
Body mass index (BMI)	Median (IQR)	28(7)	25(2)	26(3)	<0,001*
Serum creatinine(mg/dL)	Mean (SD)	1.6(0.29)	1.39(0.15)	1.5(0.3)	0.002*
Proteinuria(mg/24h)	Median (IQR)	356(280)	190.15(147)	266.5(187)	<0,001*
Hemoglobin(gm/dL)	Median (IQR)	11.6(2)	11.9(3)	11.7(3)	0.158
Platelet (10^3/mm3)	Median (IQR)	154(193)	274.5(115)	43(11)	0.165
ALT (IU/L)	Mean (SD)	40.6(10.6)	42(8.62)	41.4(9.47)	0.598
AST (IU/L)	Mean (SD)	44.45(10.19)	41.6(8.29)	42.8(9.19)	0.275
Serum albumin (g/dL)	Median (IQR)	3.4(0.4)	3.8(0.5)	3.5(0.6)	0.033*
Prothrombin time (second)	Median (IQR)	12.7(1)	12.6(2)	11.67(1)	0.722
Total cholesterol (mg/dL)	Median (IQR)	248.5(60)	179(72)	210(87)	<0,001*
LDL (mg/dL)	Median (IQR)	133.5(45)	92.5(25)	105(44)	<0,001*
Triglycerides (mg/dL)	Median (IQR)	102(93)	112.5(139)	102(113)	0.434
ESR 1st hour (mm/h)	Median (IQR)	32.5(25)	29.5(24)	30(24)	0.695
ESR 2nd hour (mm/h)	Median (IQR)	65(51)	60(47)	63(49)	0.814
DM control	UncontrolledCount (%)	16(72.7%)	9 (32.1%)	25 (50.0%)	0.005*
	ControlledCount (%)	6 (27.3%)	19 (67.9%)	25 (50.0%)	
DM duration	Median (IQR)	11 (5)	6 (4)	8 (5)	<0,001*
FBG (mg/dL)	Median (IQR)	193.5 (85)	146 (34)	154 (78)	0.006*
2hPPBG (mg/dL)	Median (IQR)	367 (172)	199.5 (70)	233 (200)	0.001*
HbA1c (%)	Median (IQR)	9(3.5)	7.15(1)	7.65(2.4)	0.001*
SLE duration	Median (IQR)	7.5(5)	5(2)	6(3)	0.002*
SLEDAI score	Median (IQR)	8.5(6)	5(2)	6(3)	<0,001*
SLE activity	Active Count (%)	19(86.4%)	8(28.6%)	27(54.0%)	<0,001*
	Not activeCount (%)	3(13.6%)	20(71.4%)	23(46.0%)	

**Significant as P value <0.05*

Our study found that there was significant statistically association between hypertension and IMT (carotid & femoral) in SLE patients. These findings are in agreement with Tektonidou, M. G., et al. [15].

Our study showed that there was a significant association between proteinuria and IMT in SLE patients. This finding is in agreement with Atukorala, I., (2015) whose proved that subclinical CVD detected by thickened carotid IMT is increased in patients with lupus nephritis, and patients with lupus nephritis have an abnormality in their endothelial function, more disease activity and more persistent inflammation that increase risk of atherosclerosis and CVD [16].

Our study showed that there was a significant association between hypercholesterolemia and increased LDL and IMT in SLE patients. These findings are in agreement with Ajeganova, S., (2019) [17]. Also our study revealed that SLE activity using SELDAI score and increased disease duration have a significantly increased proportion of subclinical atherosclerosis in SLE patients as the increased disease activity and duration result in repeated chronic inflammation and steroid exposure or alternatively, patients may also be more exposed to atherosclerotic damage like other organs, all of these help in atherosclerosis progression [18]. These results are in agree with Haque, [19] and Soliman et al. [12].

The results of our study showed that 24% of our T2DM patients (group II) showed subclinical atherosclerosis, this finding is in agreement with Owoeye, S. C. et al. [20] who proved that carotid IMT were significantly higher in the diabetic patients than in the controls [20].

The univariate analysis of our results revealed that older age, increased BMI, proteinuria, hypoalbuminemia, hypercholesterolemia, increased LDL and hypertension; all have a significant statistically association to subclinical atherosclerosis in T2DM patients. These results are in agreement withWijaya, (2016) who proved that albuminuria and dyslipidemia increases carotid IMT and CVD risk in type 2 diabetic patients [21].

Our study revealed that increased disease duration, uncontrolled DM, increased FBS, 2hPPBS and HbA1c levels all have significant increased proportion of subclinical atherosclerosis in DM patients. This finding is in agreement with, Lee SW et al. [22] and Ghari et al. [23] who proved that HbA1c levels were positively associated with carotid atherosclerosis, as assessed by carotid IMT.

Like our study, Moemenam [24], and Soud, M. I. A., et al. [25] reported that diabetes duration was associated with a higher carotid IMT in T2DM patients.

The results of our study showed that 44% of our SLE diabetic patients (group III) showed subclinical atherosclerosis. These results are in agreement with Ohmura, et al. [26] who proved that the SLE diabetic patients were expected to have significantly higher cardiovascular events [26]. The fact is that those patients have two independent CVD risk factors.

The univariate analysis of our results revealed that that older age, increased BMI, proteinuria, hypoalbuminemia, increased serum creatinine, hypercholesterolemia, increased LDL and hypertension; all have a significant statistically association to subclinical atherosclerosis in SLE diabetic patients.

At the same time, our study showed that the proportion of subclinical atherosclerosis is significantly higher in all studied populations than in the non-SLE non-diabetic control one with p-value of 0.021, 0.012 and <0,001 and odds ratio estimates of 4.419, 4.947 and 12.31 in groups I (SLE patients), group II (T2DM patients) and group III (SLE diabetic patients) respectively. At the same time, our results showed that the proportion of subclinical atherosclerosis in Group III was significantly more than that in both groups I and II with p-value of 0.019 and 0.035 respectively, while this difference failed to be significant between group I and group II (p value 0.812).These results are in agreement with Ohmura, et al. [26] who proved that The SLE diabetic patient is expected to have significantly higher cardiovascular events [26]. The fact is that these patients have two independent CVD risk factors so it would be interesting to study the frequency of CVD with a controlled trial of SLE alone and SLE with DM.

Our results have important clinical implications in the routine care of SLE patients, a comparable increased risk of atherosclerotic plaques between SLE and DM patients indicates that SLE patients may need CVD risk management measures similar to DM. Meticulous long-term

management of modifiable traditional cardiovascular risk factors; along with efforts to minimize disease-related factors may improve cardiovascular outcomes in patients with SLE. Further research should be aimed at developing SLE-specific CVD risk stratification tools, including subclinical atherosclerosis ultrasonography data, as well as clarifying the benefit of potential preventative therapies in higher-risk patients. As the SLE diabetic patient is likely to be at significantly higher risk for CVD, we recommend good management of CVD risk factors in diabetic SLE patients.

The limitations of the study were small sample size and being single center study. Further studies on large number of patients are recommended to determine the risk of CVD in SLE, DM, and combined SLE diabetic patients.

5. CONCLUSION

Our results support that Doppler examination of the carotid and femoral arteries provides a useful non-invasive technique to measure the IMT for detection of premature atherosclerosis. SLE is associated with increased risk of premature atherosclerosis and CVD similar to DM. The occurrence of premature atherosclerosis increases with increased SLE activity and disease duration. Also subclinical atherosclerosis in SLE diabetic patients was significantly more than that in both SLE patients and diabetic patients. So control of disease activity and prevention of organ damage can help to reduce incidence of atherosclerosis in SLE patients.

CONSENT AND ETHICAL APPROVAL

This study was cross sectional survey study, which was performed at the departments of internal medicine and radiology, Tanta University Hospitals (a tertiary hospital), in the period from August 2017 to August 2018, after obtaining an informed written consent and the approval from Tanta University Ethical Committee.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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