Al-Azhar Med. J . (Medicine). *Vol. 52 (1), January, 2023, 173 - 186*
DOI: 10.21608/amj.2023.273690
https://amj.journals.ekb.eg/article_273690.html

GLUCOSE INTOLERANCE IN RHEUMATOID ARTHRITIS AND SYSTEMIC LUPUS ERYTHEMATOSUS PATIENTS

By

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ABSTRACT

Background: Glucose intolerance is an important contributor to the increased cardiovascular risk attributed to the metabolic syndrome, a constellation of cardiovascular risk factors that includes central obesity, dyslipidemia, hypertension, and disturbed glucose metabolism in patients with rheumatoid arthritis or systemic lupus erytheromatosus.

Objective: To assess glucose intolerance percentage in rheumatoid arthritis and systemic lupus erythreromatosus patients.

Patients and methods: The present study was a prospective study conducted on 90 subjects. The studied patients were recruited from Internal Medicine Clinic at Al-Hussein University Hospital during the period from January 2020 to January 2021. Patients were divided into three groups, group I: thirty patients with Systemic lupus erythromatosus (SLE), group II: thirty patients with Rheumatoid arthritis (RA) group III: included thirty healthy individuals as control group. The following laboratories were done for all groups to assess glucose intolerance (CBC, CRP, ESR, Fasting blood sugar(FBG),Post prandial blood sugar (PPBS), Haemoglobin A1C (HbA1C), Complement3 (C3), Complement4 (C4) and Albumin/creatnin (ALB/Creat ratio).

Results: There were statistically significant difference between studied groups as regard blood glucose level assessment (FBS, PPBS & HbA1C), glucose intolerance, hemoglobin, ESR, Albumin/Creatinine ratio, complement 3, glucose intolerance and (FBS, PPBS & HbA1C) in SLE, glucose intolerance and (FBS, PPBS& HbA1C) in RA group, glucose intolerance and hemoglobin in SLE group however there were no statistically significant difference between studied groups as regard CRP, WBCs, PLTs and C4.

Conclusion: SLE and RA patients appeared to have higher incidence of glucose intolerance than normal subjects.

Keywords: Glucose intolerance, Rheumatoid arthritis, Systemic lupus erytheromatosus.

INTRODUCTION

Systemic lupus erytheromatosus (SLE) is a chronic, multifaceted inflammatory disease that can attack every organ system of the body. SLE is protean in its manifestations and follows a relapsing and remitting course. Rheumatoid arthritis (RA) is a chronic systemic inflammatory disease of unknown etiology. The classic feature of this disease is persistent symmetric polyarthritis that usually involves the peripheral joints in a symmetric distribution but can affect any

joint lined by a synovial membrane *(Gazareen et al., 2014)*.

Rheumatology is a medical science devoted to the study of rheumatic diseases that include a range of musculoskeletal and systemic disorders that share the clinical involvement of joins and periarticular tissues. Rheumatoid arthritis (RA) is a systemic, autoimmune disorder that causes chronic synovial inflammation of multiple joints affecting 0.5–1% of population all over the world *(Balasubramanyam et al., 2014)*.

It affects women three times more than the men. Recent studies have shown increasing prevalence of dysglycemia in rheumatoid arthritis patients. Impaired glucose handling in RA patients is secondary to peripheral insulin resistance mediated by the inflammatory response. Role of various pro-inflammatory cytokines (including tumor necrosis factor [TNF] and interleukin-6 [IL-6]) in RA, insulin resistance (IR), and type 2 diabetes mellitus (T2DM) has been reported by several independent studies *(Hoet and Tripathy, 2016)*.

RA patients with diabetes mellitus (DM) prevalence rate was about 15% to 19%, which was significantly higher than the prevalence rate of 4% to 8% of global middle-aged population DM *(Simard and Mittleman, 2011)*.

In a study, which consists of 48,718 cases of RA patients and 40,346 cases of non-rheumatic subjects, the incidence of RA patients with DM was 0.86% higher than the 0.58% in the control group which was observed, and DM risk was 1.5-fold in RA patients when compared with control group *(Solomon et al., 2010)*.

Consistently, a study described that abnormal glucose metabolism in RA patients was up to 46% after 2 years when compared with the time point of recruitment *(Hoes et al., 2011)*.

Systemic lupus erytheromatosus is a systemic connective tissue disorder affecting mainly females. Female: male ratio was 9:1 with peak onset in the second and third decade. Systemic inflammation has been suggested as the main physiologic link between IR and SLE *(Escarcega et al., 2010)*.

The aim of the present study was to assess glucose intolerance percentage in rheumatoid arthritis and systemic lupus erytheromatosus patients.

PATIENTS AND METHODS

The present study was a prospective study conducted on 90 subjects. The studied patients were recruited from Internal Medicine Clinic at Al-Hussein University Hospital during the period from July 2020 to January 2021.

Patients were classified into three equal groups:

- **Group I:** Patients newly diagnosed with SLE based on positive ANA and anti-ds DNA tests.
- **Group II:** Patients with rheumatoid arthritis disease based on American Criteria for Rheumatoid Arthritis.
- **Group III:** Apparently healthy subjects not known to chronic diseases.

Exclusion criteria:

1. Patients having co-morbid illness like diabetes mellitus, hypertension, and coronary artery disease.

2. Family history of DM.
3. Patients on steroid treatment.

All patients were subjected to full history taking, physical examination (general and local), and laboratory investigations included:

- CBC was performed using automated CELL-DYN Ruby hematology analyzer.
- Blood glucose tests (fasting blood glucose, 2h postprandial blood glucose and HBA1c).
- C-reactive protein (CRP).
- Erythrocyte sedimentation rate (ESR).
- Complement (C3, C4).
- Alb / creat ratio.

Statistical analysis:

The collected data were coded, processed and analyzed using the SPSS (Statistical Package for the Social Sciences) version 22 for Windows® (IBM SPSS Inc, Chicago, IL, USA). Data were tested for normal distribution using the Shapiro Wilk test. Qualitative data were represented as frequencies and relative percentages. Chi square test ($\chi 2$) to calculate difference between two or more groups of qualitative variables. Quantitative data were expressed as mean ± SD (Standard deviation). Independent samples t-test was used to compare between two independent groups of normally distributed variables (parametric data). Mann–Whitney U test was used when comparing between two means (for abnormal distributed data). Kruskal wills test was used when comparing between more than two means (for abnormal distributed data). P value < 0.05 was considered significant.

RESULTS

There were statistically significant difference between studied groups as regard blood glucose level assessment (FBS, PPBS& HbA1C), glucose intolerance, Hb, ESR, ALB/Creat ratio, C3, glucose intolerance and (FBS, PPBS & HbA1C) in SLE, glucose intolerance and (FBS, PPBS & HbA1C) in RA group, glucose intolerance and Hb in SLE group but no statistical significant difference between studied groups as regard CRP, WBCs, PLTs and C4.

There was no statistical significant difference between studied groups as regard WBCs and PLTs. There was a statistically significant difference between studied groups as regard ALB/Creat ratio. There was a statistically significant difference between studied groups as regard C3. There was no statistical significant difference between studied groups as regard C4 **(Table 1)**.

Table (1): Comparisons between studied groups as regard demographic data, blood glucose level assessment, glucose intolerance, ESR & CRP, CBC, ALB/Creat ratio, and C3 & C4

Parameters \ Groups		SLE (n = 30)		RA (n = 30)		Control (n = 30)		Stat. test	P-value
Sex	**Male**	9	30%	9	30%	6	20%	X^2 = 1.02	0.6
	Female	21	70%	21	70%	24	80%	X^2 = 1.02	
Age (years)	**Median**	21.5		40.5		37.5		KW = 28.06	**< 0.001**
	IQR	17 – 30.25		30 – 48.3		21 – 47.3		KW = 28.06	
FBS (mg/dl)	**Median**	95.5		109.5		86		**KW** 10.1	**0.006**
	IQR	86.3 – 114.3		86.8 – 117.3		79.8 – 94		10.1	
PPBS (mg/dl)	**Median**	104		144		99		9.2	**0.01**
	IQR	95.5 – 167.5		101 – 166.5		90 – 120.5		9.2	
HbA1C (%)	**Median**	5.3		5.7		5.05		14.9	**0.001**
	IQR	5 - 6		5.3 – 6.1		4.8 – 5.4		14.9	
Glucose intolerance	**No**	19	63.3%	14	46.7%	26	86.7%	**Stat. test** X^2 = 10.7	**0.005**
	Yes	11	36.7%	16	53.3%	4	13.3%	X^2 = 10.7	
ESR (mm/h)	**Median**	33.5		22.4		15		KW 11.8	**0.003**
	IQR	15.8 – 50		10 – 40		10 – 25		11.8	
CRP (mg/L)	**Median**	5		7		5		3.79	0.150
	IQR	2 – 8.25		4 – 11.25		4 - 9		3.79	
Hb (g/dl)	**Median**	11.25		11		12.3		**KW** 6.04	**0.049**
	IQR	10.4 – 13		9.9 – 13		11.8 – 13.6		6.04	
WBCs ($x10^3$/ul)	**Median**	5.8		5.5		5.6		0.46	0.794
	IQR	4.75 – 7.9		4.6 – 7.5		4.9 – 7.02		0.46	
PLTs ($x10^3$/ul)	**Median**	237.5		289		259.5		4.05	0.132
	IQR	154.3 – 318.8		233 – 401.5		189.3 – 287.5		4.05	
ALB / Creat	**Median**	26		15.5		15.5		**KW** 14.4	**0.001**
	IQR	17 - 81		10 – 25		10 – 24.3		14.4	
C3 (mg/dl)	**Median**	104.5		98		116.5		**KW** 6.2	**0.045**
	IQR	77 – 129		86.8 – 114.3		94.5 – 150		6.2	
C4 (mg/dl)	**Median**	31		33.5		34		2.45	0.294
	IQR	23.8 – 39.3		25 – 41		28.8 - 40		2.45	

There was no statistical significant relation between glucose intolerance and age, ESR, CRP, WBCs, PLTs, ALB/Creat ratio, C3 and C4 in SLE group. There was a statistical significant relation between glucose intolerance and FBS, PPBS and HbA1C in SLE group. There was a statistically significant relation between glucose intolerance and Hb in SLE group **(Table 2)**.

Table (2): Post-Hoc test for multiple comparisons between studied groups as regard significant laboratory data

Parameters \ Groups		SLE vs RA	SLE vs Control	RA vs Control
age	**LSD**	-16.2	-12.4	3.8
	p-value	< 0.001 HS	< 0.001 HS	0.177 NS
FBS	**LSD**	-3.7	8.6	12.3
	p-value	0.336 NS	0.025 S	0.002 S
PPBS	**LSD**	-10.7	15.7	26.4
	p-value	0.203 NS	0.063 NS	0.002 S
HbA1C	**LSD**	-0.2	0.3	0.5
	p-value	0.087 NS	0.015 S	< 0.001 HS
ESR	**LSD**	6.4	16.8	10.4
	p-value	0.214 NS	0.001 S	0.044 S
Hb	**LSD**	-0.1	-1.1	-1.0
	p-value	0.84 NS	0.028 S	0.045 S
ALB/Creat	**LSD**	95.0	96.9	1.9
	p-value	0.001 S	0.001 S	0.947 NS
C3	**LSD**	-3.9	-19.2	-15.2
	p-value	0.626 NS	0.019 S	0.62 NS

As regard age, there were significant differences between SLE and RA and control groups, while no statistical significant difference between SLE and RA groups.

As regard FBS, there were no statistical significant difference between SLE and RA groups, while significant difference between SLE, RA and Control groups.

As regard PPBS, there were no statistical significant difference between SLE, RA and control groups, while significant difference between SLE and RA groups.

As regard HbA1C, there were no statistical significant difference between SLE and RA groups, while significant difference between SLE, RA and Control groups.

As regard ESR, there no statistical significant difference between SLE and RA groups, while there were significant difference between SLE, RA and Control groups.

As regard Hb, there were no statistical significant difference between SLE and RA groups, while there were significant difference between SLE and Control group's significant difference between SLE and RA groups.

As regard ALB/Creat, there were statistically significant differences between SLE, RA and control groups. But no statistical significant difference between SLE and RA groups.

As regard C3, there were no statistical significant difference between SLE and RA groups but there were a significant

difference between SLE and control groups **Table (3)**.

Table (3): Relation between glucose intolerance & studied data in SLE group

SLE groups \ Glucose intolerance		No (n = 19)	Yes (n = 11)	Stat. test	P-value
Age (years)	**Mean**	24.3	22.5	MW = 96	0.735
	±SD	9.3	7.0		
FBS (mg/dl)	**Mean**	89.3	115.5	T = 11.1	**< 0.001**
	±SD	5.7	7.1	T = 11.1	
PPBS (mg/dl)	**Mean**	98.6	170.5	T = 17.4	**< 0.001**
	±SD	10.5	11.4	T = 17.4	
HbA1C (%)	**Mean**	5.1	6.1	T = 9.5	**< 0.001**
	±SD	0.3	0.2	T = 9.5	
ESR (mm/h)	**Mean**	33.3	37.7	MW = 97.5	0.767
	±SD	18.5	25.1		
CRP (mg/L)	**Median**	4	7	MW = 73	0.185
	IQR	2 - 7	2 – 29	MW = 73	
Hb (g/dl)	**Mean**	12.0	10.3	T = 2.26	**0.032**
	±SD	2.1	1.8	T = 2.26	
WBCs ($x10^3$/ul)	**Mean**	6.7	5.7	MW = 79	0.287
	±SD	2.6	2.1		
PLTs ($x10^3$/ul)	**Mean**	247.7	246.7	MW = 97	0.767
	±SD	126.3	95.2		
ALB / Creat	**Median**	25	27	MW = 89	0.525
	IQR	16 – 123	20 – 67	MW = 89	
C3 (mg/dl)	**Median**	110	78	MW = 64	0.085
	IQR	89 - 132	70 - 128	MW = 64	
C4 (mg/dl)	**Mean**	31.5	30.1	MW = 90	0.553
	±SD	9.4	8.1		

There was no statistical significant relation between glucose intolerance and age, ESR, CRP, Hb, WBCs, PLTs, ALB/Creat ratio, C3 and C4 in RA group.

There was a statistical significant relation between glucose intolerance and FBS, PPBS and HbA1C in RA group **(Table 4)**.

Table (4): Relation between glucose intolerance & studied data in RA group

RA groups \ Glucose intolerance		No (n = 14)	Yes (n = 15)	Stat. test	P-value
Age (years)	**Mean**	40.4	39.4	MW = 108	0.886
	±SD	10.7	10.4		
FBS (mg/dl)	**Mean**	86.6	116.5	T = 18.4	**< 0.001**
	±SD	4.0	4.8	T = 18.4	
PPBS (mg/dl)	**Mean**	105.9	161.7	T = 10.3	**< 0.001**
	±SD	16.2	13.2	T = 10.3	
HbA1C (%)	**Mean**	5.2	6.1	T = 9.5	**< 0.001**
	±SD	0.3	0.2	T = 9.5	
ESR (mm/h)	**Median**	23.8	32.8	MW = 77.5	0.154
	IQR	8.75 – 33.5	16.3 – 43.8	MW = 77.5	
CRP (mg/L)	**Median**	15.1	11.2	MW = 107	0.854
	IQR	4 – 11.25	4.3 – 11.5	MW = 107	
Hb (g/dl)	**Mean**	11.9	11.1	T = 1.25	0.221
	±SD	1.8	1.8	T = 1.25	
WBCs (x10³/ul)	**Mean**	6.4	5.7	MW = 82	0.331
	±SD	2.2	2.2		
PLTs (x10³/ul)	**Median**	324.6	338.4	MW = 111	0.984
	IQR	246 – 370	185 – 441.5	MW = 111	
ALB / Creat	**Median**	18.9	19.3	MW = 87.5	0.313
	IQR	12.3 – 25.3	10 -22.3	MW = 87.5	
C3 (mg/dl)	**Mean**	105.7	107.9	MW = 104.5	0.759
	±SD	29.1	31.9		
C4 (mg/dl)	**Mean**	31.1	34.4	MW = 93	0.448
	±SD	10.2	8.6		

There was no statistical significant relation (p-value > 0.05) between glucose intolerance and studied laboratory data except for (FBS, PPBS and HbA1C) **(Table 5)**.

Table (5): Relation between glucose intolerance and studied data in control group

Glucose intolerance / Control group		No (n = 26)	Yes (n = 4)	MW	p-value
Age (years)	**Mean**	36.9	30.0	38.5	0.425
	±SD	13.6	11.5		
FBS (mg/dl)	**Mean**	85.4	121.8	0.0	< 0.001
	±SD	6.7	3.3		
PPBS (mg/dl)	**Mean**	100.0	169.3	0.0	< 0.001
	±SD	14.0	15.2		
HbA1C (%)	**Mean**	5.0	5.9	0.0	< 0.001
	±SD	0.3	0.1		
ESR (mm/h)	**Mean**	16.2	31.3	44.5	0.659
	±SD	9.7	34.2		
CRP (mg/L)	**Mean**	5.7	6.3	44.5	0.659
	±SD	3.2	2.5		
Hb (g/dl)	**Mean**	12.5	12.6	42.5	0.576
	±SD	1.3	2.7		
WBCs (x10³/ul)	**Mean**	6.3	6.2	41.5	0.536
	±SD	1.8	2.7		
PLTs (x10³/ul)	**Mean**	252.9	313.3	28	0.157
	±SD	76.2	81.9		
ALB / Creat	**Mean**	17.4	16.5	46	0.746
	±SD	9.1	9.3		
C3 (mg/dl)	**Mean**	123.2	114.8	47	0.791
	±SD	32.0	19.5		
C4 (mg/dl)	**Mean**	34.2	34.5	50.5	0.930
	±SD	6.7	6.6		

DISCUSSION

In the present study in comparing between studied groups as regard demographic data, there was a statistical significant difference, between SLE and RA groups, and between SLE & control groups as regard age with. No statistical significant difference between studied groups as regard sex. This was in agreement with *El-gendi et al. (2018)* who reported that no significant sex difference between studied diseased groups. This result was in contrast with *Julie and Chaim (2012),* who showed that SLE typically affects females more than males. However, male SLE patients often have more severe disease than females.

In the current study, there were statistically significant differences between studied groups as regard blood glucose level assessment (FBS, PPBS & HbA1C). This result was in agreement with *Chung et al. (2010)* and *Gazareena et al. (2014)* who reported that RA patients have significantly higher fasting blood glucose, fasting insulin than SLE patients. This can be explained by some

factors such as older age in RA patients than SLE and longer duration of disease in RA patients reflecting the burden of such disease.

In the current study, there was a statistically significant difference between studied groups as regard ESR. However, there was no statistical significant difference between studied groups as regard CRP. These results were in agreement with *Seriolo et al. (2010)* and *Ormseth et al. (2012)* who reported that there were statistically significances higher ESR and CRP in RA patients than in controls. Also, these results were in agreement with *Lozovoy et al. (2011)*, who showed that SLE patients with hyperinsulinemia had significantly higher ESR and CRP.

In the present study, there was a statistically significant difference between studied groups as regard Hb, while, no statistical significant difference between SLE and RA groups. There were statistically significant differences between SLE and control groups and statistically significant difference between RA and control groups. These results were in agreement with *Rattarittamrong et al. (2016)* who reported that 64% of SLE patients in the study suffered from anemia.

Anemia was found in about 50% of SLE patients. Many mechanisms contribute to the development of anemia, including inflammation, renal insufficiency, blood loss, dietary insufficiency, medications, haemolysis, infection, hypersplenism, myelofibrosis, myelodysplasia, and aplastic anemia that is suspected to have an autoimmune pathogenesis *(Schett et al., 2010)*.

On the contrary, *Levine and Erkan (2011)* disagreed with the current study, as they reported leukocyte abnormalities in up to 75% of the patients in the study. Also, *El-gendi et al. (2018)* reported that that WBCs, MCV and MCH had no significant different between difference groups.

In the present study, there was a statistically significant difference between studied groups as regard ALB/Creatinine ratio, SLE and RA groups, SLE and control groups. However, there was no statistical significant difference between RA and control groups with. These results were in agreement with *Sui et al. (2014)* who reported that those patients with inactive SLE nephritis had significantly higher 24h-protien in comparison to those without nephritis and the control group.

In the current study, there were statistically significant difference between studied groups as regard C3, statistically significant differences between SLE and control groups with, and RA and control groups.

These results were in agreement with the results of *El-gendi et al. (2018)* who reported that patients without SLE nephritis had significantly higher level of C3 and C4 in comparison to control group. There results were disagreed with *Narayanan et al. (2010)* who observed in their prospective study that 92.3% SLE patients had low C3 levels, and 84.6% had low C4 levels. *Birmingham et al. (2010)* demonstrated the poor clinical utility of serial serum C3 or C4 measurements alone to forecast or identify an SLE renal flare. The reasons for the discrepancies across studies are multifactorial including differences in study design, ethnicity,

baseline clinical characteristics and renal parameters.

In the present study, there were statistical significant relations between glucose intolerance and FBS, PPBS and HbA1C in SLE group, and RA group. These results were in agreement with *Magadmi et al. (2010)* who reported that SLE patients had significantly worsened insulin resistance than healthy control patients. Also, *Gazareen et al. (2014)* reported that, with respect to insulin sensitivity profile, SLE patients have significantly higher fasting insulin, HOMA IR, HOMA b-cell, and C-peptide than controls, and there is a positive correlation between IR and fasting glucose, HOMA b-cell, and c-peptide in SLE patients. This relationship is independent of age, sex, BMI, total cholesterol, LDL, and HDL. There was a statistically significant higher ESR and CRP in RA patients with IR, and there was positive correlation between IR and fasting insulin, ESR, and serum CRP. This was similar to what was reported by other investigators such as *Gheita et al. (2012)* who found that SLE patients had high HOMA IR and HOMA b-cell and are associated with increase in disease activity and damage. In contrast, *Ormseth et al. (2012)* found no statistically significant difference between SLE patients and controls with respect to HOMA IR.

This did not agree with the studies by *Karimi et al. (2011),* and *Stagakis et al. (2012),* who found no statistically significant higher ESR and CRP in RA patients with and without IR depending on activity of disease.

CONCLUSION

SLE and RA patients appeared to have higher incidence of glucose intolerance than normal subjects.

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قابلية إختلال السكر فى مرضى الروماتويد المفصلى والذئبة الحمراء

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خلفيـــة البحـــث: يعـــد قابليـــة اخـــتلال الســـكر مســـاهماً مهمـــاً فـــي زيـــادة مخـــاطر القلـــب والأوعيـــة الدمويـــة المنســـوبة إلـــى متلازمـــة التمثيـــل الغـــذائي، وهـــي مجموعـــة مـــن عوامـــل الخطـــورة القلبيـــة الوعائيـــة التـــي تشـــمل الســـمنة المركزيـــة، وخلـــل شـــحميات الـــدم، وارتفـــاع ضـــغط الـــدم، واســـتقلاب الجلوكـــوز المضـــطرب، فـــي المرضـــى الـــذين يعـــانون مـــن التهـــاب المفاصـــل الروماتويدي و الذئبة الحمراء.

الهـــدف مـــن البحـــث: تقيـــيم نســـبة قابليـــة إخـــتلال الســـكر فـــي مرضـــى إلتهـــاب المفاصل الروماتويدي ومرضى الذئبة الحمراء.

المرضـــى وطـــرق البحـــث: الدراســـة الحاليـــة عبـــارة عـــن دراســـة إســـتباقية أجريـــت علـــى 90 شخصًـــا. تـــم إســـتقدام المرضـــى الخاضـــعين للدراســـة مـــن عيـــادة الباطنـــة العامـــة بمستشـــفى الحســـين الجـــامعي خـــلال الفتـــرة مـــن ينـــاير2020 إلـــى ينـــاير 2021. تـــم تقســـيم المرضـــى الـــى ثلاثـــة مجموعـــات: ثلاثـــون مريضـــا مصـــاب بالروماتويـــد المفصـــلى وثلاثـــون مريضـــا مصـــاب بالدئبـــة الحمـــراء وثلاثـــون شـــخص طبيعـــى وتـــم عمـــل الفحوصـــات ومقارنـــة المجموعات الثلاث.

نتـــائج البحـــث: كـــان هنـــاك فرقـــا يعتـــد بـــه إحصـــائيا بـــين المجموعـــات المدروســـة فيمـــا يتعلـــق بتقيـــيم مســـتوى الســـكر فـــي الـــدم (ســـكر صـــائم وســـكر فـــاطر وســـكر تراكمـــى)، وقابليـــة اخـــتلال الســـكر، هيموجلـــوبين، ســـرعة

الترسيب، نسبة البومين/كرياتنين، الاختبار التكميلى3في مرضى الذئبة الحمراء، كذلك هناك فرقا يعتد به احصائيا وقابلية اختلال السكر و (سكر صائم، سكر فاطر و سكر تراكمى) في مجموعة الروماتويد المفصلى، ولكن لا يوجد فرق إحصائي مهم بين المجموعات المدروسة فيما يتعلق البروتين المتفاعل سي و كرات الدم البيضاءوالصفائح الدموية و الاختبار التكميلى 4.

الاستنتاج: مرضى الروماتويد المفصلى والذئبة الحمراء يمتلكون معدلات أعلى لحدوث إختلال السكر من الاشخاص العاديين.

الكلمات الدالة: إختلال السكر، إلتهاب المفاصل الروماتويدي، الذئبة الحمراء الجهازية.