# Frequency and Patterns of Cardiac Involvement in Systemic Lupus Erythematosus

**Abstract:**

The aim of the study was to determine the prevalence of myocardial and circulatory complications among lupus patients, such as pericarditis, heart failure, endocarditis, ventricular dysfunction, and Myocarditis. It was also to assess the association of lupus with increased myocardial injury and to evaluate therapeutic ( choriokinin, corticosteroids, and glucocorticoids) interventions to reduce the complication of cardiovascular disease.

This study focused on the immunological aspect of the effect of immunosuppressive therapy in systemic lupus erythematous (SLE), specifically choriokinin, corticosteroids, and glucocorticoids (GCs), on the exposure of cardiovascular disease. The study period extended from 2022 to early 2024 and relied on the results of screening of 88 SLE patients and a control group of 85 patients. The causes of heart damage in SLE patients were identified to assess the relationship with disease activity, duration, and rheumatic treatment. Previously, we first determined N\_terminal prohormone of brain natriuretic peptide (NT\_proBNP) levels in SLE patients not receiving specific rheumatic treatment and identified a relationship between biomarker and immunological marker concentrations of SLE activity (increased serum levels of anti cardiolipin IgG, anti\_dsDNA, antinuclear antibodies, and decreased complement C4) and markers reflecting impaired kidney function [1, 2]. Corticosteroids are effective in preventing the progression of SLE and increasing patient survival, while reducing the exposure of atherosclerosis and thrombosis [1, 3, 4] and hypercholesterolemia [2, 5], ultimately reducing a exposure of cardiovascular disease at SLE patients, despite isolated reports of cardiac toxicity [4].

Noticed a statistical difference between the two groups in NT\_proBNP, as it was shown that the second group had a lower NT\_proBNP result 0.05 than the first group, and the same applies to Anti\_JO1, which gives us evidence beyond doubt that immunosuppressive treatments work positively in treating heart muscle diseases resulting from lupus.

**Keywords :** immunosuppressant , SLE , immunosuppressive, Myocarditis , BNP , ACA , Anti\_JO1

# Introduction:

Systemic lupus erythematous (SLE) is a chronic, multisystem immunological disorder of unknown causality and genetic predisposition it presents with diverse clinical features and follows an unpredictable progression [1]. Myocarditis affects more than 55% of SLE patients and is a leading cause of fatal condition. All cardiac anatomical structures can be affected by SLE, such as the pericardium, endocardium, coronary vessels, and myocardium, with heart failure (HF) being the final stage of cardiac and circulatory disease [2].

Recent studies indicate that systemic lupus erythematosus (SLE) is a major risk factor for Myocarditis and has a significant impact on causing actual damage to the myocardic, pulmonary valve, and ventricular, endocardial, and pericardial dysfunction, which requires continuous monitoring in the intensive care unit( ICU) [3 , 11] . alarming results have been recorded for deaths due to heart failure in several countries around the world. In the Middle East, Saudi Arabia occupied an advanced position in deaths, with 36% of deaths due to heart muscle failure due to pericarditis, followed by Egypt with a rate of 21.5% of deaths due to myocardial infarction, and Jordan with a rate of 12% due to myocardial infarction[14]. while the death rate due to heart failure in Latin America was recorded in 2023 alone, more than 470 deaths due to Myocardial infarction resulting from lupus, in Mexico there are no official statistics on these cases, but medical investigative reports suggest that the percentage is increasing due to myocardial infarction[13 , 14]. the American Heart Association reported that approximately 2 million patients suffer from fatal Myocardial infarctions, most of whom are women of childbearing age. There are Myocardial infarctions that have become chronic and life-threatening, and diseases resulting from hypertension and repeated Myocardial infarction [12, 14].

The pathogenesis of cardiac damage in SLE is thought to depend on the combined or independent effects of chronic immunological disorder inflammation, accelerated progression of atherosclerosis, and the use of certain ant rheumatic drugs [3]. According to an epidemiological study (n = 500 people), in patients with

systemic lupus erythematous M cardiac damage was more frequently recorded

[6]: arrhythmia and electrical disturbances in 22% and 6%, coronary Myocarditis - in 13% and 4%, major valve insufficiency in 11% and 2%, myocardial infarction (MI) - in 6% and 2%, cor pulmonale in 2.2% and 0.2%, heart failure (HF) in 1.0% .

In Iraq, there are no statistics that show us the death rate resulting from systemic lupus erythematosus. The last statistics for Iraq reached 11% in 1989, and Baghdad, the capital, topped the list. All deaths were among fertile women between the ages of 25 - 45, and the statistics indicated myocardial infarction.

After years of war, annual statistics were absent. In this research, I focused on studying the effect of lupus on the heart muscle for the year 2022 to the beginning of 2024, in terms of the effect of lupus on heart function within the available medical data and for the same age group in 1989.

# Methodology:

The study included 88 patients, 80 (92%) females and 8 (9.5%) males, with a mean age of 35 years, making it an ideal sample for the study. A control group of 85 patients was selected. We did not care about gender matching, although the majority of participants in the control group were female and had similar symptoms and conditions to the study group. The patients were monitored at Al- Mawaddah Private Hospital and private laboratories specializing in immunodiagnostics from 2022 to 2024.

1. The medical history of each participating patient was analyzed, including confirmation of lupus and cardiac symptoms such as impaired carbohydrate metabolism and cardiac and circulatory complications in patients with rheumatoid arthritis and systemic lupus erythematosus.
2. Identifying the immune damage caused by lupus other than cardiac muscle, to help us understand the context of cardiac damage, whether direct or indirect (Table 1)
3. All participants underwent an ECG and Echo-cardiography to confirm cardiac involvement or detect signs of myocardial ischemia ( Table 2).
4. Laboratory and immunological examinations of the participants were performed to provide a clear statistical picture.

# Note / This study paid special attention to the immunological aspect of the effect of systemic lupus erythematosus on the cardical seeking to understand the role of autoimmune diseases (lupus) on the cardic muscle.

**Table 1: key immune related manifestation of condition**.

|  |
| --- |
| main immunological manifestations of diseases |
| SLEDAI\_2K | 4- 15 coordinates |
| SLICC\_DI (damage index) | 0- 1 coordinates |
| hematological disorders | 22% |
| nephritis | 31% |
| arthritis | 25% |
| skin lesions | 22% |
| sororities | 22% |
| antinuclear factor (ANF) | 94%, antibodies to double-strandedDNA (anti\_dsDNA) |
| ant phospholipid syndrome | 76% of patients ( positive) |
| Sjogren's syndrome | 7%( positive) |

**Figure 1: Volumetric Effect Of Immunological disorder Manifestations Of The Disease**

sororities

18%

hematological disorders

18%

skin lesions

18%

nephritis

25%

arthritis

21%

hematological disorders

nephritis

arthritis

skin lesions

sororities

**Table 2 : Immunodiagnostic with systemic lupus erythematous cautilizing myocardial infection**

|  |  |  |
| --- | --- | --- |
| N. SLE | Immunological disorder cardiomyopathyby systemic lupus erythematous | Rate values |
| 16 | pericardium | 26% |
| 18 | Adhesive pericarditis | 30% |
| 15 | Exudative pericarditis | 10% |
| 4 | acute myocarditis | 5% |
| 5 | Ischemic Myocarditis (IHD) | 3% |
| 10 | Heart Failure (HF) | 15% |
| 2 | myocardial infarction (MI) | 1% |
| 16 | cardiac arrhythmias | 9% |
| 2 | patients with SLE respectively | 1% |

**Figure 2: values cardiomyopathy by systemic lupus erythematous**

Adhesive pericarditis

patients with SLE cardiac arrhythmias respectively

12% 1%

myocardial infarction (MI)

1%

Exudative pericarditis

acute myocarditis

Adhesive pericarditis

41%

Ischemic Heart Disease (IHD) Heart Failure (HF)

20%

Heart Failure (HF)

myocardial infarction (MI)

Ischemic Heart Disease

(IHD)

4%

cardiac arrhythmias

Exudative pericarditis

14%

patients with SLE respectively

acute myocarditis

7%

# Exclusion Criteria:

This study specifically targeted patients aged 30 ± 15 years, based on the statistical system issued by the Iraqi Ministry of Health before 1989 regarding the rate of infections and deaths from heart diseases resulting from lupus.

The following categories were excluded:

1. The following categories of people under the legal age of 18 were excluded for legal considerations, which require the research body to obtain judicial approval and be under judicial supervision
2. Those over 45 years of age were excluded, as most of the study participants were women and must be fertile. As is the practice in current research regarding lupus, participants must be no older than 45.
3. those with comorbidities such as tumors, malignancies, or infectious diseases

The medical reports provided by cardiologists were reviewed for all patients, and they had conventional hazard factors for cardiac and circulatory disease .

Transthoracic echocardiography was performed. Patients were evaluated for left ventricular diastolic and systolic function, ejection fraction (EF), left ventricular myocardial thickness, chamber sizes, systolic pulmonary artery and diastolic pressure , pericardial status, and valves.

# Laboratory and Statistical Diagnosis:

Concentration of the N-amino terminal fragment of (probrain- natriuretic – peptide) (NT\_proBNP) in serum was determined by electrochemical analysis (ECL) on a Cobas analyzer utilizing the Elecsys proBNP II test system.

Typically, NT\_proBNP levels are ≤125 pg/ml, according to procedural guidelines [8]. Statistical analysis was performed utilizing parametric and nonparametric statistical methods of Applied Statistics NT\_proBNP in the two groups is equal to

12. Variables were presented as medians (ME), with the upper and lower quartiles indicated in parentheses (25th percentile; 75th percentile). The reliability of variations between the two groups was assessed utilizing the (SPSS software) criterion,. Qualitative indicators were compared in two unrelated

groups in association table utilizing the χ2 test. The significance level was p < 0.05 [9].

# Procedures:

median duration of SLE was two years [, with moderate disease activity (SLEDAI\_2K) 9 coordinates [4] , and the SLICC\_DI damage index was 2 coordinates [ 2]. The main clinical manifestations of the disease embraced hematological disorders (49%), nephritis (45%), arthritis (37%), skin lesions (34%), and synovitis (33%). Eighty-six patients (98%) had immune disorders: elevated levels of antinuclear factor (ANF) were recognition in 95%, and anti\_dsDNA antibodies were recognition at 78% of SLE patients. Associated antiphospholipid syndrome (allergic fungal sinusitis (AFS) and Sjögren's syndrome were identified in 9 (9%) and 10 (11%) patients, respectively. [10]

Valvular regurgitation with varying degrees of regurgitation (not requiring surgical treatment) was the most frequently observed cardiac abnormality in systemic lupus erythematous (SLE), identified in 79 patients (90%): 78 patients (89%) with tricuspid regurgitation, 72 patients (82%) with mitral regurgitation, 57 patients (7%) with pulmonary artery regurgitation, and 15 patients (14%) with aortic regurgitation. Endocarditis was identified in 26 patients (30%), and mitral or tricuspid valve prolapse in 30 patients (34%). The rarest was mitral, tricuspid, and aortic stenosis, identified in one patient (1%). The median concentration of NT\_proBNP was 92 (27–332) pictograms per milliliter, an elevated level of NT\_proBNP (> 125 pictograms per milliliter) was identified in 29 patients (32%).

The possible association of cardiac disorders with therapy used in SLE, patients had divided into two groups.

* **Group 1** : embraced 43 patients (40 women - 3 men) with a median age of 30 ± 15 years who, at time of examination, had not received glucocorticoids (GC), immunosuppressant
* **Group 2** : embraced 45 patients (40 women - 5 men), the median age was 30 ± 15 years patients took a variety of combinations of these drugs . choroquine, corticosteroids at a dose of 150 mg/day. Among them were both newly ill and

long-term patients with SLE, but who canceled previously prescribed therapy. HA was received by 44 (98%) of them, the median dose was 20 mg /day when calculated for prednisone. During the entire period of the disease, cyclophosphamide was used in 23 (48%) patients, azathioprine in 17 (38%),

mycophenolate mofetil in 16 (36%), methotrexate in 9 (19%), choroquine ,

corticosteroids in 43 (96%).

# Results immunological:

Immunology and laboratory characteristics of patients of both groups are presented in Table2. The groups were comparable in age and gender, women predominated (93% - 90%).

Patients of the 2nd group had a longer duration of illness than participants of the 1st group p < 0.00001), less activity (SLEDAI\_2K and 12 coordinates, p < 0.001), a higher damage index (SLICC\_DI 1 - 0 coordinates, p < 0.001), they exhibited a lower incidence of skin lesions (11% and 57%, p < 0.0001), joints (22% - 52%, p < 0.05) and hematological disorders (24% and 73.8%, p < 0.0001) .

The main immunology examination manifestations of SLE in group 1 patients have hematological disorders (74%), skin lesions (57%), kidneys (55%) and joints (52%), all also revealed immunological disorders an increase the level of antinuclear factor (ANF) in 100%, anti dsDNA in 80% patients.

* **Group 1 :** Nephritis (36%), sororities (29%), hematological disorders (24%) and

arthritis (22.2%) .

* **Group 2:** Immunological disorders have identified in 96% of patients. no relevant variations were identified in the concomitant Sjogren's syndrome and Allergic Fungal Sinusitis (AFS).

This, division of patients with SLE into groups upon taking ant rheumatic therapy (immunosuppressant, GIBP) allowed us to assess its relationship with the activity and duration of SLE, since the groups differed relevantly in these indicators.

value level of NT\_proBNP in untreated patients was elevated than in group 2nt (151 - 33 pictograms per milliliter, respectively, p < 0.01), while exceeding normal values

# Note / that the mean value of and the mean value of the control group is equal to 4, noting that P. value NT\_proBNP equal 0.05 mean value lupus value is equal 19.37

**Table 3 : Comparison immunological and laboratory characteristics of patients of both groups are presented**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| **Characters** | **SLE (n = 88)** | **Control (n=85)** | **P. value** | **Notes** |
| NT\_proBNP (mean) | 19.37 | 4 | 0.05 | Based on the assumption that the overall mean for both groups is equal to 12, the probability value indicates thedifference in statistical significance |
| Age (mean ± SD) | 30 ± 15 | 30 ± 14 | <0.001 | There is a difference in the statistical results regarding the distribution of ages |
| Gender (male) | 3 (10%) | 5 ( 4%) | <0.0001 | The statistical difference inresults is very high between the two genders |
| Gender ( female ) | 40 (90%) | 40 ( 96%) | <0.0001 | The statistical difference in results is very high betweenthe two genders |
| Total sample size | 88 | 85 |  |  |
| N. of individualswith determined gender | 43 | 45 |  |  |

# Anti\_ENA (Extractable Nuclear Antibodies):

antibody ranges intermediate to SLE sufferers and manipulate organization is supplied in table 5. The evaluation outcomes reveal there haven’t extensive variations ( P > 0.015 ) within the detection results of Anti\_JO1 and anti\_Scl70 antibody <0.001 intermediate to SLE patients and manage institution. but, the SLE patient institution exhibited relevantly raised of antiRNP levels <0.001 , anti\_Sm

<0.001 , anti SSA, and anti SSB compared with group of control, with all variations being statistically relevant (P < 0.01). These results in the bottom

**Table 4 : Comparison Levels Anti\_ENA**

|  |  |  |  |
| --- | --- | --- | --- |
| Antibody | SLE (n = 88) | Control (n = 85) | P. values |
| Anti\_JO1 |  |  |
| Negative | 6(7%) | 78(92%) | <0.001 |
| Positive | 82(93%) | 7(8%) |
| Anti-RNP |  | <0.001 |
| Negative | 13(15%) | 72(85%) |
| Positive | 75(85%) | 13(15%) |
| Anti\_Scl70 |  | 0.15 |
| Negative | 77(88%) | 81(95%) |
| Positive | 11(12%) | 4(5%) |
| Anti\_Sm |  | <0.001 |
| Negative | 66(75%) | 83(98%) |
| Positive | 22(25%) | 2(2%) |
| Anti\_SSA |  | <0.001 |
| Negative | 35(40%) | 68(80%) |
| Positive | 53(60%) | 17(20%) |
| Anti\_SSB |  |  | 0.005 |
| Negative | 72(82%) | 77(90%) |
| Positive | 16(18%) | 8(10%) |

# Analysis of Associate Chartered Accountant (ACA) levels:

Results comparing ACA levels in patients with SLE to those in the control group are outlined in Table 6. The analysis revealed that positive rate of ACA antibodies had increased over time , ACA (IgA, IgG, IgM), are elevated in SLE group compared

to the control group, with these variations achieving statistically relevant (P < 0.05) as shown in Table 6.

* + P25 represents the first quartile of the 25% percentile.
	+ P75 represents the middle quartile of the 75% percentile.
	+ Interquartile range (IQR) measure of spread and dispersion shows the least effect of infection and the result is the first quartile - the middle quartile.

**Table 5 : Comparison of ACA detection results between SLE group and the control group.**

|  |  |  |  |
| --- | --- | --- | --- |
| ACA (n (%) | SLE (n = 88) | Control (n = 85) | P. values |
| Negative | 59(67%) | 53(62%) | <0.001 |
| Positive | 29 (33%) | 32(38%) |
| Antibody | Median | IQR = P75- P25 | P. values |
| ACA IgA (P75-P25) | 5 (7.67-2.32) | 5.35 | 0.017 |
| ACA IgG (P75-P25) | 6 (8.75-2.63) | 6.12 | 0.001 |
| ACA IgM (P75-P25) | 9 (13.51-4.82) | 8.69 | 0.004 |

# B2\_GPI Autoantibodies levels:

When detecting antibodies B2\_GPI Autoantibodies and comparing the results with the lupus disease and control group, it was identified that positive results for antibodies B2\_GPI Autoantibodies were higher in lupus patients compared to the control group as shown in Table 7

**Table 6 : Comparison of B2\_GPI Autoantibodies detection results between SLE group and control group .**

|  |  |  |  |
| --- | --- | --- | --- |
| **antibody** | **SLE (n = 88)** | **Control (n = 85)** | **P. value** |
| B2\_GPI Autoantibodies | 25(28%) | 14(17%) | <0.001 |
| IgA ( median) | 8(12.94-4.08) | 6(8.36-2.77) | <0.001 |
| IgG ( median) | 6(8.36-2.8) | 5(7.84-2.53) | <0.001 |
| IgM ( median) | 6(8.35-3) | 5(7.84-2.53) | <0.001 |

# Result and Discussion:

The influence of immunological disorder inflammation in the endocardium, valves, aortic dissection and hypertension and HA therapy are discussed as possible causes of these changes. The use of HA can lead to rapid "healing" of inflammation of a valve especially of the heart (valvulitis). [6]

Despite the fact that bacterial or nonbacterial thrombotic endocarditis is a characteristic classic valve lesion in SLE[13], most researchers agree that the most common change in heart valves is insufficiency with varying degrees of regurgitation[14]. Some indicate a high incidence of aortic valve insufficiency, others indicate mitral valve insufficiency [2].

In my study, tricuspid and mitral valve insufficiency was more common, and aortic valve insufficiency was less common; the changes were not clinically relevant and did not require surgical correction.

An increase in the level of natriuretic peptide (BNP) is not specific for myocarditis, Based on the dynamics of the NT\_proBNP level, the effectiveness of treatment is evaluated and a prognosis is made. While maintaining its high concentration, it can be assumed that there is active inflammation, its carbonization. Therefore, according to the recommendations immunologic and cardiologists, all patients with clinical suspicion of myocarditis should conduct a study of the initial level of NT\_proBNP and its dynamics [4].

They do not have any special features, and associations with SLE-specific autoantibodies and therapy have also not been identified. In the present study, rhythm disturbances were recognition in 18% of patients

The number of atherosclerotic plaques in the coronary and femoral arteries is twice as high in patients with disease as in people suffering from rheumatoid arthritis and DM diseases with high cardiovascular hazard [5].

In my study, I did not observe a statistical difference in the incidence of various Myocarditiss and heart failure between those who received immunosuppressant and high blood pressure treatments and those who did not receive them, but the difference was clear in those who developed lupus.

98% of cases, HA was used, hypertension, an increase in total cholesterol and BMI values were more often observed, which confirms the opinion about the effect of HA on the frequency of TFR.

Currently, the determination of the concentration of NT\_proBNP is used for the screening of HEART FAILURE (HF), assessment of its severity and prognosis, as well as monitoring the effectiveness of therapy. In addition, a high level of NT\_proBNP is an independent hazard factor not only for Heart Failure (HF),

There are isolated studies on the concentration of BNP NT\_proBNP in immunological disorder inflammatory diseases. In SLE, the level of this biomarker was determined only in patients receiving pathogenesis therapy [ 3,6]. The concentration of BNP NT\_proBNP in patients with SLE was higher than in the control group, this increase was primarily associated with myocardial dysfunction, but was not associated with vascular damage, including atherosclerotic, inflammatory markers, and SLE activity.

# Conclusion:

Despite the young age of patients, therapy (primarily with glucocorticoids, HA) and a long duration of certain SLE are linked to a higher prevalence hazard factors (hypertension, hypercholesterolemia, overweight), and myocarditis A practicing rheumatologist should pay attention to the need for joint management of patients with SLE with a cardiologist and immunological , assessment of markers of Heart Failure (HF) as a potentially fatal complication, especially in patients with high disease activity under medical control of TFR utilizing a minimum dose of HA during remission and low activity.

# Limitations:

1. Despite the extreme importance of the study, we were unable to include larger areas, at least in southern Iraq, due to legal restrictions on allowing data collection and involving a larger study sample.
2. Medical records were used to collect information for patients, especially the control group.
3. There is no similarity between patients in the severity and duration of the injury
4. Limited laboratory materials and CT scan results for patients prompted us to communicate directly with patients for re-evaluation.

# Recommendations:

* 1. Raising awareness among doctors about the risks of lupus and its direct impact on heart function, especially in its early stages.
	2. It is necessary to deepen the studies and establish a longitudinal study to increase understanding of the effect of lupus on the heart.
	3. Encourage teamwork among cardiologists, rheumatologists, and immunologists to develop an early picture of lupus disease progression and the potential for cardiac damage.
	4. Requiring lupus patients to undergo heart function tests even if they do not show heart symptoms and making it a mandatory work routine

**Table 7 : Operational definition**

|  |  |  |
| --- | --- | --- |
| Immunologicaltest | Examination (Manufacturer) | Aim |
| SLLICC\_DI | N/N | The aim of the examination is to assess the amount of accumulated damage to theorgans resulting from lupus |
| Anti\_dsDNA | ELISA test ( thermo fisher scientific ) | It aims to detect autoantibodies, which are responsible forattacking the body's tissues. |
| NT\_proBNP | Cobas (Beckman Coulter) | The purpose of screening is to diagnose a heart condition by detecting the level of peripheralbrain peptide |
| ACA | ELISA test (Euroimmun) | The aim is to detect anti- centromere antibodies responsible for attacking healthy tissues in an immunologicaldisorder disease. |
| Anti\_ENA | ELISA test (Inova) | It aims to detect antibodies tonuclear antigens and is used to |

|  |  |  |
| --- | --- | --- |
|  |  | detect Sjögren's syndrome andpolymyositis. |
| SLEDAI\_2K | N/N | The index is used to evaluate lupus disease and helps doctors assess the patient's response totreatment. |
| Anti\_JO\_1Antibody | ELISA test (Inova) | Type of Anti \_ENA test |
| Anti\_RNPAntibody | ELISA test (Inova) | Type of Anti \_ENA test |
| Anti\_SCI70 Antibody | Topoisomerase test | The aim of detecting SCI70 antibodies in the blood is todiagnose systemic scleroderma. |
| Anti\_Sm \_Antibody | ELISA test (Inova) | Type of Anti \_ENA test |
| Anti\_SSAAntibody | ELISA test (Inova) | Type of Anti \_ENA test |
| Anti\_SSBAntibody | ELISA test (Inova) | Type of Anti \_ENA test |
| B2\_GPIAutoantibodies | ELISA test (Euroimmun) | The aim of detecting antiphospholipid syndrome is to detect antibodies to beta-2protein. |

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**Ethical Approval:**

As per international standards or university standards written ethical approval has been collected and preserved by the author(s).

**Consent**

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

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# Reference:

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