Review Article

The Interaction between Systemic Lupus Erythematosus and Cardiovascular Disease: Systematic Review

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ABSTRACT

Patients diagnosed with systemic lupus erythematosus (SLE) face a much greater risk of morbidity and mortality from cardiovascular disease (CVD) when compared to the overall population. Despite numerous case reports and literature reviews delving into this connection, there are only a handful of systematic reviews that have specifically concentrated on this link. As a result, the objective of this systematic review is to assess the depth of the relationship between SLE and cardiovascular diseases. An extensive search was conducted primarily using PubMed and following PRISMA criteria. The search targeted English-language studies investigating the link between SLE and cardiovascular diseases. Clear inclusion and exclusion criteria were set to guarantee the quality and relevance of the evaluated research. The research encompassed a broad spectrum of studies from various global regions, with no particular emphasis on any specific gender or age. An evident trend revealed a significant proportion of SLE patients experiencing cardiovascular conditions. According to the findings of our investigation, in general, it was discovered that patients with SLE have a greater chance of developing CVD. It is essential to conduct additional research on the connection between identifiable SLE-specific risk factors that can be modified and the likelihood of developing CVD in order to bolster the creation of preventative and treatment measures.

Keyword: Systemic Lupus Erythematous, Cardiovascular Diseases, SLE, Myocardial, Lupus, Risk Factors.

Introduction

Systemic Lupus Erythematosus is an autoimmune illness that affects a significant portion of the global population. In 916 AD, a skin condition was originally described with the word "lupus," and Biett provided the first precise description of lupus erythematosus in 1833. Erythema centrifugum was the word used to describe it at the time of reporting. But since the turn of the 20th century, a clearer picture of SLE and its accompanying symptoms has emerged [1].

Multiple organs within the human body can be affected by SLE, exhibiting a range of clinical symptoms and severity levels (mild, moderate, and severe) [2]. Antibodies, participation of any number of organ systems, and a wide range of clinical symptoms are characteristics of this condition. These antibodies are generated by SLE and are directed against the host of self-molecules found in the cytoplasm, nucleus, and cell surfaces.

Access this article online	
Quick Response Code:	Website: www.smh-j.com
	DOI: 10.54293/smhj.v4i3.115

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Received: 3 Jul 2024 **Accepted:** 28 Aug 2024

Please cite this article as: Osman Mirghani H, Mansour M Alatawi N, Turki Alenzi M, Ahmed I Alghamdi I, Waad Salmah J Alatwai, Saleh M Alqarni G, Imad M Bahkali R, Suliaman E Alatawi N, Fraih N Alenezi A, Rand Fahad M Albaridi, Turki M Alhamrani M, Ali A Alshamrani S, Mansour F Althobiti A. The Interaction between Systemic Lupus Erythematosus and Cardiovascular Disease: Systematic Review. SMHJ [Internet]. 2024;4(3):169-180.

These include DNA that is anti-double stranded (ds-DNA), antinuclear antibodies (ANAs), present in over 95% of cases, and anti-Smith (ant-Sm) antibodies [3]. Obtaining the correct diagnosis greatly depends on the identification of these autoantibodies. Vasculitis, immune complex deposition, band or occlusive vasculopathy, inflammation, and blood vessel anomalies are the primary histological characteristics of SLE. The lack of immunological tolerance to self-antigens in this syndrome is caused by many reasons. These elements include immunological, endocrine, genetic, and environmental ones. As a result, autoantibodies are produced, which harm tissue in a variety of ways [4]. This pathogenic process is described as the activation of autoimmunity resulting from exposure to environmental stimuli and a disruption of tolerance in genetically susceptible individuals. Patients with SLE who experience cardiac involvement are more likely to experience morbidity and death, and it can have a detrimental effect on all parts of the heart and circulatory system, comprising the coronary arteries, the pericardium, valves, conveying system, and myocardium [5]. A significant contributor to the morbidity and death of lupus patients is known to be heart disease (CVD). In 1976, Urowitz et al. made the first discovery that people with SLE had an increased risk of heart disease, when they reported a bimodal pattern of death in their Toronto SLE cohort [6]. Six of the 11 fatalities in their group happened within a year of diagnosis, and the cause was identified as active SLE. At an average age of 8.6 years, four of the five patients who passed away were found to have had a fatal myocardial infarction (MI) [6]. All five patients had recently suffered from a MI. Further research has validated this bimodal pattern of cardiovascular disease-related mortality. There are significant differences in epidemiological estimates among different age, racial, and ethnic groups. The average ratio of females to males is around 10:1, the illness is more common among younger women [7].In a meta-analysis done on 2017, the incidence of SLE, which is generally uncommon, was shown to vary from 0.3/100,000 to 23.2/100,000 person-years worldwide, with North America reporting the highest incidence, whilst Europe and Australia had considerably lower rates. In some nations, the incidence is rising; however, this might be attributed to more accurate diagnosis. Interesting variations exist between ethnic groups as well. For example, SLE is said to be less prevalent in Hispanic and Asian populations are more prevalent among African and Arab people, and even less prevalent in Caucasians. It appears that estimates of prevalence differ significantly more from estimates of incidence. Different genetic susceptibilities, environmental, and socioeconomic variables, as well as variations in research design, categorization, and diagnoses, may all contribute to the variability [8]. Multiple pathophysiological pathways interact to cause cardiovascular symptoms in SLE, which raise morbidity and death rates in SLE patients. Endothelial cell dysfunction is brought on by a defect in endothelial cell activation, which leads to lectin-like oxydized low-density lipoprotein receptor 1 (LOX1). Through its ability to stimulate the synthesis of tumor necrosis factor-alpha (TNF-α), interleukin 6 (IL-6), and interleukin 12 (IL-12), the inflammatory substances receptor LOX-1 raises the risk of CVDs in people with SLE. These cytokines are required to attract monocytes to the cells of the vasculature. Another pathway for heart disease in SLE patients is impaired innate immunity. Low-density granulocytes (LDG), a subpopulation of pro-inflammatory neutrophils associated with lupus that are seen in greater quantities in SLE and that stimulate endothelial cell injury in vitro, are linked to this immunological dys-regulation. These LDGs cause neutrophil extracellular traps (NETs) to form, which promotes the development of thrombus and unstable coronary plaque. The impairment of adaptive innate immunity is a third cause of cardiac illness in sickle cell disease (SLE). This happens as a result of CD4+ T cells and other T lymphocytes being over-activated, which encourages thrombus formation and vascular damage through IFN-1 signaling [9]. As a result, CVD plays a significant role in SLE morbidity and mortality. It is brought on by a number of variables that have atherogenic or prothrombotic characteristics. The conventional risk factors, which include diabetes, hyperlipidemia, and hypertension, are to be lessened by well-established treatments used in the overall prevention of CVD. Recently, there has been positive data reported on the use of statins in both hyperlipidemia and APS in SLE. Naturally, statins were developed to precisely target the LDL receptor (LDLR) in order to reduce LDL levels. As a result, they are widely acknowledged as an essential medication for the treatment of CVD, including primary and secondary prevention [10]. There is a paucity of data on statin therapy in SLE. While microRNA mechanisms are relatively distinct, PCSK9 inhibition has shown comparable effects to statins on the activation of plaque T cells produced by OxLDL [11]. Additionally, OxLDL stimulates PCSK9 in DC,

particularly in SLE patients compared to controls, and PCSK9 is linked to disease activity in SLE. Therefore, PCSK9 may have an immune function in SLE. Crucially for SLE management, hydroxychloroquine also surprisingly contributes to CVD prevention. A recent meta-analysis revealed that hydroxychloroquine was able to lower the risk of thromboembolic events by 49% [12]. Limited information is available from controlled investigations regarding the potential effects of small molecule medicines, a type of therapy for SLE, on cardiovascular disease and atherosclerosis. It is worth noting that new biologic medicines have significantly advanced the management of rheumatoid arthritis and other rheumatic conditions; however, this progress has not been as pronounced for SLE, despite the availability of belimumab and rituximab, which target B-cell signaling pathways [13]. Thus, the aim of this systematic review is to assess the strength of the relationship between cardiovascular illnesses and SLE. In addition, the evaluation will look at possible risk factors for cardiovascular disorders in SLE patients and investigate how various treatment approaches affect cardiovascular outcomes. Additionally, the review will assess the quality of existing evidence and highlight any gaps in the current literature, providing recommendations for future research in this area.

Methods

For the purpose of this comprehensive investigation, We followed the PRISMA (favored Reporting Items for Systematic Evaluations and Meta-Analyses) guidelines.

Study Design with Duration: This comprehensive review was finished in the month of December in the year 2023.

Search strategy: A thorough exploration was carried out in five prominent databases, consisting of Look for relevant studies using EBSCO, Google Scholar, PubMed, Web of Science, and Science Direct. Our search was restricted to English and considered the individual criteria of each database. In order to pinpoint relevant research, the terms "lupus, cardiovascular disease, systematic lupus erythematous, immune system, atherosclerosis" were transformed into words for PubMed Mesh. To link the important phrases, the boolean operators "OR" and "AND" were used. The findings of the search encompassed publicly available articles, clinical trials involving humans, and publications in the English language.

Selection criteria

Inclusion criteria: For the purpose of this evaluation, we took into account the subsequent aspects:

- Investigation on lupus and cardiovascular disease
- Absence of gender and age limitations
- Availability of complimentary articles.

Exclusion criteria: Editorial letters, case studies, and rebuttals to disputes were not included in the selection process. Additionally, submissions in foreign languages were not taken into account. The selection process was also limited to articles published within a certain time frame and in specific journals or databases. This was done to ensure that the articles chosen were of high quality and met the criteria for the study. The researchers also conducted a thorough analysis of the articles, looking for common themes and trends in the research. This allowed them to draw meaningful conclusions about the state of the field and identify areas for future research. Overall, the selection process was rigorous and comprehensive, ensuring that only the best and most relevant articles were included in the study.

Data extraction: The results of Rayyan's research method revealed duplicates (QCRI). Examining the titles and abstracts to determine their importance, The investigators combed through all of the search results using a number of inclusion/exclusion criteria. All papers satisfying the inclusion requirements went through a thorough review process by the evaluators. The authors detailed different approaches for addressing conflicts after conducting a comprehensive analysis. They have the ability to view the titles, authors, year of study, country, participants, gender, drug dosage, key findings, and conclusions of the studies.

Strategy for data synthesis: Data from relevant studies were used to create a thorough description of the study's results and components. Once the data was retrieved, the best method for using the information from the study papers that were part of the systematic review was selected.

Evaluation of bias risk: The non-randomized treatment trials were assessed for quality using the ROBINS-I risk of bias assessment method. Seven topics were taken into account: confusing, selection of participants, categorization of interventions, departures from planned treatments, incomplete data, assessment of outcomes, and choosing of results to be published.

Results

Search outcomes: A thorough investigation revealed a total of 910 research papers, and after eliminating 360 duplicates, the remaining number stood at 550. Upon

reviewing the titles and abstracts of these studies, 90 papers were deemed irrelevant. Among the 460 reports searched for recovery, 160 were not located. Subsequently, 300 papers underwent a comprehensive full-text evaluation, leading to the rejection of 210 due to inaccurate research outcomes and 81 because the wrong kind of population was used. In the end, this comprehensive analysis encompassed 9 suitable research papers. (Figure 1) offers a comprehensive depiction of the study selection process. Features of the studies that were included (Table 1): Members' Socio-demographic Features

(Table 1) summarizes the demographic information of participants from nine distinct studies and includes a substantial number of cases and health controls from references. [14-22]. These studies were carried out in various locations around the world, including two in China [15,21], two in Sweden [17,22], one in Taiwan [14], one in France [16], one in Canada [18], one in the USA [19], and one in South Korea [20]. The research methodology of the study reviewed in reference [14] involved the use of observational study, while the studies cited in references [15, 22] utilized retrospective study. Moreover, the studies referenced in [17, 19, 20] employed cohort study, the study mentioned in reference [18] utilized Population-Based Study, and the study cited in reference [21] utilized Mendelian randomization study. The participants' age range did not pose any distractions, and all patients were adults. Specifically, the ages recorded were 34-50 years old, 28±13 years, 16-57 years, and 55+18 years, according to Chuang, Ya-Wen et al. 2015 [14], Zhang, Li, et al. 2015 [15], Thomas, Guillemette, et al. 2017 [16], and Tornvall, Per et al. 2021 [22], respectively. Arkema, Elizabeth V., et al. 2017 [17] reported a mean age of 49, while Barnado, April, et al. 2018 [19] reported the mean age of patients as 40. According to Avina-Zubieta et al. 2017 [18], the age was over 18. Lim, S. Y., et al. 2018 [20] and Gao, Ning et al. 2022 [21] did not record the age in their studies.

In all the studies analyzed in this research, the percentage of women was consistently higher than that of men. For example, Chuang, Ya-Wen et al. 2015 [14] found 8770 female and 1374 male patient cases, and 8763 female and 1381 male health control cases. Zhang, Li, et al. 2015 [15] reported 22 out of 25 SLE cases and 88 out of 100 health control cases were female. Thomas, Guillemette, et al. 2017 [16] noted 26 out of 29 patients were female. Arkema, Elizabeth V., et al. 2017 [17] and Tornvall, Per et al. 2021 [22] found that around 83-85% of the cases were female. Avina-Zubieta et al. 2017 [18] included 4,199 female out of 4863 individuals with incident SLE. Barnado, April, et al. 2018 [19] and Lim, S. Y., et al. 2018 [20] observed around 90-91% of female cases. Gao, Ning et al. 2022 [21] did not provide patient counts in their study. These findings support the conclusions of Arkema, Elizabeth V., et al. 2017 [17], which suggest that the gender has an impact and women are at a higher risk. (Table 2): Type of CVD associated with lupus, results and major outcomes of the included studies. The studies examined the various types of cardiovascular diseases (CVD) associated with systemic lupus erythematosus (SLE). Chuang, Ya-Wen et al. 2015 [14] found a connection between SLE and peripheral arterial occlusive disease. Zhang, Li, et al. 2015 [15] and Thomas, Guillemette, et al. 2017 [16] highlighted the correlation between myocarditis and SLE. Arkema, Elizabeth V., et al. 2017 [17], Avina-Zubieta et al. 2017 [18], and Lim, S. Y., et al. 2018 [20] reported on the relationship between stroke and SLE. Avina-Zubieta et al. 2017 [18], Barnado, April, et al. 2018 [19], Lim, S. Y., et al. 2018 [20], and Tornvall, Per et al. 2021 [22] explained the association between myocardial infarction and SLE. Gao, Ning et al. 2022 [21] provided a general overview of cardiovascular diseases and SLE in their study. Several research studies have yielded noteworthy findings. As per Chuang, Ya-Wen et al. 2015 [14], individuals diagnosed with SLE exhibit a significantly higher occurrence and an independently elevated risk of PAOD in comparison to the general population. Furthermore, The research carried out by Zhang, Li, et al. 2015 [15] together with Guillemette, Thomas, et al. 2017 [16] revealed that heightened SLE disease activity could potentially forecast the onset of lupus myocarditis during the early stages of SLE, with myocarditis manifesting as the initial indication of SLE in nearly 60% of the patients. Additionally, Arkema, Elizabeth V., et al. 2017 [17] affirmed that those with SLE face twice the risk of stroke when compared to the general population. Avina-Zubieta et al. 2017 [18] and Tornvall, Per et al. 2021 [22] further emphasized the concern regarding myocardial infarction. In their study, Barnado, April, et al. 2018 [19] conveyed that African Americans, specifically those with SLE, were three times more prone to CVD in contrast to Caucasians with SLE. Moreover, Lim, S. Y., et al. 2018 [20], and SLE. Gao, Ning et al. 2022 [21], expounded on the fact that systemic lupus erythematosus represents an autonomous risk factor for heart disease, underscoring the urgent necessity for cardiac evaluation and management in those suffering from erythematous systemic lupus.

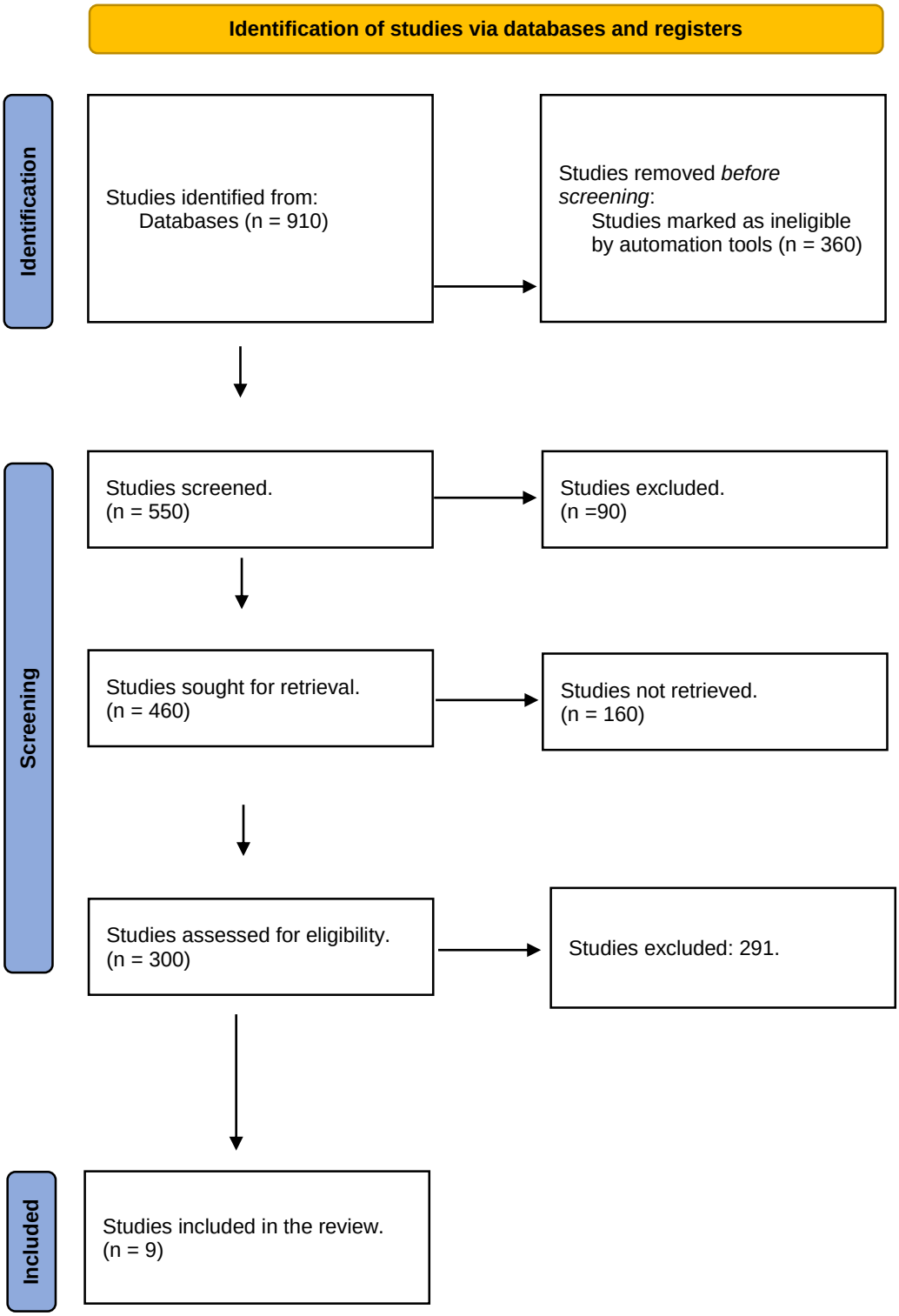

Figure 1: PRISMA flowchart summarizes the study selection process.

Table 1: Socio-demographic characteristics of the included participants.

Study	Country	Study design	No of Patients	Gender	Age	Duration of study
Chuang, Ya-Wen et al. 2015 [14]	Taiwan	OBSERVATIONAL STUDY	10,144 SLE patients	8770 female 1374 male	34- 50 years old	NA
			10,144 control subjects	8763 female 1381 male		
Zhang, Li, et al. 2015 [15]	China	retrospective case–control study	25 patients	22 female	28±13 years	NA
			100 control	88 female		
Thomas, Guillemette, et al. 2017 [16]	French	retrospective, multicenter study	29 patients	26 female 3 male	16-57 years	January 2000 to May 2014
Arkema, Elizabeth V., et al. 2017 [17]	Sweden	cohort study	3,390 patients	85% female	Mean age 49	May 2015 and April 2016
			16,730 comparators			
Avina-Zubieta et al. 2017 [18]	Canada	Population-Based Study	4,863 SLA	4,199 female	≥18 years	January 1996 and December 2010
			49,316 non-SLA	42,346 female		
Barnado, April, et al.2018 [19]	USA	Cohort study	1097 patients	90% female	Mean age: 40	NA

			5735 control		NA	
Lim, S. Y., et al. 2018 [20]	South Korea	Cohort study	18 575 patients 92 875 control	91% female	NA	2008–2014
Gao, Ning et al. 2022 [21]	china	Mendelian randomization study	NA	NA	NA	NA
Tornvall, Per et al. 2021 [22]	Sweden	retrospective cohort study	4 192 patient 41 892 control	83.2 % female	55 + 18 years	NA

Table 2: type of CVD associated with lupus, results and major outcomes of the included participants.

Study	**CVD associated with lupus**	**Results**	**Major outcomes**
Chuang, Ya-Wen et al. 2015 [14]	peripheral arterial occlusive disease (PAOD)	Patients with SLE exhibit a higher incidence and an independently higher risk of PAOD compared with the general population.	The PAOD risk was elevated in young patients with SLE. the risk of PAOD in patients with SLE was highest in the first year after SLE diagnosis and then it declined.
Zhang, Li, et al. 2015 [15]	myocarditis	High SLE disease activity might potentially predict the occurrence of lupus myocarditis at the early stage of SLE.	Echocardiographic findings could confirm the diagnosis of lupus myocarditis. Early aggressive immunosuppressive therapy could improve the cardiac outcome of lupus myocarditis

Thomas, Guillemette, et al. 2017 [16]	myocarditis	Myocarditis was the first manifestation of SLE in almost 60% of the patients.	Among the patients already diagnosed with SLE, the median time between SLE onset and myocarditis was 8.5 years
Arkema, Elizabeth V., et al. 2017 [17]	stroke	Individuals with SLE have twice the risk of ischaemic stroke compared to the general population	the relative risk differed by age and sex, with a higher relative risk in females and individuals younger than 50 years of age
Avina-Zubieta et al. 2017 [18]	Myocardial infarction (MI), stroke	SLE was associated with an increased incidence of MI, stroke, or the combined outcome of MI or stroke (CVD)	The risks of CVD elevated with 6- fold during the first year after SLE diagnosis and remained significantly high even after 5 years.
Barnado, April, et al.2018 [19]	MI	African Americans with SLE were three times more likely to have CVD compared to Caucasians with SLE	African Americans with SLE have an increased comorbidity burden compared to Caucasians
Lim, S. Y., et al. 2018 [20]	MI and stroke	Systemic lupus erythematosus was an independent risk factor for cardiovascular disease, thus cardiac assessment and management are critical in systemic lupus erythematosus patients.	NA
Gao, Ning et al. 2022 [21]	NA	genetic liability to SLE is associated with an increased risk of HF, IS, VTE, and a lower T2DM risk	NA

Tornvall, Per et al. 2021 [22]	Acute myocardial infarction	Systemic lupus erythematosus was associated with an increased incidence of AMI	the incidence of AMI is increased in a European population of patients with SLE

Important results were obtained from the many study studies. Chuang, Ya-Wen et al. 2015 [14] observed that young individuals with SLE encountered a heightened risk of PAOD. This risk was most pronounced within the initial year following the diagnosis of SLE, after which it gradually declined. Zhang, Li, et al. 2015 [15] ascertained that echocardiographic findings could validate the identification of lupus myocarditis, and early and aggressive immunosuppressive treatment could ameliorate its cardiac consequences. Thomas, Guillemette, et al. 2017 [16] unveiled that among patients previously diagnosed with SLE, the median duration between the onset of SLE and the occurrence of myocarditis was 8.5 years. Arkema, Elizabeth V., et al. 2017 [17] confirmed that the relative risk exhibited variations based on age and gender, with a greater relative risk in females and individuals under 50 years of age. Avina-Zubieta et al. 2017 [18] documented a six-fold escalation in the risk of CVD within the initial year following an SLE diagnosis, and this elevated risk persisted significantly even after 5 years. Barnado, April, et al. 2018 [19] validated that African Americans with SLE bear a heavier burden of comorbidities compared to Caucasians. Tornvall, Per et al. 2021 [22] reported an increased incidence of AMI within a European population of SLE patients.

Discussion

One autoimmune illness that may affect every organ in the body is systemic lupus erythematous.This disease is complex. The SLE mortality curve experiences a second peak due to cardiovascular problems. Cardiovascular mortality is still high despite modest improvement in SLE prognosis and overall mortality. A growing body of research indicates that SLE has an independent impact on CVD. In line with our findings, After 20 observational studies were meta-analyzed, it was shown that individuals with SLE were more likely to experience peripheral vascular disease, heart failure, and stroke [23]. Compared to controls, patients with SLE had a two–three times higher risk of stroke, according to another meta-analysis [24]. Patients with SLE had a considerably greater frequency of hyperlipidemia and T2DM, according to a case-control study [25]. In a similar vein, SLE patients had an increased risk of CVD, according to the observational research for Avina-Zubieta et al. in our review [18].

Nonetheless, inconsistent findings have been found in certain research. A prospective research that compared SLE patients to controls found no evidence of a statistically significant rise in the chance of stroke [26]. There is no discernible change in cardiovascular characteristics between individuals with SLE and healthy controls at similar risk of CVD, according to observational studies [27]. A number of pathogenic pathways that operate simultaneously cause cardiovascular involvement in SLE, which results in several cardiac events occurring at a younger age than in the overall population [4]. In a 2003 research published by Asanuma et al., The sample group consisted of 134 individuals that were the same age, sex, and race: There were 65 SLE patients (mean age of 40.3 years) and 69 control subjects (mean age of 42.7 years). Who had never before experienced coronary artery disease. The Agatston score was utilized to evaluate the extent of coronary artery calcification subsequent to the investigation's use of electron beam computed tomography to identify its existence. The study's findings demonstrated that compared to controls (6 of 69 participants), Out of 65 individuals, 20 have SLE had a higher prevalence of coronary artery calcification. The study discovered a higher prevalence of coronary artery atherosclerosis and manifests sooner in SLE patients [28]. In individuals with SLE, Common cardiac sequelae include pericarditis, myocarditis, valve problems, and conducting system problems. [29]. Based on some postmortem examination studies, the pericardium is one of the most often affected areas in SLE, with a frequency of around 62%. Smiti et al.(2009) completed a retrospective research [30] involving 97

SLE patients from Tunisia who underwent echocardiography in order to screen for pericarditis and who received their diagnosis between 1987 and 2005.Thirty eight individuals had pericarditis on average (36.4 years old), whereas the 59 patients without pericarditis had an average age of 28.8 years old, according to the research. The research findings indicate that whereas anticardiolipin antibodies are considerably positive in cases of cardiac valve involvement but seem to be less common in pericarditis. The most frequent cardiac-related occurrence in SLE is pericarditis, which is related to pleuritis. Despite being uncommon, lupus myocarditis poses a significant risk to the heart because of its impact on the functioning and conducting mechanism of the heart. Echocardiography is used to diagnose myocarditis in patients with SLE, cardiac magnetic resonance imaging (MRI) or endomyocardial biopsy when there is a high degree of suspicion and progresses according to disease activity and ethnicity [31]. According to the multicenter study which are included in the review, Thomas et al. (2017) reported that out of the 29 SLE participants chosen for the research, 17 had myocarditis as their initial symptom and 19 possessed an LVEF (left ventricular ejection percent), of less than 45%. These findings improved after the patients started immunosuppressive treatment, indicating that the lack of the appropriate medications is the cause of myocarditis in SLE [16]. Many echocardiographic abnormalities were identified in 599 SLE individuals without clinically significant diagnosed cardiac illness. These abnormalities included pericardial thickening (6.8%), hypertension of the pulmonary arteries (8.5%), effusion around the heart (13.6%), systolic function reduction (3.4%), valvular lesions (47.5%), and left ventricular hypokinesia (1.7%). Furthermore, 44.1% of the individuals showed no abnormalities [32]. Official guidelines for preventing CVD in the context of SLE are currently lacking, despite the elevated risk of CVD in SLE patients. Various obstacles, such as recruitment and retention, have been identified in efforts to reduce risk factors in SLE. Drawing from evidence in other high-risk groups (e.g., diabetics), recommendations are made for potentially modifiable risk factors. Additionally, a lack of understanding among patients and doctors about the issue leads to underestimation of its importance. Patients also cite medication concerns as a major barrier to their participation in studies. Adding new drugs for primary or secondary prevention of CVD in individuals with existing medication issues will be challenging [33]. It is worth noting that there has been some suggestion that statins themselves may modulate autoimmune diseases. In a pilot trial involving 14 SLE patients, it was found that rosuvastatin, when taken for three months, significantly reduced lipid levels but had no effect on erythrocyte sedimentation rate, complement, double-stranded DNA antibodies, or inflammatory cytokines [34,35]. Ruiz-Irastorza et al.(year) demonstrated in a meta-analysis that hydroxychloroquine is beneficial as a first-line treatment for thrombotic episodes in individuals with cardiovascular risk factors (CVRFs) and sickle cell disease (SLE). The impact of anti-malarial on CV risk in individuals with SLE has been investigated in a few studies. Individual investigations have found a correlation between anti-malarial and a lower incidence of thrombotic events [36]. According to a Penn et al. (year) research, antimalarial medicine was taken by half of the 149 SLE patients who were assessed and did not have diabetes. Insulin resistance was reduced in the antimalarial group (HOMA-IR: 194 vs. 179) as well as the levels of glucose during fasting (87.1 mg/dL vs. 91.5 mg/dL) compared to the control cohort. Moreover, the LDL levels of the treated group were lower than those of the untreated group. (102 mg/dL and 118 mg/dL, in that order). With a SIGN 1+ B grade of recommendation and a degree of scientific evidence indicating its efficacy, hydroxychloroquine is recommended for the main prevention of thrombotic events in patients with SLE [37].

Conclusion

Evidence for a possible causal link between SLE and a higher risk of certain CVDs was shown by this investigation. The risk of cardiovascular events in adult sufferers of SLE, especially women, is significantly higher compared to the broader population or healthy guidelines for CVD. This increased risk is thought to be due to the chronic inflammation and immune system dysfunction associated with SLE, as well as traditional cardiovascular risk factors. Understanding the various mechanisms underlying this increased Risk will help treatment and preventative plans, as well as patient and physician decision-making that is well-informed.

Conflict of Interest

None

Funding

None

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