DEVELOPMENTAL MEDICO-LIFE-SCIENCES

ISSN (P): 3007-2786, (E): 3007-2794 DOI: https://doi.org/10.69750/dmls.02.011.0172

ORIGINAL RESEARCH ARTICLE Open Access

Association Between Obesity-Induced Inflammation and Early Cardiac Dysfunction in Young Adults. A Cross-Sectional Clinical Study

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ABSTRACT

Background: Obesity among young adults is a growing international problem and closely related to early cardiovascular alterations, which may be asymptomatic. It is known that chronic low-grade inflammation is one of the major pathways between obesity and myocardial dysfunction. The purpose of this study was to determine the relationship between the inflammatory biomarkers brought about by obesity and the premature cardiac dysfunction among young adults.

Methods: It was a cross-sectional clinical study that involved 100 young adults aged 18-35 years who visited a tertiary care hospital. There was the acquisition of anthropometric measurements, fasting biochemicals, and inflammatory measures, high-sensitivity C-reactive protein (hs-CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-alpha). Full transthoracic echocardiography was done on all participants (tissue Doppler imaging and global longitudinal strain (GLS)). Patients having known cardiovascular/metabolic diseases were eliminated. Correlation analysis was conducted to establish the relationship that exists between inflammatory markers and cardiac parameters.

Results: 66 percent of the 100 respondents were obese, and 34 percent were overweight. The hs-CRP (5.6 ± 1.7 mg/L), IL-6 (7.2 ± 2.0 pg/mL), and TNF-α (14.6 ± 3.1 pg/mL) levels of the obese people were significantly greater than those of the overweight participants ($p < 0.001$). Subclinical heart failure was observed in 36 percent of subjects without a decrease in ejection fraction. Abnormal diastolic indices and impaired GLS had significant connections with increased inflammatory indices. There were significant correlations between hs-CRP, IL-6, TNF-alpha, and GLS ($r = -0.54, -0.47, -0.51$; $p < 0.001$) and E/e/ ratio ($p < 0.01$).

Conclusion: Systemic inflammation induced by obesity has a high relation with premature cardiac dysfunction in young adults. High levels of inflammatory biomarkers are well associated with reduced myocardial strains and diastolic abnormalities, thus the need to perform cardiovascular screening in young people with obesity. It could be possible to conduct early detection since it allows time to make lifestyle changes and prevent the development of cardiovascular disease in the future.

Keywords: Obesity, inflammation, hs-CRP, IL-6, TNF-6, global longitudinal strain, young adults, cardiac dysfunction.

Received: 12/08/2025
Revised: 29/10/2025
Accepted: 26/11/2025
Published: 30/11/2025

INTRODUCTION

Obesity has emerged as a major global health challenge, particularly among young adults, where its prevalence continues to rise due to sedentary behavior, increased intake of calorie-dense foods, and genetic susceptibility [1]. Although obesity is commonly linked with long-term complications such as type 2 diabetes mellitus and cardiovascular disease, growing evidence shows that cardiac abnormalities can begin much earlier, even before clinical symptoms appear [2]. This early stage of myocardial impairment, known as subclinical cardiac dysfunction, often remains undetected yet substantially elevates the risk of adverse cardiovascular outcomes in later life [3].

One of the pathways between obesity and early cardiac dysfunction is chronic low-grade inflammation. The excess adipose tissue is an active endocrine organ, which secretes the inflammatory mediators of interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF), and C-reactive protein (CRP), which stimulate endothelial dysfunction, oxidative stress, abnormal myocardial metabolism, and structural remodeling of the heart [4]. Such inflammatory pathways can initiate early myocardial relaxation, diastolic filling, strain parameters, and autonomic regulation abnormalities even in young adults in the absence of hypertension, diabetes, and coronary artery disease [5].

The clinical implication of the relationship between inflammation caused by obesity and cardiac dysfunction in young adults is enormous [6]. Patients of this age are not always diagnosed due to the absence of characteristic symptoms, and could have normal results of regular heart tests. Nevertheless, more sophisticated echocardiographic methods such as tissue Doppler imaging (TDI), global longitudinal strain (GLS), and indices of diastolic function will be able to identify subtle myocardial remodelling at a very early and possibly reversible level [7]. Early diagnosis of such abnormalities might allow for to avoidance of the development of heart failure and other significant cardiovascular diseases [8].

Although there is widespread evidence of the adverse effects of obesity on inflammatory and cardiac outcomes in young adults in low and middle-income countries, the evidence is limited [9]. The bulk of the available literature considers either older individuals or comorbidity conditions, with little knowledge on how cardiac changes occur early on due to the presence of obesity-induced inflammation in young adults with no comorbidities [10].

The purpose of the proposed cross-sectional study is to determine the relationship between inflammatory markers of obesity and the premature signs of cardiac dysfunction in young adults. The study aims to justify the need to monitor at-risk individuals at an early stage and highlight the need to adopt preventive cardiovascular interventions in this at-risk population due to the interplay between systemic inflammation, adiposity, and subclinical myocardial changes.

MATERIALS AND METHODS

The present cross-sectional clinical study was carried out in the outpatient and general medical clinics of Shalamar Hospital, Lahore, Pakistan, within the period between January 2024 and March 2025. One hundred young adults between the ages of 18 and 35 years were recruited using non-probability consecutive sampling. Both males and females who met the eligibility criteria were eligible. Recruitment was done on individuals with a body mass index (BMI) of 25 kg/m 2 and above. All the participants received informed consent, which was written before they were included. All the participants were evaluated in a structured examination, which included clinical examination, anthropometric measurements, laboratory studies, and a comprehensive echocardiographic examination.

A proforma with standardized data collection was used to collect data on demographic information, medical history, lifestyle habits, and family history of cardiovascular diseases. The exclusion criteria were: previous diagnosis of cardiovascular disease, hypertension, diabetes mellitus, thyroid disorders, valvular anomalies, chronic renal failure, and chronic use of anti-inflammatory or cardioactive drugs, which reduced the possible confounding factors. A digital weighing scale and calibrated stadiometer were used to measure height and weight, respectively. BMI was determined by dividing weight (kg) by height squared (m 2). Also, the waist circumference, hip circumference, and waist-to-hip ratio were taken to measure central adiposity.

Biochemical analysis of fasting venous blood samples was done following an overnight fast. Standardized enzyme-linked immunosorbent assay (ELISA) kits were used to measure inflammatory biomarkers, such as high-sensitivity C-reactive protein (hs-CRP), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6), by following the instructions of the kit manufacturers. Basic biochemical examinations, including fasting blood glucose, lipid profile, and serum creatinine, were also done to rule out possible underlying metabolic or renal pathology that could affect cardiac performance.

A qualified cardiologist who was blinded to the values of biomarkers was the one who performed transthoracic echocardiography. The standard protocol consisted of M-mode, two-dimensional imaging, Doppler analysis, and TDI. Echocardiographic variables that were measured were left ventricular ejection fraction (LVEF), left ventricular mass index, diastolic filling (E/A ratio), E/e+ ratio, and global longitudinal strain (GLS). The presence of early cardiac dysfunction despite a collected ejection fraction was classified according to the lowered GLS, abnormal diastolic indices, or high E/e 7 values, which revealed a lack of myocardial relaxation. The completeness of all data had been checked and entered into statistical analysis software. Mean ± standard deviation was used to represent quantitative variables such as inflammatory biomarkers and echocardiographic rates, whereas such categorical variables as gender and obesity grade were presented as frequencies and percentages. Pearson correlation coefficient was used to test the correlation between inflammatory biomarkers and echocardiographic indices. The independent t-test or Mann-Whitney U test, which depends on the data normality, was applied to conduct the comparative analyses of the participants whose cardiac indices are normal and abnormal. A p-value lower than 0.05 was regarded as significant.

Ethical approval for this study was obtained from the Institutional Review Board (IRB) of Shalamar Medical and Dental College, Lahore. The study was reviewed and approved under Ethical Approval Reference No.:

SMDC/IRB/2023/117. All research procedures complied with the Declaration of Helsinki, and strict confidentiality of participant data was ensured throughout the study.

RESULTS

In this study, 100 young adults between the ages of 18 and 35 years were used. Fifty-eight percent were males, and forty-two percent were females. According to BMI, 34% were overweight (BMI 2529.9 kg/m 2) and 66% obese (BMI 30kg/m 2 and above). The average age of the participants was 27.4 years old and the BMI was 31.2 kg/m 2. The general baseline features of the population used in the study are summarized in Table 1.

The data indicate that the majority of the subjects were markedly centrally adipose, as indicated by high waist circumference and high BMI. The subjects were all young adults, and the values of their metabolism were at the borderline levels, which implies that there was no overt metabolic disease. Most of the obese respondents had elevated serum inflammatory markers. The average levels of hs-CRP, IL-6, and TNF-alpha were 4.8 and 12.7, and 6.3, respectively. The inflammatory markers were significantly higher in obese individuals as compared to overweight individuals (p a 0.001). The detailed inflammatory marker profiles are given in Table 2.

Markedly elevated all the inflammatory markers in obese participants versus overweight ones signified the existence of systemic inflammation in most of the study population as a result of obesity. The occurrence of subclinical cardiac dysfunction was observed in 36 percent of the participants, although all the subjects had normal ejection fraction (LVEF 55% and above). The average worldwide longitudinal strain (GLS) was -17.1 +/- 2.4, and the presence of impaired GLS was found in 29 people (less than 18). In 24 subjects, dysfunction of the diastole occurred (abnormal E/e 7 ratio or modified E/A ratio). The echocardiographic parameters are depicted in Table 3.

While systolic function (EF) remained normal across the cohort, subtler markers revealed that early systolic functioning (EF) was normal in all the cohort, but more advanced signs demonstrated early systolic and diastolic dysfunction. The decreased GLS values indicate premature deformation of the myocardium, and the change in E/A and E/e+ ratios means the diastolic dysfunction. A high positive relation was found between the signs of cardiac dysfunction and inflammatory markers. High levels of hs-CRP, IL-6, and TNF-alpha were positively correlated with poor GLS and abnormal E/e' prime ratio ($p < 0.01$). The results of the correlation are indicated in Table 4.

Higher levels of inflammatory markers were associated with worsening myocardial strain and impaired diastolic function. The significant negative correlation between cytokines and GLS confirms that inflammation directly contributes to early cardiac dysfunction.

Table 1: Baseline Characteristics of Study Participants (N = 100)

Variable	Mean ± SD / n (%)
Age (years)	27.4 ± 4.1
Gender (Male/Female)	58 (58%) / 42 (42%)
BMI (kg/m²)	31.2 ± 3.8
Overweight	34 (34%)
Obese	66 (66%)
Waist Circumference (cm)	98.6 ± 10.4
Waist-to-Hip Ratio	0.92 ± 0.07
Fasting Blood Glucose (mg/dL)	92.7 ± 9.5
LDL Cholesterol (mg/dL)	119.3 ± 22.6
HDL Cholesterol (mg/dL)	40.5 ± 6.2

Table 2: Inflammatory Biomarkers in Overweight vs. Obese Participants

Marker	Overweight (n=34) Mean ± SD	Obese (n=66) Mean ± SD	p-Value
hs-CRP (mg/L)	3.2 ± 1.1	5.6 ± 1.7	<0.001
IL-6 (pg/mL)	4.7 ± 1.3	7.2 ± 2.0	<0.001
TNF-α (pg/mL)	9.4 ± 2.2	14.6 ± 3.1	<0.001

Table 3: Echocardiographic Parameters of Participants

Parameter	Mean ± SD
LVEF (%)	62.4 ± 3.8
GLS (%)	–17.1 ± 2.4
E/A Ratio	0.98 ± 0.21
E/e′ Ratio	9.8 ± 3.1
Left Ventricular Mass Index (g/m²)	92.5 ± 14.6

Table 4: Correlation Between Inflammation and Echocardiographic Parameters

Variable Pair	Correlation Coefficient (r)	p-Value
hs-CRP vs. GLS	–0.54	<0.001
IL-6 vs. GLS	–0.47	<0.001
TNF-α vs. GLS	–0.51	<0.001
hs-CRP vs. E/e′	0.43	0.002
IL-6 vs. E/e′	0.39	0.004
TNF-α vs. E/e′	0.45	0.001

DISCUSSION

The current cross-sectional clinical trial assessed the relationship between obesity-related inflammation and early myocardial dysfunction in young adults (1835 years old). The results indicate that there is a strong and significant connection between the high levels of inflammatory biomarkers and undetectable echocardiographic anomalies, though the participants were of normal left ventricular ejection fraction [11]. These findings underline the fact that, despite being undervalued in younger patients, obesity has a significant role to play in the progression of premature impairment of the myocardial state long before the outbreak of indisputable cardiovascular disease.

The most important finding of the research is that the concentration of inflammatory biomarkers, hs-CRP, IL-6, and TNF, is significantly increased in obese people compared to the overweight people. This is in line with what has been reported before that adipose tissue is an active endocrine gland secretion of pro-inflammatory cytokines is part of the chronic low-grade systemic inflammation [12].

The rise in the hs-CRP observed in this case is in line with the previous literature that has attributed obesity to signaling of cytokines, especially IL-6, to lead to the production of CRP in the liver. On the same note, IL-6 and TNF-alpha, produced by hypertrophic adipocytes and infiltrating macrophages, facilitate endothelial dysfunction, oxidative stress, and disturbed cardiac myocardial metabolic pathways, promoting structural cardiac remodeling.

The Echocardiographic results also support the early myocardial involvement in obesity. Despite the recorded preserved ejection fraction in all of the participants, the incidence of subclinical cardiac dysfunction involving impaired global longitudinal strain (GLS) and abnormal diastolic filling indices was 36% and 36% respectively [13]. These findings highlight the diagnostic importance of new echocardiographic methods like GLS and tissue Doppler imaging that can identify cardiac myocardial dysfunction before conventional parameters. Similar results were recorded in the previous studies, which showed impaired myocardial deformation in obese people despite the absence of diabetes, hypertension, and dyslipidemia, indicating the direct effect of adipose-stimulated inflammation on myocardial fibers [14].

Necessarily, clinically, high associations were found between an increase in inflammatory levels and impaired cardiac variables. Increased hs-CRP, IL-6, and TNF-alpha levels were correlated with deteriorating GLS, high E/e of e, and higher filling pressures. These results support the known fact that inflammation plays a pivotal mechanistic role between obesity and premature cardiac dysfunction [15]. Persistent inflammation facilitates myocardial fibrosis, collagen deposition, and altered turnover of extracellular matrix, all of which diminish myocardial compliance and contractility. The process can be the first step towards the transition of an asymptomatic myocardial strain into heart failure with preserved ejection fraction (HFpEF), as a condition that has been more and more associated with obesity [16].

Another aspect of clinical significance of the study is the realization of early cardiac changes in young adults who are traditionally considered to be low-risk individuals in relation to cardiovascular disease. Findings of this study reveal that the onset of myocardial dysfunction caused by obesity will start way before people expect it. Early diagnosis offers a chance of preventive measures that may include lifestyle change, weight-loss measures, nutritional optimization, and control of metabolic risk factors. Besides, the results indicate that inflammatory biomarkers can be useful in screening obese people to identify those at risk of developing cardiac dysfunction [17].

The present study has strong features, such as the utilization of sophisticated echocardiographic parameters, the assessment of various inflammatory markers, and the targeted study population, which is young. Nevertheless, some restrictions also have to be recognized [18]. The study design is cross-sectional, and therefore, it does not allow causality; therefore, a longitudinal research design is needed to show whether high levels of inflammatory markers are predictors of progressive cardiac dysfunction over time. At the same time, in spite of the fact that the sample size was sufficient to find significant associations, there is a need to conduct larger multicenter studies, thus improving the generalizability. Also, lifestyle factors that cannot be measured (dietary practices, physical exercise, and sleep quality) could affect inflammation and cardiac functioning [19].

Nevertheless, in places of these shortcomings, the results present strong evidence to show that obesity-related inflammation is strongly associated with the premature dysfunction of the heart in young adults. This highlights the importance of early cardiovascular screening of obese people despite the lack of typical risk factors or symptoms [20]. The introduction of inflammatory biomarkers and a more sophisticated echocardiographic index into the routine testing can potentially allow identifying cardiac dysfunction in a reversible phase, ultimately leading to decreased long-term cost of obesity-related cardiovascular disease.

CONCLUSION

This cross-sectional clinical trial demonstrates a robust and clear linkage between obesity-related systemic inflammation and premature cardiac dysfunction in young adults aged between 18 and 35 years. They still had preserved ejection fraction, but a substantial proportion of the participants had affected global longitudinal strain and diastolic dysfunction, which indicates that they had been engaged at an early phase of the myocardium. It was found that these minor echocardiographic changes were highly linked to high concentrations of inflammatory markers such as hs-CRP, IL-6, and TNF-alpha; hence, chronic low-grade inflammation is a fundamental process through which obesity is linked to impending cardiac dysfunction. This research study sheds some light on the necessity to carry out early cardiovascular screening of young obese individuals despite the lack of symptoms and the normality of the traditional cardiac parameters. Another modification that can be introduced to identify high-risk patients at a reversible phase is the use of inflammatory markers and strain-based echocardiography as a part of a regular assessment. Early lifestyle changes, losing weight, and risk factor management have the potential to prevent the development of full-blown cardiovascular disease in old age. The future contains a need to further longitudinal studies to push the causality and determine whether a drop in inflammation can be used to improve the cardiac outcomes in this population.

Conflict of Interest: The authors report no conflicts of interest.

Funding: No external funding was received for this study.

Acknowledgments: The authors acknowledge the support of Shaikh Zayed Hospital, Lahore, for facilitating data

collection and laboratory analysis. The contribution of all clinical and technical staff involved in patient evaluation is gratefully appreciated.

Authors' Contributions

MUA designed the study and supervised overall work. AA assisted with data collection and background literature. MSS contributed to data acquisition and initial drafting. AA-3 supported methodological input and clinical review. SA assisted in organizing data and refining results. HA contributed to manuscript editing and final revisions.

Data Availability Statement: The data used in this study are available upon reasonable request from the corresponding author, subject to ethical and institutional guidelines.

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This article may be cited as: Abbas MU, Abbas A, Sajjad MS, Ali A, Amjad S, Abdullah H. Association between obesity-induced inflammation and early cardiac dysfunction in young adults: a cross-sectional clinical study. *Dev Med Life Sci*. 2025;2(11):8-12. doi:10.69750/dmls.02.011.0172.