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Original Article

Relationship between physical activity and cardiovascular disease in adults

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Abstract: Objective: Cardiovascular disease (CVD) remains a leading cause of morbidity and mortality worldwide, with physical inactivity identified as a critical modifiable risk factor - the association between physical activity levels and cardiometabolic markers warrants further exploration to inform preventive strategies. We investigate the association between physical activity levels and cardiovascular disease in the adult population. Methods: This retrospective study was conducted at a Tertiary Care Hospital. A total of 554 participants were recruited, including 277 cases diagnosed with CVD and 277 age- and gender-matched controls with no CVD. Physical activity levels were categorized as poor, intermediate, or recommended, based on the weekly activity level. Demographic and clinical data were collected, including Body Mass Index (BMI), blood pressure, lipid profiles, and smoking status. Results: Physical inactivity was significantly higher among patients with CVD, with only 17.9% meeting recommended activity levels compared to 82.1% of controls ($P<0.001$). CVD patients also exhibited significantly higher BMIs, systolic blood pressures, LDL cholesterol levels, and triglyceride levels, as well as lower HDL levels. Conclusion: Physical inactivity is strongly associated with cardiovascular disease and adverse cardiometabolic profiles. Promoting regular physical activity is crucial for reducing cardiovascular risk.

Keywords: Cardiovascular disease, physical activity, case-control study, cardiometabolic markers

Introduction

Cardiovascular disease (CVD) is a leading cause of death and disability worldwide, accounting for a significant burden on healthcare systems and economies [1]. Regular physical activity is a well-established preventive and therapeutic tool for CVD. It improves myocardial efficiency, enhances endothelial function, optimizes lipid profiles, and regulates blood pressure and glucose metabolism - each playing a vital role in mitigating cardiovascular risk [2].

Guidelines from the World Health Organization (WHO) and the American Heart Association recommend that adults engage in at least 150 minutes of moderate-intensity or 75 minutes of vigorous-intensity aerobic activity per week [3]. However, adherence to these recommendations remains low in both developed and developing countries, primarily due to sedentary lifestyles, lack of awareness, psychosocial barriers, and socioeconomic constraints [4].

Physical activity exerts cardioprotective effects not only by modifying direct risk factors - such as obesity, hypertension, dyslipidemia, and type 2 diabetes mellitus - but also by reducing systemic inflammation and enhancing autonomic regulation [5]. Physical activity reduces central adiposity, a potent factor that drives metabolic syndrome and atherogenic dyslipidemia [6]. Exercise improves insulin resistance, thereby reducing endothelial dysfunction

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caused by hyperglycemia, an initial event in atherosclerosis [7]. A growing body of evidence demonstrates that these physiological benefits follow a dose-response pattern, with greater frequency and intensity of exercise conferring greater protection [8].

Among individuals already diagnosed with CVD, structured exercise programs form a key component of cardiac rehabilitation [9]. Studies have shown that such programs significantly improve functional capacity, reduce the incidence of rehospitalization, and lower cardiovascular mortality [10]. Meta-analyses confirm these outcomes across diverse patient populations, especially post-myocardial infarction or after coronary revascularization procedures.

Several meta-analyses have established that exercise-based rehabilitation, as demonstrated in randomized controlled trials, decreases hospital readmissions and improves quality of life in both post-myocardial infarction and post-coronary artery bypass graft patients [11, 12]. Other physiological benefits include increased myocardial oxygen consumption, reduced systemic inflammation, and improved autonomic balance, as reflected by enhanced heart rate variability [13]. Despite the strong evidence on the role of physical activity in reducing the burden of CVDs, population adherence to regular physical activity remains suboptimal [14]. Overall, a sedentary lifestyle, socioeconomic constraints, and a lack of awareness have contributed to the high prevalence of physical inactivity [15]. Therefore, health professionals should be involved in patient education and promoting behavior modification. Physical activity is a non-pharmacological therapy with minimal cost and significant cardiovascular benefits [16].

Studies such as that by Kubota *et al.* highlight the substantial differences in CVD risk among individuals with poor (47.6%), intermediate (44.0%), and recommended (38.1%) physical activity levels. These statistics emphasize the need for better implementation of preventive strategies and targeted interventions [17].

This study examines the relationship between physical activity levels and cardiovascular disease in adults within a regional population. It aims to provide practical, locally relevant evidence to guide clinical practice and inform public-health strategies for CVD prevention.

Methodology

Study design

Our retrospective case-control study examined the relationship between physical activity levels and cardiovascular disease (CVD) in adults. The study was conducted at a Tertiary Care Hospital (from the 15th of April 2024 to the 15th of April 2025). This study encompassed 600 inpatient beds, multiple specialty departments, and a dedicated cardiology and internal medicine unit. The hospital admits a high number of patients each month, and its healthcare staff comprises physicians, nurses, allied health professionals, and administrative personnel. This setting provided a diverse patient population suitable for this case-control study. The study followed the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) guidelines for observational case-control studies.

Sample size and sampling

The sample size was calculated based on the odds ratio (OR) of 1.25 for poor physical activity levels associated with CVD, as reported in the study by Kubota *et al.* [17]. Since we focused on exploring potential risk factors for CVD, a case-control design was employed to compare individuals with CVD (cases) to those without CVD (controls). A total of 554 participants (277 cases and 277 controls) were recruited for this study. The sample size was determined using the formula for case-control studies:

$$\frac{Z^2_{1-\frac{\alpha}{2}}(P_1(1-P_1)+P_2(1-P_2))+Z^1_{1-\beta}(P_1-P_2)^2}{(P_1-P_2)^2}$$

Where:

- $Z_{1-\alpha/2}$=1.96 (for 95% confidence level),

- $Z_{1-\beta}$=0.84 (for 80% power),

- P_1=0.476 (prevalence of poor physical activity among cases),

- P_2=0.381 (prevalence of poor physical activity among controls).

Using a prevalence of inadequate physical activity among controls of 47.6%, a 95% confidence level, 80% power, and a 1:1 case-to-control ratio, the required sample size was deter-

mined to be 554 participants (277 cases and 277 controls). Cases were diagnosed with CVD, and controls were frequency-age and gender-matched individuals without CVD. The inclusion criteria were strict to guarantee comparability between groups and to minimize confounding variables.

Inclusion criteria

1. Participants aged 30-80 years. 2. Individuals who are willing to give written informed consent. 3. Participants with diagnosed CVD based on medical records or clinical examination. 4. Individuals with no prior history of CVD.

Participants aged 30 to 80 years were selected to focus on the adult population most vulnerable to cardiovascular disease. This range excludes younger adults with low baseline risk and older individuals (>80) who may have complex multimorbidities that could confound the relationship between physical activity and cardiovascular outcomes.

Exclusion criteria

1. Patients at the advanced stage of malignancies. 2. Patients who have chronic inflammatory diseases. 3. Participants with severe disease (e.g., NYHA Class IV heart failure, end-stage renal disease on dialysis), uncontrolled infections, or any condition that significantly limits physical activity or compromises data accuracy. 4. Patients with acute or recent cardiovascular events within the last six months (e.g., myocardial infarction, unstable angina, or recent cardiac surgery), as these conditions could affect physical activity capacity and confound outcome assessments.

Data collection tools

Demographic, clinical, and behavioral characteristics: A comprehensive set of data collection tools to obtain demographic, clinical, and behavioral characteristics. A standardized demographic questionnaire collected information (age, sex, regular quarterly physician visits, smoking or waterpipe use, occupation, and weekly physical activity patterns).

Physical activity: Physical activity was categorized into three levels - Regular, Light, and Inactive - based on the World Health Organization (WHO) Global Recommendations on Physical Activity for Health (2010), consistent with the International Physical Activity Questionnaire (IPAQ) classification framework: 1. Regular: Engaging in 30-minute walking sessions 4 to 7 days per week. 2. Light: Engaging in 30-minute walks 1 to 3 days per week. 3. Inactive: Engaging in less than one session per week.

This classification has been widely used in epidemiological studies to assess physical activity patterns in relation to chronic diseases, including cardiovascular disease. It has demonstrated content validity and reproducibility across populations and has been applied in studies such as: 1. Kubota *et al.* (2017) - Examining lifetime CVD risk by physical activity status. 2. Lee *et al.* (2012) - Estimating the global burden of disease attributable to physical inactivity. 3. Bauman *et al.* (2009) - Evaluating the use of physical activity questionnaires in population surveillance.

Clinical data: Clinical data were obtained from the patient's medical records and laboratory reports. Cardiovascular disease was confirmed through retrospective review of the records by attending physicians. Diagnoses were based on documented findings such as ECG, echocardiography, or coronary angiography reports. The data also included the history of diabetes, hypertension, nephropathy, dyslipidemia, medication use, and cardiovascular disease status. These conditions were defined according to standard clinical guidelines: 1. Diabetes mellitus was defined as fasting blood glucose ≥126 mg/dL, HbA1c ≥6.5%, or documented use of antidiabetic medication. 2. Hypertension was defined as systolic blood pressure ≥140 mmHg and/or diastolic blood pressure ≥90 mmHg or the use of antihypertensive medication. 3. Dyslipidemia was diagnosed if LDL ≥130 mg/dL, HDL <40 mg/dL (men) or <50 mg/dL (women), triglycerides ≥150 mg/dL, or if the patient was on lipid-lowering therapy. 4. Nephropathy was defined by the presence of microalbuminuria (>30 mg/day) or elevated serum creatinine levels (>1.3 mg/dL).

Diagnoses were made based on documented findings by the attending physician or based on laboratory and medication history recorded in the patient's file.

Anthropometric measurements were taken using standardized equipment. Weight was

measured using a digital balance scale with a precision of 0.1 kg, and height was measured using a wall-mounted stadiometer with an accuracy of 0.5 cm. Participants were assessed without shoes and in light clothing or underwear to ensure consistency and reduce measurement error.

Body Mass Index (BMI) was calculated using the formula:

$BMI = weight\ (kg)/height^2\ (m^2)$

Based on WHO guidelines, participants were categorized as: 1. Normal weight: 18.5-24.9 kg/m^2. 2. Overweight: 25.0-29.9 kg/m^2. 3. Obese: ≥30.0 kg/m^2.

Smoking status was self-reported by participants and classified into three groups: 1. Current smoker (smoked within the past year), 2. Ex-smoker (quit smoking ≥1 year ago), and 3. Non-smoker (never smoked).

As a result, this systematic data collection method was pivotal in maintaining the authenticity and reliability of the information gathered, providing a basis for analyzing the interrelationship between physical activity levels, cardiovascular health, and related risk factors.

Data collection process: The Ethics Review Board approved this study, and informed consent was obtained from all participants before data collection. Patients were informed about the study, and written informed consent was obtained before their participation. Data on age, gender, occupation, hospital visits, level of physical activity, and tobacco use were extracted using a standardized demographics questionnaire.

Peripheral neuropathy was assessed using the Michigan Neuropathy Screening Instrument (MNSI), which comprises both a questionnaire and a physical examination component to detect signs and symptoms of neuropathy.

Clinical data, including body mass index (BMI) and cardiovascular disease status, were retrieved from medical records. Participants with diabetes were asked to record their daily dietary intake for seven consecutive days. Weight and height were measured to calculate BMI, while blood samples were taken to perform a lipid profile, including fasting blood sugar (FBS), HbA1c, and creatinine levels. These parameters were calculated by comparing values over the study period.

Statistical analysis: The data were analyzed using IBM SPSS 30.0. Continuous variables, such as BMI, blood pressure, and lipid levels, were presented as means ± standard deviations (SD), while categorical variables, like sex and smoking status, were summarized as frequencies and percentages.

The Kolmogorov-Smirnov test was used to assess the normality of continuous variables. Independent samples t-tests and Mann-Whitney U tests were used to compare continuous variables between the case and control groups, as appropriate. Chi-square tests were applied to examine associations between categorical variables (e.g., smoking status and physical activity level). Univariate logistic regression and the Pearson Chi-square test were used to calculate the odds ratio (OR) for categorical and continuous variables, respectively. To identify independent associations between physical activity and cardiovascular disease, a multivariate logistic regression analysis was performed. This model estimated adjusted odds ratios (ORs) with 95% confidence intervals (CIs) for the association between physical activity levels and CVD status. A p-value <0.05 (two-tailed) was considered statistically significant.

Quality control and reliability: Comprehensive measures were taken to ensure the reliability and validity of the study. The collection was performed by trained health professionals using standardized procedures in a controlled clinical setting to minimize measurement bias. All measurements were taken at baseline during participants' scheduled clinic visits. All personnel were trained to become familiar with appropriate data collection processes and standardized measurement methodologies. Blood pressure, anthropometric data, and other measures were obtained using the same instruments and according to the same protocols for all participants. Data were carefully validated and amended for accuracy during the data compilation process by reviewing source documents. Furthermore, the small sample of respondents in the pre-test questionnaire revealed unrelated formatting issues and ambiguity. These steps helped to ensure the accuracy and dependability of the data acquired during the research.

Table 1. Demographics of the patients (n=554)

Demographics	Case group (n=277)	Control group (n=277)	*P*-Value
Age (years)[a]	54.646±14.82	55.180±15.30	0.7
BMI (Kg/m^2)[a]	29.950±2.82	23.426±2.85	<0.001
Systolic Blood Pressure (mmHg)[a]	160.231±11.91	119.606±5.86	<0.001
Low Density Lipoprotein (mg/dL)[a]	168.631±20.68	103.126±13.98	<0.001
High Density Lipoprotein (mg/dL)[a]	38.426±5.33	59.472±5.50	<0.001
Triglyceride (mg/dL)[a]	221.151±43.52	98.014±28.59	<0.001
Total Cholesterol (mg/dL)[a]	272.472±22.09	223.436±13.63	<0.001

Notes: Mann-Whitney U [a], Mean ± SD.

Table 2. Categorical variables by group (n=554)

Variable		Case Group n=277	Control Group n=277	*P*-Value
Age[a]	Age 30-50	117 (42.2%)	116 (41.9%)	0.93
	Age 51-80	160 (57.8%)	161 (58.1%)	
Gender[a]	Male	165 (59.6%)	123 (44.4%)	<0.001
	Female	112 (40.4%)	154 (55.6%)	
Smoking Status[a]	Non-smoker	52 (18.8%)	123 (44.4%)	<0.001
	Ex-smoker	71 (25.6%)	88 (31.8%)	
	Current Smoker	154 (55.6%)	65 (23.5%)	

Notes: Pearson Chi square [a], n (%).

Limitations: Although the study design's strengths need to be acknowledged, the study is not without limitations. The study was conducted in a single center, which has the potential for broad generalizability. Additionally, the physical activity levels were self-reported by participants, which may have been influenced by recall bias and could result in inaccurate results. Due to the study's retrospective case-control design, causality cannot be established. Thus, the results are limited to associations between the variables of interest.

Ethical considerations: This study was conducted in accordance with strict ethical guidelines to protect the rights and interests of participants. The study protocol was approved by the institutional ethics committee. All participants provided informed consent, being fully briefed about the aims and methods of the study and their involvement in the research. Participants were informed that they could withdraw from the study at any time. All data were collected and stored securely to ensure confidentiality and contained no individual identifiable information. The privacy of participants and data were protected, and the research was conducted in accordance with the Declaration of Helsinki and other applicable ethical guidelines.

Results

The demographics of the case and control groups revealed similar mean ages of 54.646± 14.82 and 55.180±15.30 years, respectively. BMI was significantly higher in the case group (29.950±2.82 kg/m^2) compared to the control group (23.426±2.85 kg/m^2). Systolic blood pressure (160.231±11.91 mmHg vs. 119.606± 5.86 mmHg), low-density lipoprotein (LDL) (168.631±20.68 mg/dL vs. 103.126±13.98 mg/dL), and triglycerides (221.151±43.52 mg/dL vs. 98.014±28.59 mg/dL) were significantly higher in cases vs. controls. Conversely, high-density lipoprotein (HDL) levels were lower in cases (38.426±5.33 mg/dL) compared to controls (59.472±5.50 mg/dL). Among categorical variables, the case group had a higher proportion of males (59.6% vs. 44.4%), current smokers (55.6% vs. 23.5%), and ex-smokers (25.6% vs. 31.8%). In comparison, the control group had a higher percentage of non-smokers (44.8% vs. 18.8%). Demographics and categorical variables are shown in **Tables 1** and **2**.

Physical activity was significantly lower in the case group, with only 17.9% of participants meeting the recommended level, compared to 82.1% in the control group. Poor physical activ-

Table 3. Comparison of physical activity levels between case and control groups (n=554)

Physical Activity Level[a]	Case group (n=277)	Control group (n=277)	*P* value
Recommended	32 (17.9%)	147 (82.1%)	<0.001
Intermediate	71 (47%)	80 (53%)	
Poor	174 (77.7%)	50 (22.3%)	
Total	227 (100%)	227 (100%)	

Notes: Pearson Chi square [a], n (%).

Table 4. Association of physical activity level based on demographic variables across both groups

Demographics variables		Group	Physical Activity Level			*P*-value
			Recommended n (%)	Intermediate n (%)	Poor n (%)	
Age (years)[a]	30-50	Case	16 (13.7%)	33 (28.2%)	68 (58.1%)	0.003
		Control	48 (41.4%)	48 (41.4%)	20 (17.2%)	
	51-80	Case	16 (100%)	38 (23.8%)	106 (66.2%)	
		Control	99 (61.5%)	32 (19.9%)	30 (18.6%)	
Gender[a]	Male	Case	22 (13.3%)	47 (28.5%)	96 (58.2%)	0.52
		Control	68 (55.3%)	28 (22.8%)	27 (22%)	
	Female	Case	10 (8.9%)	24 (21.4%)	78 (69.6%)	
		Control	79 (51.3%)	52 (33.8%)	23 (14.9%)	
Smoking[a]	Current smoker	Case	15 (9.7%)	42 (27.3%)	97 (63%)	<0.001
		Control	19 (29.2%)	38 (58.5%)	8 (12.3%)	
	Ex-smoker	Case	11 (15.5%)	17 (23.9%)	43 (60.6%)	
		Control	50 (56.8%)	16 (18.2%)	22 (25%)	
	Non-smoker	Case	6 (11.5%)	12 (23.1%)	34 (65.4%)	
		Control	78 (62.9%)	26 (21%)	20 (16.1%)	

Notes: Pearson Chi square [a].

ity was prevalent in cases (77.7%) compared to controls (22.3%), P=0.000, as shown in **Table 3**.

Stratified analyses revealed significant associations between physical activity levels and demographic variables, including age and smoking status. In some cases, poorer activity levels were observed predominantly among older individuals, females, and current smokers than their control counterparts (**Table 4**).

The correlation matrix (Supplementary Table 1) reveals strong positive correlations among BMI, LDL, total cholesterol, and triglycerides, indicating a clustered pattern of dyslipidemia in individuals with higher adiposity. HDL demonstrated significant inverse correlations with these variables, supporting its protective role against adverse cardiometabolic profiles.

The univariate logistic regression analysis showed that physical inactivity, smoking status, male gender, higher body mass index, and elevated total cholesterol were significantly associated with increased odds of cardiovascular disease (**Figure 1**). In the multivariable logistic regression model, physical inactivity, smoking, male gender, BMI, total cholesterol, and age group remained independently associated with cardiovascular disease after adjustment for potential confounders (**Figure 2**).

Univariate logistic regression was first conducted to determine the rough correlation between each independent variable and cardiovascular disease (CVD). The variables under analysis included the age group, sex, body mass index (BMI), systolic blood pressure, lipid parameters (low-density lipoprotein, high-density lipoprotein, triglycerides, and total cholesterol), smoking status, and physical activity level. The statistically significant or biologically relevant variables were then included in a multivariable logistic regression model to adjust for potential confounding. The adjusted final model only included gender, smoking status, physical activity level, BMI, total cholesterol, and age group. The lipid components (LDL, HDL, triglycerides, and systolic blood pressure) were

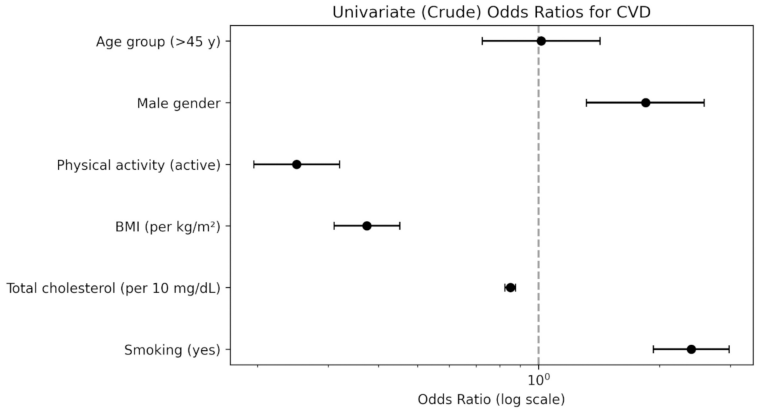

Figure 1. Forest plot showing the odds ratio calculated using the univariate analysis.

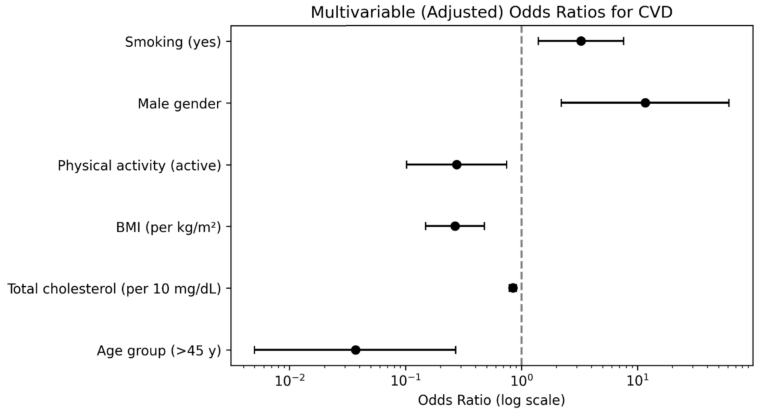

Figure 2. Forest plot showing the adjusted odds ratio calculated using the multivariate analysis.

excluded due to the presence of multicollinearity, which resulted in model instability. This was represented by adjusted odds ratios (ORs) and 95% confidence intervals (CIs) to estimate the independent contribution of each factor to the risk of CVD (Supplementary Table 2).

Discussion

This study demonstrates a strong inverse association between physical activity levels and cardiovascular disease (CVD) in adults. Participants in the control group, who were free of CVD, exhibited significantly higher rates of recommended physical activity compared to those in the case group. In contrast, poor physical activity levels were markedly more prevalent among CVD patients. These findings are consistent with earlier research, including studies by Kubota *et al.* and Lee *et al.*, which have shown that sedentary behavior is associated with increased lifetime risk of CVD and cardiometabolic dysfunction.

In our study, the most notable feature was that out of the total cases, only 17.9% of the cardiovascular disease cases were found to meet the recommended levels of physical activity as compared to 82.1% of controls, with a *p*-value of 0.000, indicating that physical inactivity is an essential factor in the development of cardiovascular disease. These findings have practical implications for regional cardiovascular prevention strategies, where the integration of structured physical activity promotion into routine outpatient visits could reduce the disease burden.

The mechanisms underlying this association are multifactorial. Physical activity is known to enhance endothelial function, reduce systemic inflammation, and improve lipid metabolism, all of which contribute to the reduction of atherogenesis. Our study supports these mechanisms, as participants with lower physical activity levels also showed higher BMI, elevated LDL cholesterol, triglycerides, and blood pressure, alongside reduced HDL levels - factors that collectively define a pro-atherogenic profile. Translating these findings into practice requires the systematic inclusion of physical activity counseling in cardiology and primary care settings. Hospitals can adopt brief physician-led counseling modules, standardized physical-activity prescriptions, and referral pathways to physiotherapists or community exercise programs. Incorporating the WHO “Exercise is Medicine” model into discharge plans or chronic-disease follow-ups may improve long-term adherence.

This work demonstrated a concordance with remarkable differences among the cases and

controls in some key cardiometabolic mar kers, including increased BMI, high systolic blood pressure, and an adverse lipid profile, in cases compared to controls: 29.950±2.82 vs. 23.426±2.85 kg/m^2; 160.231±11.91 vs. 119.606±5.86 mmHg. These findings are consistent with the literature, which summarizes regular physical activity as having various cardioprotective benefits, including improved endothelial function, enhanced lipid metabolism, and better blood pressure regulation. Taken together, this study, as well as the literature to which it refers, emphasizes the importance of a multifaceted intervention that combines the impact of physical activity with the profile of blood lipids in relation to cardiovascular risks, providing a comprehensive perspective. The mean ages for case and control groups were demographically similar: 54.646±14.82 years for cases and 55.180±15.30 years for controls. They fall within the population range that Kakita *et al.* [18] studied, where their participants were 20 years old and above. In their study, as well as in this one, age was a crucial factor in determining the amount of physical activity, with older subjects exhibiting fewer activities. This may often be due to the decreased mobility of these individuals, which is commonly associated with a greater comorbidity burden that contributes to inactivity, thus further heightening cardiovascular risk.

However, BMI in our case group was significantly higher at 29.950±2.82 kg/m^2 compared to the control group's 23.426±2.85 kg/m^2. This corresponds with a powerful link between obesity and CVD risk factors, as evidenced by Liu *et al.* [19] and Wang *et al.* [20]. Similarly, in addition, physical inactivity along with incorrect eating behaviors appeared in these two studies too, as other promotion factors of obesity, along with dyslipidemia, as well as other metabolic impairments. Authors Wang *et al.* [20] stated in this work that being sedentary, when combined with unhealthy eating, may have shown an interaction that was more than additive in increasing the disease risks to the cardiovascular system. In contrast, a significant additive interaction was observed among conditions like hypertension and type 2 diabetes. These findings further support the evidence of higher systolic blood pressure, LDL, and triglycerides, and lower HDL in cases compared to controls, thereby confirming the cumulative effect of these factors on cardiovascular health.

The differences in lipid profile in our study show that cases had higher LDL (168.631±20.68 mg/dL vs. 103.126±13.98 mg/dL) and triglycerides (221.151±43.52 mg/dL vs. 98.014± 28.59 mg/dL) and lower HDL (38.426±5.33 mg/dL vs. 59.472±5.50 mg/dL), which is in tune with the physiological benefits of physical activity on lipid metabolism as demonstrated by Alsaleh and Baniyasin [21]. Indeed, their study concluded that regular exercise improves endothelial function and reduces some modifiable cardiovascular risk factors, such as dyslipidemia. This is further supported by our correlation analysis, which reveals strong positive associations between BMI and LDL ($r=0.650$, $P<0.001$), total cholesterol ($r=0.575$, $P<0.001$), and triglycerides ($r=0.626$, $P<0.001$). In contrast, HDL has shown inverse associations with BMI ($r=-0.700$, $P=0.000$) and LDL ($r=-0.816$, $P=0.000$). This is also depicted in Supplementary Table 1. The outcome then outlines the significant contribution of the adiposity factor to the alteration of an unfavorable lipid profile and an increased risk of CVD.

The mean level of physical activity in our study, a critical determinant, was much lower among the cases, at 17.9%, compared to 82.1% in the control group. These findings are thus supported by Kakita *et al.* [18] in which a low level of physical activity has been reported, more so in elderly CVD patients. Household activities accounted for the majority of the contribution to overall physical activity. Our stratified analysis further supports this, as it has shown that a poor physical activity level is higher among females, current smokers, and those in the older age group. These findings are also in concurrence with Alsaleh and Baniyasin *et al.* [21] work, which reports that perceived barriers related to anxiety and time constraints among persons in the older class or those who are less physically active were consistently high, leading to decreased participation in exercise and physical activity among their members.

Interestingly, Liu *et al.* [19] emphasized the importance of exercise timing and its synchronization with circadian rhythms for achieving maximum benefits to cardiovascular health. This aspect was not covered in our study, but it

is one way in which physical activity interventions could be improved to better suit individual needs. On the other hand, Perry *et al.* [22] used a life-course framework, highlighting that risk from physical inactivity accumulates across life stages. These findings align with our observation that age and gender are critical demographic factors in determining activity levels and their impact on cardiovascular events.

Gender and smoking status were also significantly associated with physical activity levels. Males were more likely to be physically inactive than females, and current smokers had the highest prevalence of poor activity levels. These behavioral patterns underscore the importance of integrating lifestyle counseling - including smoking cessation and physical activity promotion - into CVD prevention and management strategies. This again complements evidence by Wang *et al.* [20] who, in evaluating smoking as a risk factor, which, when combined with poor dietary habits and physical inactivity, compounded the burden of cardiovascular risk factors like dyslipidemia and hypertension, identified it as an additive factor.

While our findings strengthen the case for promoting regular physical activity as a modifiable risk factor for CVD, they also raise concern about low adherence to physical activity guidelines in at-risk populations. The high prevalence of inactivity, even among non-CVD participants, reflects broader public health challenges such as urban sedentary lifestyles, lack of community infrastructure for exercise, and low health literacy. Bays *et al.* [23] stated that cardiovascular risk factors are multifaceted, and obesity is one of the major causes of various comorbidities, including diabetes, hypertension, and dyslipidemia. They further stated that modifying lifestyles can primarily minimize the risk of cardiovascular disease through dietary patterns, such as the Mediterranean diet and the DASH diet, as well as through regular physical exercise. Obesity, especially central, while multiplying the severity of metabolic disorders independently, increases cardiovascular risks [23]. With this, Schrader *et al.* [24] studied the benefits of regular exercise. They discovered that regular physical activity was associated with reduced cases of hypertension, diabetes, and obesity. Participants who exercised regularly also had lower stress levels, less depression, and a generally improved quality of life [24].

Occupational physical activity exerts its influence through mechanisms that are different from those of leisure-time physical activity. In contrast, Xia *et al.* [25] have found that the association of OPA with CVD mortality is U-shaped, such that moderate OPA levels are linked to the lowest mortality risk. In contrast, high or no OPA levels are associated with increased risks. This pattern was particularly pronounced within the lower socioeconomic status group, suggesting that high work strain, combined with insufficient recovery, underlies these adverse outcomes [25]. These findings were supported by those of Holtermann *et al.* [26], where, although LTPA was consistently linked with reduced risk of MACE and all-cause mortality, an OPA revealed a high-level association with increased risk. They attributed the difference to a static and longitudinally prolonged physical demand emanating from occupational activity, contrasting with the dynamic pattern of LTPA, which generally involves rest periods. Notably, the two activity forms independently impacted health outcomes, highlighting the need to take different approaches in their promotion [26].

These findings highlight the importance of considering physical activities from a contextual perspective. Leisure activities are dynamic and controlled, offering immense cardiovascular benefits, as well as psychological ones. On the other hand, occupational physical activity can be hazardous to health when jobs are highly physically demanding and do not allow for sufficient recovery time. These findings suggest that a balanced approach, weighting activities against recovery, especially in at-risk populations, presents a viable option. The consistency of the findings across studies testifies to the need for an integrated approach to health in the population, considering physical activity, eating habits, and other modifying factors such as smoking, while taking into account the peculiarities of each subject in terms of demographic and regional conditions. Overall, the findings of this study highlight that improving physical activity remains a practical, low-cost, and high-impact strategy for reducing cardiovascular disease risk in adults. The strong correlation between physical inactivity and CVD emphasizes the need to include regular physical activity testing and training in outpatient care, cardiac rehabilitation and follow-up with chronic illnesses. Policies like the encouragement of available exercise facilities, office health programs and

community education can also enhance these on a community level. Aligning these initiatives with national prevention frameworks and primary-care protocols may help bridge the current gap between evidence and practice, ultimately reducing the burden of CVD in high-risk populations.

Strengths and limitations

A major strength of this study is the use of a structured case-control design with age- and gender-matched controls, enhancing comparability. Standardized tools and objective lab measurements further strengthened data validity.

However, the study has limitations. Physical activity was self-reported, introducing potential recall bias. Additionally, as a cross-sectional case-control study, causality cannot be established. The study was conducted in a single tertiary care hospital, which may limit generalizability. Other methodological limitations are the bias of recall in self-reported physical activity questionnaires and the existence of unmeasured confounders, such as dietary patterns or medication intake, which may affect the lipid levels. Moreover, the case-control study design does not allow the establishment of temporal causality, and the associations described cannot be interpreted as demonstrating causal effects.

During the refinement of the statistical model, the variables of LDL, HDL, triglycerides, and systolic blood pressure were excluded because they caused the model to become unstable and resulted in wide confidence intervals. The total sample included in the study was adequate, but the following factors probably led to such instability: the high level of inter-correlation between lipid parameters (LDL, HDL, triglycerides and total cholesterol share the common biological pathway), the unequal distribution of data in the case and control groups, and duplication of clinical data, where systolic blood pressure and BMI already represent the associated cardiometabolic risk. Additionally, the failure to account for the variability caused by concurrent medication regimens, especially statins or antihypertensive drugs, which could have artificially lowered lipid or BMI levels among cardiovascular disease patients, made the analysis more difficult. This shortcoming also helps explain the negative relationships with several variables, including total cholesterol and BMI. To counteract data redundancy and improve the accuracy of the results, the total cholesterol was included as a representative lipid outcome, and other variables that were correlated or medication sensitive were not considered in the final adjusted model.

Future implications

Our findings reinforce the role of regular physical activity in CVD prevention and risk reduction. Public health systems must emphasize accessible, community-based exercise programs, especially for high-risk populations such as smokers, men, and individuals with obesity. Future longitudinal studies and randomized trials are necessary to investigate causal pathways and the effects of targeted interventions. These findings underscore the need for national policy frameworks that integrate exercise counseling into primary health-care protocols, with community-level monitoring systems to track adherence and cardiovascular outcomes.

Conclusion

Our study has concluded that physical inactivity is a significant contributing factor to the development of cardiovascular disease. It highlighted the apparent differences in cardiometabolic profiles between cases and controls, reinforcing the role of regular physical activity in mitigating cardiovascular risks. These findings align with existing literature, emphasizing the importance of integrating physical activity into preventive strategies for cardiovascular health. Overall, the study highlights the importance of multifaceted interventions that target physical activity and associated risk factors to reduce the burden of cardiovascular disease.

Disclosure of conflict of interest

None.

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Supplementary Table 1. Correlation matrix between BMI, lipid profile, and related cardiovascular variables

Study Variables	r	*p*-value
BMI and LDL	0.650**	<0.001
BMI and Total Cholesterol	0.575**	<0.001
BMI and Triglycerides (TG)	0.626**	<0.001
BMI and HDL	-0.700**	<0.001
LDL and Total Cholesterol	0.947**	<0.001
LDL and Triglycerides (TG)	0.754**	<0.001
LDL and HDL	-0.816**	<0.001
Total Cholesterol and TG	0.681**	<0.001
Total Cholesterol and HDL	-0.651**	<0.001

**Significant correlation.

Supplementary Table 2. Multivariable and univariate logistic regression for predictors of cardiovascular disease

Predictor	Crude OR (95% CI)	*p* value	Adjusted OR (95% CI)	*p* value
Smoking (yes)	2.40 (1.93-2.98)	<0.001	3.26 (1.40-7.59)	0.006
Male gender	1.85 (1.32-2.58)	<0.001	11.64 (2.20-61.51)	0.004
Physical activity (active)	0.25 (0.20-0.32)	<0.001	0.28 (0.10-0.75)	0.011
BMI (per kg/m^2)	0.37 (0.31-0.45)	<0.001	0.27 (0.15-0.48)	<0.001
Total cholesterol (per 10 mg/dL)	0.85 (0.83-0.88)	<0.001	0.84 (0.79-0.90)	<0.001
Age group (>45 y)	1.02 (0.72-1.42)	0.931	0.04 (0.01-0.27)	0.001