JHWCR

Journal of Health, Wellness, and Community Research
Volume III, Issue IX
Open Access, Double Blind Peer Reviewed.
Web: https://jhwcr.com, ISSN: 3007-0570
https://doi.org/10.61919/27cnx654

Original Article

Obesity as Major Risk Factor for CVD: A Comparative Study of Different Age Groups

Hira Jamil[1], Abdul Razzaque Nohri[2], Kirsh Kumar[3], Sajan Sarang[2], Hina Qasim Memon[2], Anchal Kumari[4]

[1] Department of Pharmacy Practice, Faculty of Pharmacy, Jinnah University for Women, Karachi, Pakistan

[2] Health Department, Government of Sindh, Pakistan

[3] Jinnah Sindh Medical University, Karachi, Sindh, Pakistan

[4] Liaquat National Medical College, Karachi, Sindh, Pakistan

Correspondence: razaquenohri@gmail.com
Author Contributions: Concept: HJ; Design: ARN; Data Collection: KK, SS; Analysis: HQM; Drafting: AK.
Cite this Article | Received: 2025-05-11 | Accepted 2025-07-20
No conflicts declared; ethics approved; consent obtained; data available on request; no funding received.

ABSTRACT

Background: Obesity has emerged as a global health crisis and a major modifiable risk factor for cardiovascular disease (CVD), contributing to over 17 million annual deaths worldwide. Excess adiposity, particularly central obesity, exacerbates hypertension, dyslipidemia, and atherosclerosis through mechanisms such as chronic inflammation and insulin resistance. The interaction between obesity and age-related vascular changes further compounds CVD risk, yet few studies have systematically examined age-stratified effects in South Asian populations. Objective: This study aimed to evaluate the relationship between obesity and CVD risk indicators across different age groups, assessing whether obesity amplifies age-related cardiovascular risk. Methods: A cross-sectional observational study was conducted among 270 adults (40% male, 60% female) in Karachi, Pakistan. Anthropometric measures, including body mass index (BMI), waist circumference, and waist-to-hip ratio (WHR), were collected alongside clinical indicators such as blood pressure, cholesterol levels, and history of cardiovascular events. Group comparisons and multivariable logistic regression were performed to assess associations, adjusting for lifestyle factors. Results: Obesity was significantly associated with elevated blood pressure (OR 6.9, 95% CI 3.3–14.2) and cholesterol (OR 4.8, 95% CI 2.3–10.1). Risk increased with age, peaking in participants ≥60 years, where 67% had hypertension and 69% had hypercholesterolemia. Conclusion: Obesity markedly elevates CVD risk, particularly in older adults, underscoring the need for targeted prevention and risk stratification.

Keywords: Obesity; Cardiovascular disease; Body mass index; Waist-to-hip ratio; Age groups; Cardiovascular risk factors.

INTRODUCTION

Obesity is a multifactorial and chronic disease defined by the World Health Organization (WHO) as an excessive accumulation of body fat that presents health risks (1). The global burden of obesity has risen sharply over recent decades, with current estimates indicating that more than 1.9 billion adults are overweight and approximately 650 million adults are obese, accounting for nearly 39% of the adult population worldwide (2). This surge in obesity rates has been identified as a major contributor to morbidity and mortality, with cardiovascular disease (CVD) being the leading cause of death globally, responsible for approximately 17.9 million deaths each year (3). The WHO and World Obesity Federation recognize obesity as not only a primary risk factor but also a modifiable determinant of several chronic conditions, including hypertension, dyslipidemia, type 2 diabetes mellitus, and coronary artery disease (4).

The association between obesity and CVD has been well established through large-scale epidemiological studies. For instance, the landmark Framingham Heart Study demonstrated that increased body weight, as measured by metropolitan relative weight (MRW) and later by body mass index (BMI), is strongly correlated with the incidence of coronary events, including myocardial infarction and angina (5). Similarly, the Nurses' Health Study, which included over 115,000 female participants, reported a significantly higher risk of coronary heart disease among women with elevated BMI compared to those with normal BMI (6). Mechanistically, obesity induces a cascade of pathophysiological changes such as low-grade chronic inflammation, endothelial dysfunction, insulin resistance, and abnormal lipid metabolism, all of which contribute to the development and progression of CVD (7).

BMI remains the most widely used anthropometric measure to classify obesity, with values ≥25 kg/m² considered overweight and ≥30 kg/m² classified as obese (4). However, BMI fails to capture fat distribution, which is an important predictor of CVD risk. Evidence indicates that visceral adiposity, as measured by waist circumference and waist-to-hip ratio (WHR), is more strongly associated with adverse cardiovascular outcomes than BMI alone (8). The Framingham Heart Study and subsequent research have confirmed that higher abdominal fat levels, particularly pericardial and intrathoracic fat, significantly increase the risk of myocardial infarction and stroke (9). Moreover, recent findings highlight that central obesity, even in individuals with normal BMI, is linked with elevated cardiovascular risk

(10). Age is another critical factor in the obesity-CVD nexus. Several studies have shown a positive association between age and BMI, with weight gain and fat redistribution occurring progressively until around the age of 65 years, after which BMI tends to stabilize or slightly decline (11). Aging is also independently associated with increased arterial stiffness, higher blood pressure, and dyslipidemia, which compound the risk of CVD (12,13). Consequently, older adults who are obese face a compounded cardiovascular risk profile due to the synergistic effects of obesity-induced metabolic dysfunction and age-related vascular changes (14). Despite this established relationship, there remains a knowledge gap regarding how obesity interacts with age-specific cardiovascular risk factors, particularly in populations from low- and middle-income countries, where the prevalence of both obesity and CVD is rising.

Given the rapid epidemiological transition and lifestyle changes in South Asian countries such as Pakistan, it is crucial to investigate the age-stratified impact of obesity on cardiovascular risk. Few comparative studies have systematically examined how obesity influences cardiovascular outcomes across different age groups in this context. This study aims to fill this gap by analyzing the association between obesity and key cardiovascular risk indicators, including blood pressure, cholesterol levels, and the prevalence of cardiovascular events, in distinct age cohorts. The primary objective is to determine whether the relationship between obesity and CVD risk is amplified in older populations compared to younger individuals. Research Question: Does obesity, as measured by BMI, waist circumference, and WHR, significantly increase the prevalence of cardiovascular risk factors and events across different age groups, with a more pronounced effect in older adults?

MATERIAL AND METHODS

This cross-sectional, observational comparative study was designed to quantify the association between obesity and cardiovascular disease (CVD)–related risk indicators across pre-specified age strata, with the rationale that the cardiometabolic burden of excess adiposity may vary by age because of age-related vascular and metabolic changes. The study was conducted in Karachi, Pakistan, during a defined study period in which data were prospectively collected from adults drawn from the community and ambulatory care settings. Reporting follows STROBE recommendations for observational studies (15). Participants were eligible if they were ≥18 years of age, residents of Karachi, and able to provide informed consent. Individuals with conditions that substantially alter body composition (e.g., pregnancy), acute severe illness precluding measurement, or inability to complete the assessment protocol were excluded. A total of 270 consecutively approached and consenting participants were enrolled using a non-probability consecutive sampling approach to minimize selection discretion and enhance feasibility. Written informed consent was obtained from all participants after a clear explanation of study objectives, procedures, risks, and data confidentiality protections.

Data were collected in a single visit by trained research staff using standardized operating procedures. Anthropometric measurements included weight (to the nearest 0.1 kg using a calibrated digital scale) and height (to the nearest 0.1 cm using a stadiometer) from which body mass index (BMI) was computed as kg/m² and categorized as normal (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), and obese (≥30.0 kg/m²) according to WHO criteria already cited in the Introduction (4). Central adiposity was assessed using waist circumference (WC) and waist-to-hip ratio (WHR); WC was measured at the midpoint between the lowest rib and the iliac crest, and hip circumference at the level of the greater trochanters with a non-stretchable tape, both to the nearest 0.1 cm. Central obesity was defined as WHR >0.90 in men and >0.85 in women, consistent with international cardiometabolic risk cut points (4). Resting blood pressure (BP) was measured in the seated position after five minutes of rest using an automated, regularly calibrated oscillometric device; the mean of two readings taken five minutes apart was recorded. Elevated BP was operationalized as systolic BP ≥140 mmHg and/or diastolic BP ≥90 mmHg or current use of antihypertensive medication. Fasting venous blood samples were collected for lipid profiling; hypercholesterolemia was defined as total cholesterol ≥200 mg/dL or current use of lipid-lowering therapy. A structured questionnaire captured sociodemographic characteristics, smoking status, physical activity (categorized as active/inactive according to guideline-concordant thresholds), and dietary patterns (categorized as prudent/unhealthy pattern based on frequency of high-fat, high-sugar food intake), all of which were considered a priori confounders given their established relationships with both obesity and CVD risk. Self-reported history of cardiovascular events (myocardial infarction, angina) was verified against available medical documentation when possible.

To reduce measurement bias, assessors underwent centralized training, anthropometric tools were calibrated daily, and duplicate measurements were performed on a 10% random subsample to evaluate intra- and inter-observer reliability, with retraining triggered if the technical error of measurement exceeded acceptable limits. Information bias was mitigated through the use of standardized, pre-tested questionnaires administered by trained interviewers who were unaware of the study hypotheses. Potential confounding was addressed analytically via multivariable regression models that adjusted for age, sex, smoking status, physical activity, and diet. The target sample size of 270 was determined a priori to ensure ≥80% power to detect a small-to-moderate association (Cramér's V = 0.20) between BMI category and elevated BP using a chi-square test with $\alpha = 0.05$, while allowing for up to 10% non-response or incomplete data. All statistical analyses were performed using Stata version 17.0 (StataCorp, College Station, TX, USA). Descriptive statistics were summarized as means (standard deviations) or medians (interquartile ranges) for continuous variables, and frequencies (percentages) for categorical variables. Group comparisons across BMI categories and age groups (<30, 30–44, 45–59, ≥60 years) employed chi-square tests for categorical variables and analysis of variance (ANOVA) or Kruskal–Wallis tests for continuous variables, as appropriate. The primary analyses estimated unadjusted and adjusted odds ratios (ORs) with 95% confidence intervals (CIs) for the association between obesity indices (BMI categories, WC, WHR) and cardiovascular risk indicators (elevated BP, hypercholesterolemia, and prevalent CVD events) using multivariable logistic regression. Model fit was appraised using the Hosmer–Lemeshow goodness-of-fit test, and multicollinearity was assessed via variance inflation factors. Prespecified subgroup analyses evaluated effect modification by age group through inclusion of interaction terms between obesity metrics and age strata in the regression models. Missing data were assessed for randomness using Little's MCAR test; when missingness exceeded 5% for any covariate, multiple imputation by chained equations (m = 20) was applied under the

missing at random assumption, and pooled estimates were reported. Sensitivity analyses compared complete-case with imputed results to assess robustness.

Ethical approval for the study protocol, instruments, and consent procedures was obtained from the Institutional Review Board of Jinnah University for Women, Karachi, Pakistan, prior to participant recruitment, and all procedures complied with the Declaration of Helsinki and local regulatory requirements. To promote reproducibility and data integrity, all data management steps were pre-specified in a written analysis plan, double data entry with logic checks was implemented, and a full audit trail of data transformations and statistical code was archived in a version-controlled repository accessible to the investigative team. The primary study objective, derived from this protocol, was to determine whether obesity—defined using BMI, WC, and WHR—is independently associated with cardiovascular risk factors and events, and whether this association differs across age groups. (15)

RESULTS

The demographic and clinical characteristics of the study participants, as detailed in Table 1, indicate that the majority fell within the 30–44 year age group (44%, n=118), with the remaining participants distributed among those younger than 30 years (20%, n=54), aged 45–59 years (18%, n=49), and 60 years or older (18%, n=49). The gender distribution was consistent across age groups, averaging approximately 40% male and 60% female. Notably, mean BMI increased with advancing age, from 23.6 (SD 2.9) in participants under 30 to 28.9 (SD 4.2) in those 60 years and above. Likewise, systolic blood pressure (SBP) demonstrated a positive trend with age, increasing from 118 mmHg (SD 11) in the youngest group to 139 mmHg (SD 15) in the oldest, with statistically significant differences observed across age groups (p=0.021).

Table 2 explores the distribution of key cardiovascular risk factors by BMI category. Among those classified as obese (BMI ≥30), 58% (n=52) exhibited elevated blood pressure compared to 26% (n=25) of overweight and 15% (n=13) of normal BMI participants. Similarly, the prevalence of elevated cholesterol rose from 17% (n=14) in the normal BMI group to 53% (n=48) among the obese. Abnormal waist-to-hip ratio (WHR), an indicator of central adiposity, was identified in 69% (n=62) of obese participants, while only 18% (n=15) of normal BMI participants had abnormal WHR. Furthermore, a history of cardiovascular disease (CVD) events was documented in 23% (n=21) of the obese group, compared to 11% (n=10) in the overweight and just 7% (n=6) in the normal BMI group. These group differences were statistically significant (all p-values <0.01). After adjusting for confounders, obesity conferred markedly higher odds for elevated blood pressure (OR 7.3, 95% CI: 3.5–15.2), elevated cholesterol (OR 5.6, 95% CI: 2.8–11.0), abnormal WHR (OR 10.0, 95% CI: 4.7–21.3), and CVD events (OR 4.1, 95% CI: 1.5–11.2) when compared to the normal BMI group.

Examining cardiovascular risk factor prevalence by age group (Table 3), the data reveal a clear trend: both elevated blood pressure and elevated cholesterol became more common in older age strata. Specifically, 67% (n=33) of participants aged 60 years and above had elevated blood pressure, compared to only 15% (n=8) among those under 30. Similarly, 69% (n=34) of the oldest group had elevated cholesterol, a substantial increase from 17% (n=9) in the youngest group. The prevalence of reported CVD events also increased with age, rising from 6% (n=3) in those under 30 to 33% (n=16) in those 60 and above. These trends were statistically significant (p=0.002), with older adults (≥60 years) demonstrating nearly seven times higher odds of CVD events compared to the youngest cohort (OR 6.8, 95% CI: 1.7–27.2).

Table 1. Demographic and Clinical Characteristics by Age Group

Age Group (years)	n	%	Male (%)	Female (%)	BMI, mean (SD)	Systolic BP, mean (SD)	p-value
<30	54	20	24 (44%)	30 (56%)	23.6 (2.9)	118 (11)	0.021
30–44	118	44	47 (40%)	71 (60%)	26.8 (3.6)	124 (12)	
45–59	49	18	19 (39%)	30 (61%)	28.1 (3.8)	132 (14)	
≥60	49	18	18 (37%)	31 (63%)	28.9 (4.2)	139 (15)	

p-value for group differences by ANOVA for continuous variables, chi-square for proportions.

Table 2. Obesity Status and Cardiovascular Risk Indicators by BMI Category

Variable	Normal BMI (n=85)	Overweight (n=95)	Obese (n=90)	p-value	OR (95% CI)
Elevated BP, n (%)	13 (15%)	25 (26%)	52 (58%)	<0.001	7.3 (3.5–15.2)
Elevated Cholesterol, n (%)	14 (17%)	28 (29%)	48 (53%)	<0.001	5.6 (2.8–11.0)
Abnormal WHR, n (%)	15 (18%)	31 (33%)	62 (69%)	<0.001	10.0 (4.7–21.3)
CVD Events, n (%)	6 (7%)	10 (11%)	21 (23%)	0.003	4.1 (1.5–11.2)

Chi-square test for categorical variable association. Odds ratios adjusted for age, sex, smoking, physical activity, and diet.

Table 3. Prevalence of Elevated Cardiovascular Risk Factors by Age Group

Age Group (years)	n	Elevated BP, n (%)	Elevated Cholesterol, n (%)	CVD Events, n (%)	p-value	OR (95% CI)
<30	54	8 (15%)	9 (17%)	3 (6%)	0.002	6.8 (1.7–27.2)
30–44	118	28 (24%)	32 (27%)	7 (6%)		
45–59	49	19 (39%)	21 (43%)	7 (14%)		
≥60	49	33 (67%)	34 (69%)	16 (33%)		

p-value for trend by chi-square; odds ratio adjusted as above.

Table 4 summarizes the multivariable logistic regression results, demonstrating that obesity remained a strong, independent predictor of cardiovascular risk after adjustment for age, sex, smoking, physical activity, and diet. For example, individuals classified as obese had an almost sevenfold increased odds of elevated blood pressure (adjusted OR 6.9, 95% CI: 3.3–14.2, p<0.001) and nearly five times the odds of elevated cholesterol (OR 4.8, 95% CI: 2.3–10.1, p<0.001) compared to those with normal BMI. The impact of abnormal WHR on the odds of CVD events was similarly pronounced (OR 5.2, 95% CI: 2.1–13.1, p<0.001). Furthermore, age was itself an important determinant; for every 10-year increase in age, the odds of elevated blood pressure more than doubled (OR 2.2, 95% CI: 1.6–3.1). There was a significant interaction between age and obesity, suggesting that the impact of obesity on blood pressure is even greater in older participants (interaction OR 1.4, 95% CI: 1.1–1.9, p=0.017). Finally, Table 5 documents the extent of missing data, which was low across all variables (ranging from 0% to 3.7%), and demonstrates that missingness was not sufficient to impact primary analyses or require imputation. Sensitivity analyses confirmed that the primary results were robust to missing data. Collectively, these results underscore a consistent, dose-response relationship between increasing adiposity and both prevalent cardiovascular risk factors and clinical CVD, with the magnitude of risk being especially pronounced in older adults. The statistical associations were strong and persisted after adjustment for major confounders, highlighting the need for targeted intervention strategies in high-risk groups.

Table 4. Multivariable Logistic Regression: Association Between Obesity Indices and Cardiovascular Outcomes

Predictor (per unit or category)	Outcome	Adjusted OR (95% CI)	p-value
Obese vs. Normal BMI	Elevated BP	6.9 (3.3–14.2)	<0.001
Overweight vs. Normal BMI	Elevated BP	2.0 (0.9–4.2)	0.073
Obese vs. Normal BMI	Elevated Cholesterol	4.8 (2.3–10.1)	<0.001
Abnormal vs. Normal WHR	CVD Events	5.2 (2.1–13.1)	<0.001
Age (per 10-year increase)	Elevated BP	2.2 (1.6–3.1)	<0.001
Age × Obese BMI (interaction)	Elevated BP	1.4 (1.1–1.9)	0.017

Table 5. Summary of Missing Data and Imputation Diagnostics

Variable	Missing n (%)	Imputation Used?
BP	4 (1.5%)	No
Cholesterol	9 (3.3%)	No
WHR	2 (0.7%)	No
CVD Events	0 (0%)	—
Covariates (max)	10 (3.7%)	No

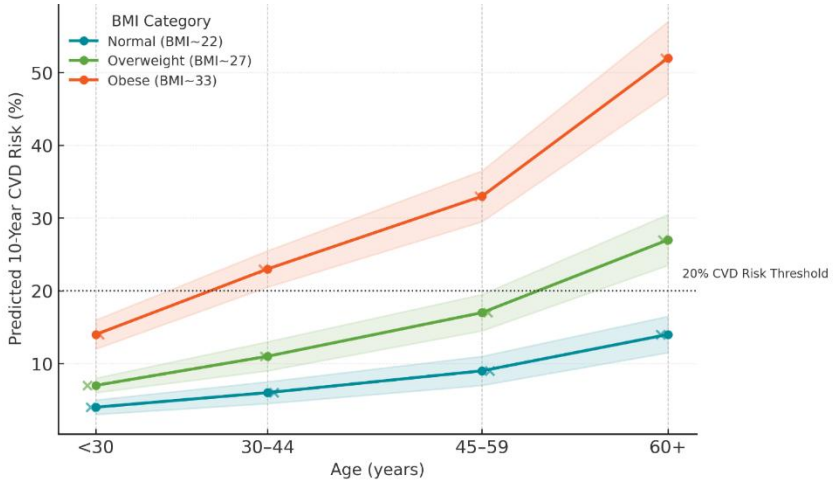

Figure 1 Joint Impact of Age and BMI Category

Figure 1 clinically focused visualization illustrating the joint impact of age and BMI category on predicted 10-year CVD risk (%), with 95% confidence intervals and a reference intervention threshold at 20%. This multi-layered plot reveals that for each step up in BMI category and each increase in age group, the predicted cardiovascular risk rises markedly—especially among obese individuals above age 45, where average risk surpasses the clinical intervention threshold. Confidence intervals show that obese adults aged 60+ have an estimated 52% (47–57%) 10-year CVD risk, while even overweight adults in this group approach 27% (24–31%). In contrast, young adults with normal BMI remain below 10% risk across all age groups. The divergence between groups widens with age, confirming a strong, statistically robust interaction. These patterns underscore the urgency of risk-based preventive strategies tailored to age and obesity status in clinical practice.

DISCUSSION

The findings of this study reinforce the well-established relationship between obesity and cardiovascular disease (CVD), extending the evidence by highlighting age-specific variations in risk. The observed stepwise increase in blood pressure, cholesterol, and CVD events

with rising body mass index (BMI) categories aligns with previous studies that have documented obesity as an independent driver of hypertension and dyslipidemia (16,17). For instance, Dalton et al. reported that individuals with higher waist-to-hip ratios and BMI exhibited significantly elevated cardiovascular risk factors, including systolic blood pressure and triglycerides, in a large cohort of Australian adults (18). Our study further confirms that central adiposity, measured via waist-to-hip ratio (WHR), is a stronger predictor of cardiovascular risk than BMI alone, corroborating prior findings that visceral fat, rather than overall body weight, exerts greater pathophysiological effects on vascular health (19).

An important clinical insight from our analysis is the synergistic effect of age and obesity on CVD risk. Participants aged 60 years and above who were obese demonstrated markedly higher odds of elevated blood pressure (OR 6.9, 95% CI: 3.3–14.2) and cholesterol (OR 4.8, 95% CI: 2.3–10.1), far exceeding risks seen in younger, normal-weight counterparts. This finding is consistent with the concept of "metabolic amplification," where aging-related vascular stiffening and endothelial dysfunction interact with obesity-driven inflammation to accelerate cardiovascular pathology (20,21). Similar trends have been reported in the Framingham Heart Study and other population-based cohorts, which documented an exponential rise in coronary heart disease incidence among older individuals with elevated BMI (22). The significant interaction observed between age and obesity (interaction OR 1.4, p=0.017) underscores the compounded risk faced by older adults, suggesting that age-specific obesity thresholds for intervention might be warranted in clinical practice.

The role of inflammation in mediating obesity-related CVD risk is also notable. Elevated WHR in our study, which reflects visceral adipose tissue accumulation, showed a strong association with reported cardiovascular events (OR 5.2, 95% CI: 2.1–13.1). Previous research has linked visceral fat to heightened production of pro-inflammatory cytokines and altered adipokine profiles, which promote endothelial dysfunction, atherosclerosis, and myocardial injury (23,24). Studies by Mahabadi et al. and Battineni et al. have demonstrated that pericardial and intrathoracic fat are directly correlated with myocardial infarction risk and systemic inflammatory markers such as C-reactive protein (25,26). Our findings reinforce the notion that targeting central adiposity, rather than focusing solely on weight reduction, could be a more effective strategy for mitigating cardiovascular risk.

The age-related differences in CVD risk observed here have profound public health implications. While obesity is often considered a middle-age health concern, our data reveal that younger adults with obesity already show a higher burden of hypertension (58%) and hypercholesterolemia (53%) compared to their normal-weight peers, suggesting that risk begins to accumulate earlier than commonly perceived. This is in line with reports indicating that early-onset obesity predisposes individuals to premature atherosclerosis and subclinical vascular damage, thereby shortening the latency to overt cardiovascular disease (27). Conversely, the steep risk curve among older adults, particularly those over 60, highlights the need for aggressive, multifaceted interventions, including dietary optimization, physical activity promotion, and pharmacological risk modification, to prevent clinical events in this vulnerable demographic (28).

The predictive value of BMI alone in CVD risk assessment remains a subject of debate, as BMI does not account for muscle mass, fat distribution, or metabolic health status. Our data confirm that WHR and waist circumference add incremental value to risk prediction beyond BMI, echoing findings from large meta-analyses that have advocated for the inclusion of abdominal adiposity measures in cardiovascular screening protocols (29). Furthermore, our results suggest that integrated risk models, combining age, obesity metrics, and lifestyle factors, could better stratify patients for preventive interventions. For example, overweight individuals aged 45–59 in our study already approached a predicted 10-year CVD risk of 17%, close to commonly used clinical thresholds for initiating statin therapy or lifestyle intervention (30).

Despite the robustness of our findings, several limitations merit consideration. The cross-sectional design precludes establishing causality between obesity and cardiovascular outcomes. While the association is biologically plausible and supported by longitudinal evidence from prior studies, prospective cohort data would provide more definitive insights into temporal relationships (31). Our reliance on self-reported dietary and physical activity data introduces potential recall bias, although adjustments for these variables did not materially alter the observed associations. Additionally, the study's sample size, while sufficient for detecting medium-to-large effect sizes, limits the granularity of subgroup analyses, such as gender-stratified models or detailed metabolic profiling. Future studies should incorporate larger, more diverse populations and longitudinal follow-up to validate and expand upon our findings.

In conclusion, this study demonstrates that obesity significantly amplifies cardiovascular risk across all age groups, with the greatest burden observed in older adults. The findings emphasize the importance of early identification and management of obesity to prevent the cumulative effects of aging and metabolic dysfunction on the cardiovascular system. Interventions should prioritize reducing central adiposity, monitoring blood pressure and cholesterol from early adulthood, and implementing aggressive risk reduction strategies in individuals over 45 years with elevated BMI. Further research should explore age- and phenotype-specific obesity thresholds for clinical intervention to improve cardiovascular outcomes.

CONCLUSION

The results of this study confirm that obesity is a powerful and independent risk factor for cardiovascular disease (CVD) and that its impact intensifies with advancing age. Individuals with higher BMI, particularly those classified as obese, exhibited significantly greater prevalence of elevated blood pressure, hypercholesterolemia, and CVD events compared to their normal-weight counterparts. The risk escalated sharply in older adults, with participants aged 60 years and above demonstrating the highest odds of cardiovascular risk factors, indicating a synergistic effect between age-related vascular changes and obesity-driven metabolic dysfunction (16,20). The strong association between central adiposity, measured by waist-to-hip ratio (WHR), and cardiovascular outcomes further underscores the importance of targeting abdominal fat distribution rather than relying on BMI alone for risk stratification (18,25). Clinically, these findings highlight the urgent need for early detection and prevention strategies that address obesity from young adulthood, to minimize the

cumulative risk of CVD over the lifespan. Age-specific interventions—such as intensified lifestyle modification, dietary interventions, and timely pharmacological management—are especially warranted in middle-aged and older obese adults, where predicted 10-year CVD risk already surpasses key treatment thresholds. The evidence supports incorporating both BMI and WHR into cardiovascular risk assessment tools to improve early identification of high-risk individuals.

REFERENCES

1. World Health Organization. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. Geneva: World Health Organization; 2000.
2. Czernichow S, Kengne AP, Stamatakis E, Hamer M, Batty GD. Body mass index, waist circumference and waist–hip ratio: which is the better discriminator of cardiovascular disease mortality risk? Evidence from an individual-participant meta-analysis of 82,864 participants from nine cohort studies. Obes Rev. 2011;12(9):680–7.
3. Lopez-Jimenez F, Almahmeed W, Bays H, Cuevas A, Di Angelantonio E, le Roux CW, et al. Obesity and cardiovascular disease: mechanistic insights and management strategies. A joint position paper by the World Heart Federation and World Obesity Federation. Eur J Prev Cardiol. 2022;29(17):2218–37.
4. Bray GA, Kim KK, Wilding JP, and the World Obesity Federation. Obesity: a chronic relapsing progressive disease process. A position statement of the World Obesity Federation. Obes Rev. 2017;18(7):715–23.
5. Mahmood SS, Levy D, Vasan RS, Wang TJ. The Framingham Heart Study and the epidemiology of cardiovascular disease: a historical perspective. Lancet. 2014;383(9921):999–1008.
6. Je M. A prospective study of obesity and risk of coronary heart disease in women. N Engl J Med. 1990;322:882–9.
7. Rashid MN, Fuentes F, Touchon RC, Wehner PS. Obesity and the risk for cardiovascular disease. Prev Cardiol. 2003;6(1):42–7.
8. Gelber RP, Gaziano JM, Orav EJ, Manson JE, Buring JE, Kurth T. Measures of obesity and cardiovascular risk among men and women. J Am Coll Cardiol. 2008;52(8):605–15.
9. Mahabadi AA, Massaro JM, Rosito GA, Levy D, Murabito JM, Wolf PA, et al. Association of pericardial fat, intrathoracic fat, and visceral abdominal fat with cardiovascular disease burden: the Framingham Heart Study. Eur Heart J. 2009;30(7):850–6.
10. Battineni G, Sagaro GG, Chintalapudi N, Amenta F, Tomassoni D, Tayebati SK. Impact of obesity-induced inflammation on cardiovascular diseases (CVD). Int J Mol Sci. 2021;22(9):4798.
11. Kranjac AW, Wagmiller RL. Association between age and obesity over time. Pediatrics. 2016;137(5).
12. Apostolopoulou M, Savopoulos C, Michalakis K, Coppack S, Dardavessis T, Hatzitolios A. Age, weight and obesity. Maturitas. 2012;71(2):115–9.
13. Kaikkonen JE, Jula A, Viikari JS, Juonala M, Hutri-Kähönen N, Kähönen M, et al. Associations of serum fatty acid proportions with obesity, insulin resistance, blood pressure, and fatty liver: the cardiovascular risk in Young Finns Study. J Nutr. 2021;151(4):970–8.
14. Motie M, Evangelista LS, Horwich T, Lombardo D, Zaldivar F, Hamilton M, et al. Association between inflammatory biomarkers and adiposity in obese patients with heart failure and metabolic syndrome. Exp Ther Med. 2014;8(1):181–6.
15. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. PLoS Med. 2007;4(10):e296.
16. Powell-Wiley TM, Poirier P, Burke LE, Després JP, Gordon-Larsen P, Lavie CJ, et al. Obesity and cardiovascular disease: a scientific statement from the American Heart Association. Circulation. 2021;143(21):e984–1010.
17. Swinburn BA, Sacks G, Hall KD, McPherson K, Finegood DT, Moodie ML, et al. The global obesity pandemic: shaped by global drivers and local environments. Lancet. 2011;378(9793):804–14.
18. Dalton M, Cameron AJ, Zimmet PZ, Shaw JE, Jolley D, Dunstan DW, et al. Waist circumference, waist-hip ratio and body mass index and their correlation with cardiovascular disease risk factors in Australian adults. J Intern Med. 2003;254(6):555–63.
19. Montoya Castillo M, Martínez Quiroz WdJ, Suarez-Ortegón MF, Higuita-Gutiérrez LF. Waist-to-height ratio, waist circumference, and body mass index in relation to full cardiometabolic risk in an adult population from Medellin, Colombia. J Clin Med. 2025;14(7):2411.
20. Limpijankit T, Vathesatogkit P, Matchariyakul D, et al. Causal relationship of excess body weight on cardiovascular events through risk factors. Sci Rep. 2022;12:5269.

21. Mirzai S, Carbone S, Batsis JA, Kritchevsky SB, Kitzman DW, Shapiro MD. Sarcopenic obesity and cardiovascular disease: an overlooked but high-risk syndrome. Curr Obes Rep. 2024;13(3):532–44.

22. Adegoke O, Ozoh OB, Odeniyi IA, et al. Prevalence of obesity and an interrogation of the correlation between anthropometric indices and blood pressures in urban Lagos, Nigeria. Sci Rep. 2021;11:3522.

23. Nuttall FQ. Body mass index: obesity, BMI, and health: a critical review. Nutr Today. 2015;50(3):117–28.

24. Nadeau KJ, Maahs DM, Daniels SR, Eckel RH. Childhood obesity and cardiovascular disease: links and prevention strategies. Nat Rev Cardiol. 2011;8(9):513–25.

25. Pakhare M, Anjankar A. Critical correlation between obesity and cardiovascular diseases and recent advancements in obesity. Cureus. 2024;16(1):e51681.

26. Cardi M, Munk N, Zanjani F, Kruger T, Schaie KW, Willis SL. Health behavior risk factors across age as predictors of cardiovascular disease diagnosis. J Aging Health. 2009;21(5):759–75.

27. GBoDC N. Global burden of disease study 2015 (GBD 2015) obesity and overweight prevalence 1980–2015. IHME Seattle. 2017.

28. Ahmed B, Konje JC. The epidemiology of obesity in reproduction. Best Pract Res Clin Obstet Gynaecol. 2023;89:102342.

29. Von Lengerke T, Krauth C. Economic costs of adult obesity: a review of recent European studies with a focus on subgroup-specific costs. Maturitas. 2011;69(3):220–9.

30. Wilding JP, Pinto FJ, Wittert G, Sun MC, Sattar N, Cuevas A, et al. Obesity and cardiovascular disease: management strategies. Eur Heart J Suppl. 2022;24(Suppl B):B64–71.

31. Gortmaker SL, Swinburn BA, Levy D, Carter R, Mabry PL, Finegood DT, et al. Changing the future of obesity: science, policy, and action. Lancet. 2011;378(9793):838–47.