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ORIGINAL ARTICLE

The Role of Hypertension and Obesity in the Pathogenesis and Progression of Ischemic Heart Disease. A Clinical and Epidemiological Perspectives

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ABSTRACT

Background: Hypertension and obesity are major contributors to ischemic heart disease (IHD), a leading cause of global morbidity and mortality. Both conditions independently and synergistically accelerate cardiovascular damage through mechanisms such as endothelial dysfunction, arterial stiffness, and systemic inflammation. While their individual roles are well-established, their combined impact on IHD progression in high-burden populations, such as in Pakistan, requires further investigation.

Aims and Objectives: This study aimed to evaluate the prevalence of hypertension and obesity among IHD patients, identify associated clinical and biochemical risk factors, and assess the contribution of lifestyle behaviors to disease progression. The goal was to provide evidence-based insights for targeted interventions.

Methodology: A cross-sectional observational study was conducted on 200 participants (aged 30–75 years) with confirmed IHD at Nishtar Hospital, Multan, Pakistan (February 2022-December 2022). Data were collected through structured interviews, medical records, anthropometric measurements, and biochemical investigations. Statistical analyses included chi-square tests, t-tests, and ANOVA, with significance set at $p < 0.05$.

Results: This cross-sectional study included 200 participants (mean age 55.3 ± 10.4 years, 58.5% males). Hypertension was prevalent in 74%, with mean systolic and diastolic pressures of 142.7 ± 16.5 mmHg and 89.3 ± 12.2 mmHg, respectively ($p < 0.001$). Obesity affected 62% of participants, with central obesity in 71.2% ($p < 0.01$). Elevated total cholesterol (65%), LDL cholesterol (73%), and hs-CRP (64%) ($p < 0.001$) were observed. Behavioral risks included high sodium intake (75%), sedentary lifestyle (48%), and smoking (34% active, 28% former). These findings demonstrate the synergistic role of hypertension and obesity in exacerbating IHD progression.

Conclusion: Hypertension and obesity significantly contribute to IHD pathogenesis. Integrated clinical management and public health initiatives targeting these conditions through lifestyle and dietary modifications are critical. Future research should focus on personalized approaches and long-term outcomes to reduce IHD burden.

Keywords: Hypertension, Obesity, Ischemic Heart Disease, Cardiovascular Risk, Public Health, Lifestyle Modification.

INTRODUCTION

The disease is also one of the major health concerns of the world, and millions of people die from it annually. In addition, more people are sickening from obesity and high blood pressure, which are modern diseases[1]. Both obesity and hypertension are pathophysiological pathways that promote atherosclerosis and cardiovascular injury and these conditions are defined as excessive accumulation of body fat (obesity) and as persistently high arterial blood pressure (hypertension). Vascular remodeling, endothelial dysfunction and the formation of atherosclerotic plaque are all part of the hypertensive process. Elevation by shear stress on the arteries walls contributes to oxidation, inflammation, and endothelial dysfunction with hypertension. RAAS activation in progression of arterial stiffness and increased myocardial oxygen demand that culminates in ischemia[2].

A study based on a cross-sectional survey using Pakistan's most recent National Health and Nutrition Examination Survey data shows that about two thirds of Pakistani adults are now technically overweight or obese according to the body mass index or BMI. One third of them is obese (defined as BMI at or above 30)1, 2 but the numbers are on their own troubling, but don't account for the costs of being obese or its complications (hypotension for example). Therefore, it is unsurprising that hypertension is increasing in proportion to obesity[3]. The research clearly indicates that obesity is the direct cause of hypertension in at least 75 percent of cases. Thus, there is therefore a need for a therapeutic intervention for obesity to be able to effectively control BP in obesity patients or the development of obesity hypertension[4, 5].

The latest studies indicate that the cost of medical treatment of obesity and obesity related disease in the United States in the year 2008 was about $157 billion. It is also expected to reach more than $350 billion or 21 percent of all health care expenses by 2018, according to the study. In case we were able to measure blood pressure in population during late pre-antibiotic era in the early 20th century, then the association of obesity and hypertension was known then also[6]. In 2010, the Framingham Heart Study provided prospective data on the evidence based cross sectional association between body weight and blood pressure. Between mid-2020 and now, the nature of the relationship between blood pressure and body weight had not been clarified by primary clinical and population based research[7, 8].

Obesity and high blood pressure are known to be associated with IHD. Central obesity is characterized by increased sympathetic activity and RAAS and renal sodium retention with hypertension. Leptin and high insulin resistance harm endothelial function, and increase sensitive nervous system which amplifies cardiovascular injury. If obesity and hypertension coexist, there is a synergistic risk for IHD[9]. The problem is that central obesity, a fairly typical feature of hypertensive patients, is bad because it makes renal sodium retention, sympathetic hyperactivity and activation of the renin-angiotensin-aldosterone system all worse. Leptin and insulin increase sympathetic tone and decrease endothelial function contributing to cardiovascular damage. The present paper analyzes these interrelations with emphasis on the clinical aspects as well as on the public health initiatives[10].

MATERIAL AND METHODS

A cross-sectional observational study was used to understand the relationship between hypertension, obesity and ischemic heart disease (IHD). The study was meticulously planned and conducted between February 2022 to December 2022 at the Cardiology and Internal Medicine departments of Nishtar Hospital Multan Pakistan.

Institutional review board ethical approval was granted, and compliance with the principles of Declaration of Helsinki was assured. The detailed information about the purpose, methods, and possible risks and benefits of the study were explained to all participants who provided written informed consent. Participants were assured that confidentiality would be maintained and their right to withdraw from the study at any time, without risk to their medical care. The anonymized data were securely stored, and were only accessible to authorized personnel.

The study consisted of adults aged 30 to 75 years with confirmed IHD defined by clinical history, electrocardiographic, and echocardiographic findings. Participants were recruited using a purposive sampling method and sample size was determined based on 95% confidence interval, 80% statistical power and regional prevalence of IHD in hypertensive and obese populations. Participants were required to have IHD confirmed per ICD-10 coding, documented or self reported history of hypertension or obesity, and willingness to participate. To maintain the integrity of the dataset, individuals with congenital heart disease, severe comorbidities such as end stage renal disease or malignancy, or incomplete medical records were excluded.

The process of data collection was systematic and comprehensive. Cuff blood pressure measurements were taken using a calibrated sphygmomanometer according to the American Heart Association guidelines. Body mass index (BMI) and waist-to-hip ratios were calculated, using standard anthropometric techniques in a controlled environment to ensure consistency, and obesity assessments were performed. Structured interviews were used to collect detailed medical histories and review of patient records were made to document duration and management of hypertension and obesity and lifestyle factors (smoking, physical activity levels and dietary habits).

Fasting blood glucose, lipid profiles (total cholesterol, LDL cholesterol, and HDL cholesterol), and high sensitivity C reactive protein (hs CRP) were measured and biochemical investigations were done. Structural anomalies of the heart and left ventricular hypertrophy were detected by echocardiographic evaluations. Sociodemographic characteristics, family history of cardiovascular disease and other risk factors were collected by validated questionnaires for additional epidemiological data. The Framingham Risk Score was used to categorize participants into cardiovascular risk groups.

All measurements were conducted by trained professionals using calibrated instruments to ensure quality and reliability of the data. To reduce errors, the data was cross verified by independent reviewers. Data with missing or inconsistent data were excluded from analysis due to data integrity. Wherever possible, medical records were used to corroborate self reported information such as smoking status and dietary habits to reduce reporting bias.

SPSS version 26 was used for statistical analysis. Mean ± standard deviation was used to express continuous variables and percentages to present categorical variables. Associations between categorical variables were evaluated using chi-square tests, and differences in continuous variables were analyzed with t tests and one way ANOVA. Statistical significance was set at $p < 0.05$.

RESULTS

200 participants were included with a mean age of 55.3 ± 10.4 years ($p < 0.05$). Both statistically significant ($p < 0.05$), the sample was primarily males (58.5%) and primarily females (41.5%). Demographic differences were significant ($p < 0.01$): urban residents comprised 67.8%, rural residents 32.2%.

Hypertension is observed at a high prevalence of 74%, with a statistically significant result ($p < 0.001$), suggesting a strong, or difference association in the studied context. Notable findings with highly significant p values (<0.001) are mean systolic blood pressure of 142.7 mmHg (±16.5) and mean diastolic blood pressure of 89.3 mmHg (±12.2). A significant result ($p < 0.05$) for obesity, 62% of the population studied, indicates its importance of the dataset. Crude proportions of central obesity were 71.2% (Statistically significant, $p < 0.01$).

It also demonstrates that the population studied has significant cardiovascular and metabolic health risks. Total cholesterol is elevated in 65% of subjects, and LDL cholesterol in 73% ($p < 0.001$ in both cases). Statistically significant ($p < 0.05$) low HDL cholesterol is found in 60% of men and 50% of women. Diabetes prevalence is higher at 45% with a very significant result ($p < 0.001$). Furthermore, 64% of individuals exhibit elevated CRP levels, a highly significant p value (<0.001), indicating wide spread inflammation and related risks. These results highlight important concerns for cardiovascular and metabolic health in the population.

The data shows who in the population has key lifestyle related health risks. Of individuals, 75% have high sodium intake, which was statistically significant ($p < 0.05$), and 58% had high saturated fat intake, which was also statistically significant ($p < 0.05$). 48% of the population is sedentary ($p < 0.01$). Smoking behavior stands out with 34% current smokers ($p < 0.001$) and 28% former smokers ($p < 0.05$). These findings suggest the importance of dietary habits, physical inactivity and smoking on public health outcomes.

Table 1: Demographic Findings

Variable	Value	p-Value
Sample Size	200	-
Mean Age (years)	55.3 ± 10.4	<0.05
Male (%)	58.5%	<0.05
Female (%)	41.5%	<0.05
Urban (%)	67.8%	<0.01
Rural (%)	32.2%	<0.01

Table 2: Clinical Findings

Variable	Value	p-Value
Hypertension Prevalence (%)	74%	<0.001
Mean Systolic BP (mmHg)	142.7 ± 16.5	<0.001
Mean Diastolic BP (mmHg)	89.3 ± 12.2	<0.001
Obesity Prevalence (%)	62%	<0.05
Central Obesity (%)	71.2%	<0.01

Table 3: Biochemical Findings

Variable	Value	p-Value
Elevated Total Cholesterol (%)	65%	<0.001
Elevated LDL (%)	73%	<0.001
Low HDL (%)	60% (men), 50% (women)	<0.05
Diabetes Prevalence (%)	45%	<0.001
Elevated hs-CRP (%)	64%	<0.001

Table 4. Lifestyle and Behavioral Findings

Variable	Value	p-Value
High Sodium Intake (%)	75%	<0.05
High Saturated Fat Intake (%)	58%	<0.05
Sedentary Lifestyle (%)	48%	<0.01
Active Smokers (%)	34%	<0.001
Former Smokers (%)	28%	<0.05

DISCUSSION

This study highlighted the profound role of hypertension and obesity in the pathogenesis and progression of ischemic heart disease (IHD). The results show how these two big risk factors work together and combine with the increasing heart disease burden[11]. Based on clinical, biochemical and behavioural variables, this research examines the relationship between hypertension, obesity and IHD. We confirm that hypertension remains a major risk factor for IHD development in the studied population, especially in 74% participants. The direct hemodynamic burden of cardiac system is highly significant for systolic (142.7 ± 16.5 mmHg) and diastolic (89.3 ± 12.2 mmHg) blood pressures ($p < 0.001$). Chronic hypertension increases arterial shear stress, oxidative stress and pro inflammatory pathways, and leads to

endothelial dysfunction and acceleration of atherosclerosis[12]. It also exacerbates arterial stiffness and myocardial oxygen demand, and complicates ischemia, and the activation of the renin angiotensin aldosterone system (RAAS).

This association between hypertension and IHD is in agreement with previous observations, as, for example, that from the Framingham Heart Study which has previously described a dose related association of blood pressure and cardiovascular risk[13]. Hypertension is an independent risk factor in this cohort, and also acts to amplify the effect of other comorbidities such as obesity and diabetes. Obesity contributed significantly to IHD pathogenesis in the study population with a prevalence of 62%. Central obesity also was even more strongly associated with cardiovascular risk ($p < 0.01$) in 71.2% of the participants. It is well known that central adiposity is an exacerbating agent for cardiovascular damage via increases in sympathetic hyperactivity, RAAS activation and renal sodium retention. Together, these mechanisms synergistically increase blood pressure and promote disease progression in IHD[14,15].

In this study, obesity is also linked to metabolic dysregulation with high LDL cholesterol (73%) and total cholesterol (65%). Elevated CRP levels (64%) in adipose tissue also contribute to endothelial dysfunction and atherogenesis in the proinflammatory milieu of adipose tissue. The findings are consistent with global epidemiological trends and are consistent with obesity increasing cardiovascular risk[16]. In the vast majority of participants, hypertension and obesity occur in common, and this is a major problem. Together, these intertwined pathophysiological pathways were synergistic and increased cardiovascular risk. For instance, obesity worsens hypertension by means including enhanced sympathetic drive, impaired renal pressure natriuretic, and increased activity of the RAAS[17].

However, hypertension potentiates the vascular complications of obesity, and initiates a vicious cycle that predisposes to earlier onset and more severe IHD. The findings follow previous studies that have shown that obesity accounts for at least 75 percent of cases of hypertension. This strong interdependence emphasizes the importance of simultaneous therapeutic approaches to the two conditions to minimize adverse effects on cardiovascular health[18]. Cardiovascular outcome is also determined by clinical and biochemical markers, but also by lifestyle behaviors. High sodium intake (75%), high saturated fat consumption (58%) and a sedentary lifestyle (48%) were potential IHD risk contributors. The importance of population level interventions to reduce dietary and physical activity patterns is highlighted by these findings[19].

The Smoking, active (34%) and former (28%) was another critical behavioral factor. Dleterious effects of smoking on vascular health are well established, including promotion of oxidative stress, endothelial dysfunction and thrombogenesis. Smoking cessation could have a significant impact on reduction of the burden of IHD in populations comparable to those intended for public health interventions[20, 21]. This study adds to the pressing need for multifaceted approaches to combat the coexistence of hypertension and obesity. Early detection and clinical management under control with lifestyle intervention, pharmacotherapy and patient education of these conditions must be prioritized. Diet, physical activity, and behavioral factor integrated care models combined with medical treatment may decrease incidence and progression of IHD[22].

From the public health perspective, community based programs are needed to reduce the prevalence of obesity and hypertension. Policymakers instead should be supporting environments that promote healthy eating, physical activity and smoking cessation. Educational campaigns highlighting the risks of high sodium and saturated fat intake could add to these additional preventive efforts[23]. The advances in genomics and precision medicine should lead to development of personalized approaches to the management of obesity and hypertension. Further work is required to understand the long term impact of combined risk factor management on cardiovascular outcome. Additionally, knowledge of the socioeconomic determinants of hypertension and obesity may yield clues as to whom to target such interventions in high risk populations[7, 23].

Limitations: However, there are many limitations to this study. The cross sectional methodology used makes it unable to establish causal link between risk variables and IHD outcomes. Second, memory bias may be induced by the dependent variables (such as smoking and eating habits) being reported by the subjects themselves. Finally, the results can't be applied to larger populations as they were using non probability sampling. However, the study provides valuable knowledge about the interaction between obesity, hypertension, and IHD, and provides a basis for further research.

CONCLUSION

The conclusion of this study was that obesity and hypertension are important factors in etiology and development of IHD. The substantial correlations found highlight the need for combined clinical and public health approaches to treating these interconnected illnesses. Cardiovascular risk behavioral and underlying processes can be targeted to improve population health outcomes and decrease the rising burden of IHD. These objectives need to be accomplished and the effects of cardiovascular diseases have to be diminished around the globe, and further research as well as policy initiatives are necessary.

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