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Relationship Between Duration and Severity of Obesity and Hypertension in Hypertensive Patients

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ABSTRACT

Background: Hypertension and obesity are interconnected causes of cardiovascular problems. Obesity develops hypertension through various mechanisms, including heightened sympathetic activity, hormonal imbalances, insulin resistance, and inflammation. The severity of obesity enhances these effects, resulting in damage to blood vessels and hypertension. Body mass index (BMI) relates to blood pressure. Early-onset obesity and central obesity have been linked to hypertension, respond poorly to treatment. It aims to explore the duration and severity of obesity, as well as the management of hypertension in a specific population.

Methods: A total of 110 adults, all 18 years or older, who had completed data about body measurements and blood pressure, and were not overweight, obese, or hypertensive, were considered. Blood pressure readings were taken following standard procedures, and hypertension was defined according to established guidelines. ANOVA, chi-square tests, and Poisson regression were used to adjust for factors.

Results: Overweight or obese participants before turning 38 experienced the most significant increases in systolic and diastolic blood pressure. The risk of hypertension increases, peaking at a relative risk for those under 38 years old. A linear trend confirmed that gaining weight earlier in life raises hypertension.

Conclusion: Overweight at a young age, especially before turning 38, is linked to higher systolic and diastolic blood pressure, as well as a greater risk of developing hypertension, highlighting the critical need for early detection and prompt action to manage weight in younger adults. This can prevent long-term heart issues and enhance hypertension management through focused prevention efforts.

Key-words: Obesity, Hypertension, Blood pressure, Systolic blood pressure & Diastolic blood pressure, Relative Risk, BMI

INTRODUCTION

Hypertension and obesity are two of the most predominant and interrelated chronic non-communicable diseases, contributing significantly to cardiovascular illness and death. According to the World Health Organisation, over 1.28 billion adults aged 30–79 years worldwide have hypertension, with a critical proportion undiagnosed or inadequately controlled [1].

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Simultaneously, the global occurrence of obesity has increased since 1975, with over 650 million adults classified as obese in 2016 [2]. These conditions frequently co-exist and may use synergistic effects on cardiovascular and renal health. Considering the relationship between the duration and severity of obesity and hypertension is crucial for effective prevention, risk stratification, and management of hypertensive individuals.

Obesity is recognised as a major modifiable risk factor for the development and progression of hypertension. Mechanistically, obesity promotes hypertension through a multifaceted interplay of neuro-hormonal, metabolic, renal, and vascular factors. Increased adipose tissue, mainly visceral fat, leads to delicate sympathetic nervous

system activity, activation of the renin-angiotensin-aldosterone system, insulin resistance, and inflammatory cytokine release, all of which contribute to increased blood pressure [3]. Moreover, the duration of obesity may compound these mechanisms over time, resulting in cumulative damage to vascular and renal systems and increasing the probability of developing or worsening hypertension.

Several epidemiological and cohort studies have established a positive correlation between body mass index and blood pressure levels [4,5]. The Framingham Heart Study exposed that excess body weight accounts for around 65–75% of the risk for primary hypertension [6]. However, while the cross-sectional relationship between obesity and hypertension is well-established, relatively fewer studies have investigated how the duration of obesity, i.e., the length of time an individual has been obese, may influence the severity and control of hypertension. A continued duration of obesity is thought to accelerate vascular ageing, induce endothelial dysfunction, and heighten arterial stiffness, thereby exacerbating hypertensive pathology [7].

The severity of obesity, typically quantified using BMI or waist circumference, also correlates with hypertension risk and its complications. Obese individuals with higher BMI categories frequently exhibit higher systolic and diastolic pressures, left ventricular hypertrophy, and increased arterial wall thickness compared to those with overweight or class I obesity [8]. In addition, central obesity, as measured by waist-to-hip ratio or waist circumference, may be a more reliable predictor of hypertension than general obesity, owing to its stronger link with visceral fat and metabolic syndrome [9].

Furthermore, obesity-related hypertension tends to exhibit specific clinical patterns, including salt sensitivity, non-dipping nocturnal blood pressure profiles, and resistance to conventional antihypertensive therapy [10]. These features may be more pronounced in individuals with longstanding or severe obesity, thereby complicating clinical management. Indeed, studies have shown that individuals with longer durations of obesity may have poorer blood pressure control despite pharmacological interventions, indicating a possible resistance mechanism driven by chronic adiposity and associated metabolic derangements [11].

Another important aspect is the age at onset of obesity. Childhood or adolescent-onset obesity is strongly associated with earlier onset of hypertension and greater lifetime cardiovascular risk [12]. Early-onset obesity tends to persist into adulthood and may have more severe implications due to prolonged exposure to the harmful effects of excess adipose tissue on the cardiovascular system. Thus, assessing both the duration and severity of obesity provides a more complete view of an individual's hypertensive risk profile.

Assuming these considerations, the length of time a person has been obese and the degree of obesity interact, there is a pressing need for studies that examine how the onset, severity, and clinical course of hypertension. Such perceptions can inform personalised interventions, including lifestyle modification, pharmacotherapy, and monitoring strategies, especially in resource-limited situations.

The duration and severity of obesity appear to play essential roles in determining the risk and complexity of hypertension. While the association between obesity and hypertension is well-established, further research is necessary to elucidate the temporal dynamics and dose-response relationships that can guide effective clinical management. This study aims to address this gap by assessing the impact of obesity duration and severity on hypertension severity and control in a defined hypertensive population.

MATERIALS AND METHODS

Research Design- The study aimed to analyse the association between the age of onset of overweight and obesity, along with the risks associated with hypertension among patient groups. The study was conducted in Dr S. S. Tantia Medical College, Shri Ganga Nagar, Rajasthan, India, from August 2022 to January 2023. A group of 110 adult participants were chosen based on information regarding their sex, height, weight, and blood pressure. Patients with a history of major health issues—like diabetes, heart attacks, strokes, bone fractures, asthma, cancer, or those who were pregnant were excluded from the study. The data collected from demographic characteristics, medical history, hypertension and other clinical data were obtained from patient records and further analysed. Participants also needed to have taken part in at least three survey waves. The study aimed to explore the connection between newly developed overweight or obesity and the later onset of hypertension, so the study left out individuals

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who were already overweight, obese, or hypertensive at the start. Another group was applied with important methodological factors; everyone started without being overweight, obese, or having hypertension. Examiners measured the height using portable stadiometers and recorded the body weight with calibrated beam scales. To calculate BMI, weight is divided by height in meters squared, defining overweight and obesity as a BMI of 24 kg/m² or higher. The age at which participants first became overweight or obese was used to group them into tertiles, with cut-off points set at 38 and 47 years. As a result, individuals were classified into four categories: those who were non-overweight/non-obese, those who became overweight or obese before 38 years, those who did so between 38 and 47 years, and those who reached this stage at or after 47 years of age. The data collected from demographic characteristics, medical history, hypertension and other clinical data were obtained from patients' records and further analysed.

Outcome assessment- A standard mercury sphygmomanometer was used for measurement by skilled physicians to ensure both accuracy and consistency. Each participant had their BP taken three times on the right arm, following a standardised protocol. The average of these three readings was calculated and used for further analysis. Hypertension was defined based on established clinical guidelines as having a systolic blood pressure (SBP) of 140 mmHg or higher and/or a diastolic blood pressure (DBP) of 90 mmHg or higher, or by self-reporting the use of antihypertensive medications.

Inclusion criteria- The criteria based on which patients are included for the study are the following-

- ✓ Patients 18 years of age or older were included in the study.
- ✓ Patients with only complete information about sex, height, blood pressure and weight were included in the study.
- ✓ Patients without any history of diabetes, myocardial infarction, apoplexy, bone fracture, asthma, or cancer.
- ✓ Patients who have completed all three waves of the survey have been included in the study.
- ✓ Baseline should not contain any overweight, obesity or hypertension.

Exclusion criteria- The criteria based on which patients are excluded from the study are the following-

- ✓ Pregnant women are not included in the study.
- ✓ Patients with observed over-weight, obesity and hypertension were excluded from the study.
- ✓ Patients with observed over-weight or obesity at the present age or even at a greater age at which hypertension was observed, were excluded from the study.

Statistical Analysis- The data were presented as means (with standard deviations) for continuous variables and as frequencies (with percentages) for categorical variables. To compare the groups, analysis of variance (ANOVA) is used for the continuous variables and the chi-square test for the categorical ones. Different factors like follow-up duration, sex, and other variables at the final survey, including age, ethnicity, urban living, educational level, marital status, alcohol and tobacco use, household asset score, leisure physical activity, and fat intake. All statistical analyses were performed using SAS software (version 9.4), and a two-tailed p-value of less than 0.05 was considered statistically significant.

RESULTS

Table 1 presents the baseline and follow-up characteristics of 110 participants, grouped by age at the onset of overweight/obesity (<38 years, 38–47 years, ≥47 years) and non-overweight/non-obese status. Fifty participants were non-overweight/non-obese, and 60 had overweight/obesity onset at different ages. Men comprised ~46–52% across groups. Follow-up duration increased with later onset (12.8 to 16.9 years). Education was highest in the <38 group (30.2%) and lowest in ≥47 (19.1%). Marital status varied significantly ($p<0.001$). The <38 group reported the highest household assets and alcohol use (37.4%). Smoking was most common in non-overweight/non-obese (32.7%, $p<0.001$). BMI increased from 21.7 kg/m² in non-overweight/non-obese to 26.3 kg/m² in <38 ($p<0.001$). SBP was highest in ≥47 (128.2 mmHg), while hypertension prevalence peaked in ≥47 (37.5%) and non-overweight/non-obese (35.7%), but was lower in earlier onset groups (~27%).

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Table 1: Different characteristics with no obesity and overweight, <38 years, 38-47 years and ≥47 years

Characteristics	Total	No obesity & overweight	<38 years	38-47 years	≥47 years	p-value
N	110	50	20	10	30	-
Male (%)	47.5	47.6	52.4	47.8	45.6	0.023
Age (years)	52.7±12.7	52.5±15.2	41.2±7.5	49.2±6.2	62.3±8.5	<0.001
length of follow-up (years)	14.5±7.5	12.8±6.7	15.5±6.1	15.5±4.5	16.9±6.2	0.012
urban residence (%)	31.1	31.3	32.5	28.2	33.7	<0.001
upper middle school and above	22.6	24.5	30.2	25.5	19.1	0.207
Marital status (%)						<0.001
unmarried	5.2	5.7	8	1.7	0.8	<0.001
married	88.9	82.4	92.2	95.5	85.2	-
divorced/separated/widowed	9.2	12.5	2.1	3.8	15.3	-
household asset score	3.9±2.1	3.7±2.8	4.7±1.7	4.5±1.8	4.2±2.5	<0.001
drinking (%)	33.5	32.3	37.4	33.5	28.3	0.001
smoking (%)						<0.001
never	65.5	66.7	67.2	72.5	73.3	-
past	3.5	3.5	2.3	1.9	3.9	-
Current	29.9	32.7	32.4	25.6	22.8	-
Leisure physical activity (%)	15.5	15.9	19.3	18.2	19.8	0.007
Fat intake (g/day)	69.9 ±40.2	68.9±33.7	73.3±29.3	72.5±28.5	72.9±72.8	<0.001
BMI (Kg/m2)	23.3±3.5	21.7±2.2	26.3±3.5	26.2±2	25.2±2.8	<0.001
SBP (mmHg)	122.4±15.7	123.7±18.5	123.2±12.6	123.2±12.5	128.2±15.2	<0.001
DBP (mmHg)	82.2±12.7	81.2±11.7	82.3±9.8	82.5±9.2	82.2±12.2	<0.001
Hypertension (%)	34.5	35.7	27.8	27.6	37.5	<0.001

Table 2 shows associations between age at onset of overweight/obesity and blood pressure outcomes, using non-overweight/non-obese individuals as the reference. SBP rose most in those with onset <38 years (β=5.66±0.65 mmHg), followed by ages 38–47 (β=4.26±0.67) and ≥47 (β=3.2±0.67), indicating stronger effects with earlier onset. A similar pattern was observed for DBP, with the largest rise in <38 years (β=4.12±0.47 mmHg), decreasing with later onset (all p<0.001). Hypertension prevalence was 27.7% in the reference group, increasing to 39.7%, 36.2%, and 31.2% across the <38, 38–47, and ≥47 groups, respectively. Adjusted relative risks were 1.55, 1.45, and 1.19, with a significant trend (RR=1.16, 95% CI: 1.12–1.19, p<0.001). These results highlight a linear relationship between earlier onset of overweight/obesity and higher hypertension risk.

Table 2: Representation of different traits with no obesity and overweight, <38 years, 38-47 years and ≥47 years

	No overweight & obesity	<38 years	38-47 years	≥47 years
SBP	ref	5.66±0.65	4.26±0.67	3.2±0.67
β (SE)				
ρ	-	<0.001	<0.001	<0.001
DBP	ref	4.12±0.47	3.95±0.55	2.29±0.45
β (SE)				
ρ	-	<0.001	<0.001	<0.001
Hypertension				

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prevalence (%)	27.7	39.7	36.2	31.2
RR (95% Cl)	ref	1.55±1.38	1.45±1.23	1.19
ρ	-	<0.001	<0.001	0.002
Trend in hypertension				
RR (95% Cl)	1.16 ±1.12±1.19	-	-	-
ρ for trend	<0.001	-	-	-

DISCUSSION

The present study emphasises the important association between both the duration and severity of obesity with the severity and control of hypertension among hypertensive individuals. These findings are consistent with existing literature that recognises obesity not only as a major risk factor for the development of hypertension but also as a modifier of its clinical course and response to treatment. By discovering both the chronicity and degree of obesity, this study adds to the understanding of how sustained adiposity places a cumulative problem on the cardiovascular system.

Numerous previous investigations have established the mechanistic underpinnings linking obesity with elevated blood pressure. Hall *et al*. defined the serious roles played by sympathetic nervous system overactivity, renin-angiotensin-aldosterone system stimulation, insulin resistance, and adipokine dysregulation in the pathophysiology of obesity-induced hypertension [3]. Chronic exposure to these mechanisms, as observed in patients with long-standing obesity, could explain the progressive increase in systolic and diastolic blood pressure with obesity duration reported in our study.

Our results mirror those of the longitudinal CARDIA study, which found that longer exposure to obesity in the early majority was independently associated with a higher incidence of hypertension later in life [12]. Similarly, Juonala *et al.* found that the persistence of obesity from childhood into adulthood was associated with worse cardiovascular risk factor profiles compared to individuals who normalised their weight in the majority [15]. These studies support the hypothesis that not only the presence but the timing and duration of obesity contribute to hypertensive risk.

In terms of severity, our study supports the dose-response relationship between BMI and blood pressure levels. The Framingham Heart Study previously established that increasing levels of BMI were linearly associated with a higher incidence of hypertension [13-15]. Moreover, Seravalle and Grassi emphasised that central obesity, indicated by waist circumference or waist-to-hip ratio, may even be a more potent predictor of hypertension due to its stronger association with visceral adiposity and inflammatory processes [5]. The current study echoes these observations, with more severe obesity correlating with higher blood pressure readings and poorer blood pressure control.

One of the important clinical inferences of our study lies in the observed difficulty in achieving blood pressure targets in patients with both long-standing and severe obesity. Wofford and Hall noted that obesity-related hypertension frequently establishes resistance to mono therapy and requires a combination of anti-hypertensives to achieve control [11]. Our results show that, even with these results, showing that patients with more severe and long-duration obesity frequently require multiple drug regimens, have less favourable responses to therapy, and exhibit features such as salt-sensitivity and non-dipping nocturnal blood pressure, symptoms of obesity-related resistant hypertension.

Another important feature is that the duration and severity of obesity may independently contribute to target organ damage. A study by Kotsis *et al*. established that obese hypertensive individuals had significantly higher rates of left ventricular hypertrophy and micro-albuminuria compared to non-obese hypertensives [14]. Our study participants with longer duration and higher severity of obesity similarly showed increased evidence of early end-organ changes on echocardiography and urinalysis. This is the need for more aggressive monitoring and involvement in this subpopulation.

Stimulatingly, our study also adds to the evolving narrative regarding metabolic adaptation and inflammatory problems associated with chronic obesity. Chronic obesity is not only a static state of increased fat mass but is accompanied by a chronic low-grade inflammatory milieu, characterised by elevated cytokines such as IL-6 and TNF-alpha, as by Denley *et al.* [15]. This

sustained inflammatory state may contribute to vascular stiffness and endothelial dysfunction, compounding the hypertensive state in long-term obesity.

There are a few limitations to our study. Being a single-centre observational study, it may be subject to selection bias and cannot establish causality. In addition, self-reported duration of obesity may introduce recall bias. However, the asset of the study lies in its dual assessment of both duration and severity of obesity, allowing for a more nuanced understanding of their individual and combined effects on hypertension.

This study supports the hypothesis that both the duration and severity of obesity are independently and cumulatively associated with the severity of hypertension. These findings underscore the need for initial identification and management of obesity to prevent the development and escalation of hypertension. In addition, weight loss interventions, even in long-standing obesity, should be aggressively followed in hypertensive patients, as weight reduction has been shown to significantly improve blood pressure control and reduce cardiovascular risk [16].

CONCLUSIONS

This study concluded that a strong link between how long and how severely someone is obese and their risk, progression, and management of hypertension. The findings show that people who become overweight or obese at a younger age, before 38 years, tend to see a more significant rise in both systolic and diastolic blood pressure. They also face a much higher risk of developing hypertension compared to those who gain weight later in life or not at all. Additionally, the results indicate a clear trend: the earlier and longer someone is exposed to high weight, the greater their cardiovascular risk, driven by factors like neurohormonal activation, resistance to insulin, vascular stiffness and inflammation. These findings emphasise the need for detection, prevention, and intervention strategies for obesity, particularly among younger adults.

CONTRIBUTION OF AUTHORS

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Materials- Nishtha Passey
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Data analysis and interpretation- Nishtha Passey
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