The Relationship between Body Mass Index (BMI) And Hypertension Incidence at Royal Prima Hospital in 2024

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Abstract.

Background: Hypertension affects 1.4 billion adults globally and remains inadequately controlled. Body mass index (BMI) accounts for 65 to 75 percent of primary hypertension cases, yet hospital-based evidence from Southeast Asian populations remains limited. Objective: This study determined the relationship between BMI and hypertension incidence at Royal Prima Hospital Medan in 2024. Methods: This cross-sectional analytical study employed total population sampling including 69 respondents meeting inclusion criteria. Data were extracted from medical records and analyzed using univariate analysis and Spearman rank correlation test. Results: Among respondents, 84.1 percent experienced hypertension, with 52.2 percent possessing a BMI greater than 25 kg/m². All respondents with BMI greater than 25 kg/m² exhibited hypertension compared to 66.6 percent of those with normal BMI (p=0.000). Conclusion: A statistically significant relationship exists between elevated BMI and hypertension incidence, confirming obesity as a critical modifiable risk factor for hypertension development in hospital settings. Weight management through lifestyle modification represents an essential intervention strategy for hypertension prevention and control.

Keywords: *Blood Pressure; Body Mass Index; Cardiovascular Risk; Hypertension and Obesity.*

I. INTRODUCTION

Hypertension represents a critical global public health challenge, affecting approximately 1.4 billion adults aged 30 to 79 years worldwide, constituting 33% of this population segment. The disease burden associated with elevated blood pressure has escalated dramatically, with attributable deaths increasing from 6.8 million in 1990 to 10.8 million in 2019. Despite being both preventable and treatable, hypertension remains inadequately controlled, with only 23% of affected individuals achieving blood pressure targets globally. This silent epidemic disproportionately affects low- and middle-income countries, where two-thirds of hypertensive cases are concentrated, largely due to increasing cardiovascular risk factors in recent decades. The economic implications are equally alarming, with hypertension accounting for approximately 10% of global healthcare expenditure and projected to cost low- and middle-income countries approximately US$ 3.7 trillion between 2011 and 2025. In Indonesia specifically, the prevalence of hypertension among adults aged 18 years and above reached 34.1% according to the 2018 Basic Health Survey, representing a substantial increase from 25.8% in 2013.The obesity epidemic represents a parallel and interconnected global health crisis, with rates more than doubling since 1990, affecting 16% of adults worldwide in 2022. Projections indicate that by 2035, global overweight and obesity prevalence will reach 51%, with the economic burden potentially exceeding US$ 3 trillion. Body mass index (BMI), a simple yet widely used anthropometric indicator calculated as weight in kilograms divided by height in meters squared, serves as the primary screening tool for assessing body composition and obesity status.

The relationship between elevated BMI and hypertension has been extensively documented, with obesity accounting for 65% to 75% of primary hypertension risk. Individuals with overweight or obesity demonstrate a two to six-fold increased risk of developing hypertension compared to those with normal body weight. In Southeast Asia, approximately 25% of the population is affected by hypertension, with Indonesia recording prevalence rates that surpass neighboring countries such as Bangladesh, Korea, and Thailand. The intersection of rising obesity rates and hypertension prevalence in productive age populations poses significant implications for demographic dividend realization and economic productivity in developing nations.The pathophysiological mechanisms linking obesity to hypertension are multifaceted and complex,

involving sympathetic nervous system (SNS) overactivation, renin-angiotensin-aldosterone system (RAAS) stimulation, insulin resistance, endothelial dysfunction, and renal sodium retention. Excessive adipose tissue, particularly visceral fat, produces adipokines and pro-inflammatory cytokines that contribute to vascular dysfunction and increased peripheral resistance. Physical compression of renal structures by perirenal and intrarenal fat accumulation leads to activation of the RAAS and altered pressure natriuresis, shifting blood pressure regulation toward hypertensive levels.

The adipocyte-derived hormone leptin plays a particularly important role in obesity-induced hypertension by selectively increasing renal sympathetic nerve activity while paradoxically demonstrating resistance in metabolic regulation. Furthermore, perivascular adipose tissue in obesity transitions from producing protective nitric oxide to secreting pro-inflammatory mediators that enhance vasoconstriction and endothelial dysfunction. Recent evidence from the Global Burden of Disease Study 2021 demonstrates that high BMI has risen from the fourth to the second position among risk factors for hypertensive heart disease, with population attributable fractions for mortality increasing from 33% in 1990 to 44% in 2021.Despite substantial evidence establishing the association between BMI and hypertension in high-income Western populations, significant research gaps persist regarding this relationship in hospital settings within Southeast Asian contexts, particularly Indonesia. Previous Indonesian studies have predominantly utilized community-based samples or older datasets from 2014 to 2015, with limited representation of hospital-based populations where disease severity and comorbidities may differ substantially. Furthermore, most existing cross-sectional investigations have focused on elderly populations or specific demographic subgroups, leaving the broader adult population across diverse age ranges underexplored. The heterogeneity of ethnic backgrounds, dietary patterns, genetic predispositions, and healthcare access in Indonesia necessitates population-specific research to inform locally relevant prevention and intervention strategies.

Additionally, while global studies have examined BMI-hypertension associations using various classification systems, inconsistencies in BMI categorization specific to Asian populations (using WHO Asia-Pacific criteria versus standard WHO classifications) contribute to methodological heterogeneity and limit direct comparisons.The present study aims to determine the relationship between BMI and hypertension incidence among patients at Royal Prima Hospital Medan in 2024, using a cross-sectional analytical approach with secondary medical record data. Specifically, this investigation seeks to: first, characterize the BMI distribution among hospital patients; second, determine the proportion of hypertensive patients across BMI categories; and third, analyze the statistical association between BMI and hypertension occurrence using appropriate correlation methods. This research addresses critical gaps by providing contemporary hospital-based evidence from an urban Indonesian setting, employing standardized WHO Asia-Pacific BMI classifications appropriate for Asian populations, and contributing to the limited body of knowledge regarding obesity-hypertension relationships in tertiary healthcare facilities within low- and middle-income countries. The findings will inform targeted interventions for hypertension prevention through weight management strategies in clinical settings, support evidence-based policy development for cardiovascular disease prevention in Indonesia, and provide baseline data for longitudinal investigations examining temporal trends in obesity-related hypertension. Given that weight management represents a modifiable risk factor with established efficacy in blood pressure reduction (5-20 mmHg systolic reduction per 10 kg weight loss), understanding the BMI-hypertension relationship in local hospital populations is essential for developing culturally appropriate and clinically effective intervention programs that can reduce the substantial morbidity, mortality, and economic burden associated with these interconnected epidemics.

II. METHODS

Research Design and Study Method

This research employed an analytical quantitative approach using a cross-sectional study design, which represents a form of observational (non-experimental) research. According to Sugiyono (2022), a cross-sectional design is a research method that examines the correlation between risk factors and their effects through observational approaches or simultaneous data collection at a single point in time, known as a point time approach. This design was selected as most appropriate for achieving the research objectives,

which aimed to determine the relationship between body mass index (BMI) and hypertension incidence at Royal Prima Hospital Medan during 2024. The analytical observational approach enabled researchers to examine causal relationships and associations between variables without introducing or assigning the study factor to participants, allowing observation of how exposure to elevated BMI results in hypertension outcomes. The cross-sectional design possesses several methodological advantages for this study, including relative ease of execution, cost-effectiveness, rapid data acquisition, capability to examine multiple variables simultaneously, and utilization of readily available populations from general hospital settings. Additionally, this design minimizes the risk of loss to follow-up, a common limitation in longitudinal investigations.

Study Population and Sampling Method

The study population comprised all patients registered at Royal Prima Hospital Medan, Medan City, North Sumatra Province, with the study conducted at Jl. Father No. 68A, Sei Putih, Kec. Medan Petisah, Medan, North Sumatra, Postal Code 20118, during 2024. The sampling technique employed total population sampling (total enumeration sampling), a type of purposive sampling method in which all members of the target population meeting predetermined criteria were included in the study. Total population sampling represents a non-probability sampling technique that involves examining the entire population possessing particular characteristics relevant to the research objectives. According to Sugiyono (2022) and methodological literature, this sampling approach becomes appropriate when the target population size is well-defined and sufficiently limited, thereby eliminating potential sampling bias and ensuring comprehensive representation of all population members. The total sample included 69 respondents meeting both inclusion and exclusion criteria. Inclusion criteria were established as follows: (1) patients with confirmed diagnosis of hypertension; (2) patients aged 18 years or older; and (3) possession of complete data regarding body weight and height measurements in medical records. Exclusion criteria encompassed: (1) patients with incomplete medical record data at Royal Prima Hospital Medan during 2024; and (2) patients with other chronic diseases affecting body weight, such as cancer or human immunodeficiency virus (HIV) infection.

Study Variables and Operational Definitions

The independent variable was body mass index (BMI), a categorical ordinal variable, operationally defined as the numerical result obtained by dividing body weight in kilograms by height in meters squared, subsequently categorized according to WHO Asia-Pacific classification criteria as follows: less than 18.5 kg/m² indicating underweight; 18.5 to 22.9 kg/m² indicating normal weight; 23.0 to 24.9 kg/m² indicating overweight; 25.0 to 29.9 kg/m² indicating obesity class I; and greater than or equal to 30.0 kg/m² indicating obesity class II. The dependent variable was hypertension incidence, a categorical nominal variable, operationally determined according to blood pressure measurements recorded in patient medical records, with classification as follows: normal blood pressure (less than 120/80 mmHg); prehypertension (120-139/80-89 mmHg); and hypertension (greater than or equal to 140/90 mmHg). Blood pressure classifications were based on clinical facility measurements, which represents the standard diagnostic approach in hypertension research[2.1.1].

Data Collection Methods and Analysis Procedures

Data utilized in this investigation constituted secondary data obtained from patient medical records at Royal Prima Hospital Medan, collected during the 2024 calendar year. According to current methodological standards, secondary use of electronic health records for research purposes represents a valid and efficient means of using existing data to answer research questions, particularly when data quality and completeness are assured. The data collection procedure involved submitting formal research authorization requests to Royal Prima Hospital Medan administrative personnel through the Faculty of Medicine at Prima Indonesia University. Secondary data sources were selected to access complete and accurate medical information without requiring direct patient contact, thus ensuring both research efficiency and patient privacy protection. Data extraction encompasses patient demographics, body weight measurements, height measurements, and blood pressure readings recorded during clinical assessments. All data were processed and analyzed using SPSS version 25 (Statistical Package for the Social Sciences), a statistical computing software application designed for quantitative data analysis.Data analysis consisted of two sequential

components. Univariate analysis was initially conducted to characterize the frequency distribution of hypertension classification among study respondents and to describe the distribution of BMI values across the sample, presented in frequency and percentage tables.

Univariate analysis, according to descriptive statistical methodology, involves calculating and summarizing the basic features of individual variables to provide a comprehensive understanding of data characteristics, using measures of central tendency including mean, median, and mode, and measures of dispersion including range and standard deviation. Subsequently, bivariate analysis was performed to examine the association between BMI and hypertension incidence. The Spearman rank correlation test was selected as the appropriate statistical procedure for this analysis. The Spearman correlation test is applied when the relationship between two variables is not linear, when bivariate normal distribution is absent, or when data are collected on measurement scales not truly interval in nature. This nonparametric test calculates the correlation coefficient (rho) by determining the difference between ranks of corresponding variables and is particularly suitable for assessing monotonic relationships where increases in one variable magnitude accompany increases or decreases in the other variable magnitude. Statistical significance was established at the conventional alpha level of p less than 0.05, with p-value 0.000 indicating highly significant association between variables.

Research Procedures and Ethical Considerations

The research procedure followed a systematic sequence incorporating IRB approval requirements and institutional compliance standards. Initially, formal research proposals were submitted to the Research Ethics Committee and institutional administration at Royal Prima Hospital Medan to obtain authorization for accessing medical records and conducting the study. Subsequently, medical records meeting inclusion and exclusion criteria were systematically reviewed to extract pertinent data including age, sex, BMI classification, and blood pressure measurements. All extracted data were coded numerically to ensure patient confidentiality and maintain anonymity throughout the analytical process. Data quality verification procedures were implemented to verify completeness and accuracy of extracted information, addressing potential missing values or data inconsistencies. The research adheres to fundamental ethical principles including informed consent principles (through institutional authorization to access existing records), beneficence (potential knowledge generation to improve hypertension prevention strategies), and non-maleficence (minimal risk associated with retrospective data analysis), in accordance with international research ethics guidelines. All procedures were conducted in strict compliance with institutional policies regarding secondary data utilization and patient privacy protection.

III. RESULT AND DISCUSSION

Results

Table1. Frequency distribution based on hypertension classification

Hypertension	Amount	Presentation
Normal	1	1.4%
Pre-hypertension	10	14.5%
Hypertension	58	84.1%
Total	**69**	**100.0**%

Based on table 1 above, it shows that the frequency based on medical record data observed by researchers based on hypertension classification groups at Royal Prima Hospital, Medan City for the normal group with a total of 1 respondent with a percentage of 1.4%, for the pre-hypertension group with a total of 10 respondents with a percentage of 14.5%, and for the hypertension group with a total of 58 respondents with a percentage of 84.1%.

Distribution Based on BMI

Table 2. Frequency distribution based on BMI

Grading	Amount	Presentation
18.5 – 24.9	33	47.8%
>25	36	52.2%
Total	**69**	**100.0**%

Based on table 2 above, it shows that the frequency based on medical record data observed by researchers based on BMI groups at Royal Prima Hospital, Medan City for the BMI 18.5 group with a total of 33 respondents with a percentage of 47.8%, and for the BMI >25 group with a total of 36 respondents with a percentage of 52.2%.

Bivariate Analysis

The Relationship Between Body Mass Index (BMI) and Hypertension

After collecting and processing data to determine the relationship between body mass index and hypertension, a bivariate test was conducted to determine the relationship using the Spearman test, resulting in the following data:

Table 3.The relationship between BMI and hypertension

BMI value	**Hypertension**						**Total**		***P*-value**
	Normal		Pre-hypertension		Hypertension				
	n	%	n	%	n	%	n	%	
18.5 – 24.9	1	3	10	30.3	22	66.6	33	100	0,000
>25	0	0	0	0	36	100	36	100	

Based on table 4 above, it shows that among the respondents included in the normal group with a BMI value of 18.5 - 24.9, there was 1 respondent (3%), and a BMI value >25 was 0 respondents (0%). Furthermore, respondents included in the pre-hypertension group with a BMI value of 18.5 - 24.9 were 10 respondents (30.3%), and a BMI value >25 was 0 respondents (0%). Furthermore, respondents included in the hypertension group with a BMI value of 18.5 - 24.9 were 22 respondents (66.6%), and a BMI value >25 was 36 respondents (100%). In the results of the statistical test, a p-value (0.000) was obtained, so it can be concluded that there is a relationship between hypertension and BMI values in patients at Royal Prima Hospital, Medan City.

Discussions

Body Mass Index and Hypertension: Evidence from Hospital-Based Analysis

The findings of this cross-sectional study demonstrate a statistically significant relationship between body mass index and the incidence of hypertension at Royal Prima Hospital Medan in 2024, with a p-value of 0.000 indicating strong association (p less than 0.05). Among the 69 study respondents, 84.1 percent experienced hypertension while 52.2 percent possessed BMI values exceeding 25 kg/m², which classifies them as overweight or obese according to WHO Asia-Pacific criteria. Notably, among respondents with BMI between 18.5 and 24.9 kg/m² (normal weight category), only 66.6 percent exhibited hypertension, whereas 100 percent of respondents with BMI greater than 25 kg/m² demonstrated hypertension. This progressive relationship between increasing BMI and hypertension prevalence aligns with established epidemiological evidence demonstrating that obesity accounts for approximately 65 to 75 percent of primary hypertension cases in industrialized countries. The hospital-based setting provides particular relevance to this finding, as patients presenting to tertiary healthcare facilities often represent more severe or poorly controlled disease presentations compared with community-dwelling populations, potentially reflecting the cumulative cardiovascular consequences of prolonged obesity.The pathophysiological mechanisms underlying the relationship between elevated BMI and hypertension are multifactorial and involve complex interactions among neuroendocrine, metabolic, and vascular systems. Insulin resistance, a cardinal metabolic consequence of obesity, occupies a central position in obesity-induced hypertension pathogenesis.

Recent prospective cohort studies have demonstrated that the triglyceride-glucose index, serving as a surrogate marker of insulin resistance, partially mediates the relationship between visceral adiposity and hypertension incidence, with insulin resistance specifically in adipose tissue emerging as a stronger predictor of hypertension development than hepatic or skeletal muscle insulin resistance. The mechanism by which insulin resistance contributes to blood pressure elevation encompasses antinatriuretic effects of insulin in renal tubules, augmented sympathetic nervous system responses to endogenous vasoconstrictors, altered vascular membrane cation transport, and impaired endothelium-dependent vasodilatation. Furthermore, hyperinsulinemia directly stimulates vascular smooth muscle cell growth, promoting arterial wall thickening and increased peripheral vascular resistance.Sympathetic nervous system activation represents another critical mechanism linking obesity to hypertension, with adipocyte-derived leptin serving as a primary

neurohumoral mediator of this relationship. Leptin, a circulating hormone that increases in proportion to total body fat mass, exhibits selective preservation of its renal sympathoexcitatory effects despite the development of metabolic leptin resistance in obesity. This selective sympathoactivation occurs in the renal circulation specifically, potentially explaining why metabolic leptin resistance does not abolish the pressor effects of elevated leptin in obesity.

Increased renal sympathetic nerve activity promotes direct vasoconstriction of resistance vessels and simultaneously enhances renal sodium reabsorption through direct tubular effects and augmented renin release. In conscious animal models, chronic leptin administration induces sustained blood pressure elevation through sympathetic activation even when systemic metabolic responses to leptin are blunted. Circulating catecholamine levels are increased in obese humans, and muscle sympathetic nerve activity is elevated by approximately 40 percent in obese compared with lean normotensive subjects. This sympathoactivation precedes and contributes independently to blood pressure elevation beyond the direct effects of increased body weight.The renin-angiotensin-aldosterone system undergoes significant activation in obesity, representing yet another interconnected mechanism promoting hypertension development. Adipose tissue itself constitutes an endocrine organ that produces renin, angiotensinogen, and expresses angiotensin-converting enzyme, angiotensin II receptors, and aldosterone synthase, enabling local production of the complete enzymatic cascade. Circulating levels of angiotensinogen, renin activity, angiotensin-converting enzyme activity, angiotensin II, and aldosterone are all elevated in obese individuals. The adipocyte-derived hormone leptin stimulates renin secretion and angiotensinogen production through activation of the sympathetic nervous system and direct effects on juxtaglomerular cells. Additionally, mechanical compression of renal structures by perirenal and intrarenal fat accumulation may reduce renal perfusion pressure, triggering baroreceptor-mediated RAAS activation and sodium retention.

Weight loss of just five percent produces meaningful reductions in circulating angiotensinogen, renin activity, aldosterone, and angiotensin-converting enzyme activity, with the magnitude of decrease in waist circumference serving as a superior predictor of RAAS suppression compared with weight loss per se. Concurrent with RAAS suppression, five percent weight loss produces approximately seven mmHg reduction in systolic ambulatory blood pressure. This weight-loss-responsive component of the RAAS in obesity has important therapeutic implications, suggesting that modest lifestyle modifications targeting weight reduction may produce meaningful blood pressure improvements through suppression of multiple mechanisms.Adipokines and inflammatory mediators produced by expanded adipose tissue contribute substantially to obesity-associated vascular dysfunction and hypertension development. Progressive adipocyte expansion and differentiation accompanied by reduced blood supply leads to adipocyte hypoxia, necrosis, and infiltration of classically activated macrophages, inducing a chronic low-grade inflammatory state within adipose tissue. This pro-inflammatory adipose tissue microenvironment produces increased quantities of leptin, visfatin, chemerin, and resistin while simultaneously reducing secretion of anti-inflammatory adipokines including adiponectin and omentin. Circulating leptin concentrations are significantly elevated in obesity and independently associated with blood pressure elevation through sympathetic activation as previously discussed. Visfatin, another pro-inflammatory adipokine, demonstrates positive correlation with visceral fat content and inflammation markers.

Omentin-1, an anti-inflammatory adipokine expressed primarily in visceral adipose tissue, exists at reduced concentrations in obesity and exerts protective effects through suppression of endothelial adhesion molecules, promotion of anti-inflammatory macrophage polarization, and inhibition of nuclear factor-kappa B signaling pathways. The shift from anti-inflammatory to pro-inflammatory adipokine profiles, along with increased secretion of pro-inflammatory cytokines including tumor necrosis factor-alpha, interleukin-6, and monocyte chemoattractant protein-1, creates a systemic inflammatory milieu that damages vascular endothelium and promotes atherosclerosis.Vascular endothelial dysfunction and oxidative stress constitute fundamental mechanisms through which obesity promotes hypertension and cardiovascular disease. Endothelial cells lining resistance vessels normally produce nitric oxide through endothelial nitric oxide synthase, maintaining vascular tone through vasodilation, reducing platelet aggregation, preventing leukocyte adhesion, and inhibiting smooth muscle proliferation. In obesity, chronic low-grade inflammation

and elevated oxidative stress reduces bioavailability of endothelium-derived nitric oxide through increased reactive oxygen species production that rapidly reacts with nitric oxide to form peroxynitrite, a highly reactive molecule that damages cellular proteins and perpetuates oxidative stress in a vicious cycle. Circulating lipid peroxidation byproducts such as 8-isoprostane are significantly elevated in obese individuals compared with lean controls and demonstrate positive correlation with body mass index. Simultaneously, circulating nitrite and nitrate concentrations, which serve as intermediates in the nitric oxide pathway and reservoirs for regeneration of bioavailable nitric oxide, are reduced in obesity. The result is impaired endothelium-dependent vasodilation, increased peripheral vascular resistance, and sustained hypertension despite the initial compensatory sympathetic activation. Endothelin-1, a potent endothelium-derived vasoconstrictor, is upregulated in obesity and demonstrates significant positive correlation with both body mass index and oxidative stress markers. The cumulative effect of reduced nitric oxide availability coupled with increased endothelin-1 production creates a pro-vasoconstrictor vascular environment.

Clinical Implications and Therapeutic Significance

The robust relationship between BMI and hypertension incidence documented in this hospital-based study carries important implications for hypertension prevention and management strategies in clinical practice. Weight loss represents one of the most effective non-pharmacological interventions for blood pressure reduction, with contemporary meta-analytic evidence demonstrating that clinical systolic and diastolic blood pressure reductions of 5.79 mmHg and 3.36 mmHg, respectively, occur following mean BMI reduction of 2.27 kg/m². More substantial weight loss produces proportionally greater blood pressure reductions, with systolic and diastolic blood pressure reductions of 6.65 mmHg and 3.63 mmHg observed following a mean BMI reduction of 4.12 kg/m². Importantly, blood pressure reductions are substantially more pronounced when BMI decreases by three or more kg/m² compared with smaller reductions. Systolic blood pressure reduction begins with modest weight loss of two to five percent of baseline body weight, while improvement in diastolic blood pressure requires five to ten percent weight reduction. Even in normotensive obese individuals, five percent weight loss produces measurable systolic blood pressure reduction, suggesting that weight management benefits extend beyond hypertensive populations. According to the 2025 American Heart Association and American College of Cardiology guideline, each kilogram of weight loss produces approximately one mmHg reduction in both systolic and diastolic blood pressure, with a target BMI of 20 to 24.9 kg/m² recommended for optimal cardiovascular outcomes.

Lifestyle-based weight reduction through dietary modification and regular physical activity remains the foundational approach for hypertension management in overweight and obese populations. The Dietary Approaches to Stop Hypertension diet, characterized by high consumption of fruits, vegetables, whole grains, and low-fat dairy products combined with reduced sodium intake, produces blood pressure reduction of approximately eleven mmHg systolic and three mmHg diastolic[2.1.6]. When combined with weight loss and sodium restriction, the DASH diet produces additive synergistic blood pressure-lowering effects. Aerobic exercise of 90 to 150 minutes weekly at 65 to 75 percent heart rate reserve produces blood pressure reductions of five to eight mmHg systolic and two to four mmHg diastolic[2.1.6]. For individuals in whom lifestyle interventions alone prove insufficient to achieve blood pressure targets, recent clinical guidelines now incorporate newer pharmacologic options including glucagon-like peptide-1 receptor agonists such as semaglutide, which simultaneously reduce body weight and blood pressure, and bariatric surgical procedures for individuals with severe obesity. The multimodal approach recognizing obesity as a major modifiable risk factor for hypertension, combined with targeted intervention addressing underlying pathophysiological mechanisms including insulin resistance, sympathetic activation, and RAAS dysfunction, represents the contemporary standard of care.

The cross-sectional design of this investigation provides important observational evidence of the BMI-hypertension relationship in a hospital-based Southeast Asian population but cannot establish causality or elucidate precise temporal relationships between BMI changes and hypertension development. Longitudinal investigations tracking BMI alterations and blood pressure changes over extended follow-up periods would provide stronger evidence regarding causal mechanisms. Additionally, this study examined BMI without measuring visceral fat content directly through imaging or assessing specific biomarkers of

insulin resistance, sympathetic activation, or RAAS function, limiting mechanistic insights. Future investigations incorporating imaging assessment of adipose tissue distribution, measurement of circulating adipokines and inflammatory markers, evaluation of sympathetic nerve activity, and assessment of RAAS components would substantially enhance understanding of the pathophysiological pathways linking obesity to hypertension in the study population. Nevertheless, the strongly significant association between BMI and hypertension demonstrated in this hospital-based sample provides compelling evidence supporting aggressive lifestyle modification targeting weight reduction as a cornerstone intervention for hypertension prevention and management in primary healthcare settings throughout Indonesia.

IV. CONCLUSION

This cross-sectional study successfully demonstrated a statistically significant relationship between body mass index and hypertension incidence at Royal Prima Hospital Medan, with all respondents possessing BMI values greater than 25 kg/m² exhibiting hypertension compared to 66.6 percent of those with normal BMI, supported by the p-value of 0.000. The findings underscore obesity as a critical modifiable risk factor for hypertension development and emphasize the urgent need for comprehensive weight management strategies in tertiary healthcare settings. The study confirms established epidemiological evidence that elevated BMI operates through multifactorial pathophysiological pathways including insulin resistance, sympathetic nervous system activation, renin-angiotensin-aldosterone system stimulation, adipokine dysregulation, and endothelial dysfunction to promote hypertension development. However, the cross-sectional design inherently limits causal inference and temporal relationship determination, preventing the establishment of whether BMI changes precede hypertension development or whether hypertension management influences weight trajectory. The absence of direct measurement of visceral adiposity, inflammatory biomarkers, or sympathetic nervous system function further constrains mechanistic understanding of the observed associations in this Indonesian population.

Future investigations should incorporate longitudinal designs tracking BMI trajectories and blood pressure changes over extended follow-up periods to elucidate temporal relationships and causal mechanisms. Research incorporating visceral fat imaging, circulating adipokine measurement, sympathetic nerve activity assessment, and renin-angiotensin-aldosterone system component evaluation would substantially enhance mechanistic understanding of obesity-induced hypertension in Southeast Asian populations. Implementation studies evaluating the effectiveness of integrated weight management and hypertension interventions within primary and secondary healthcare facilities throughout Indonesia are critically needed to inform evidence-based policy development and optimize cardiovascular disease prevention. Primary care providers should recognize and treat obesity as a chronic medical condition using evidence-based behavioral interventions, lifestyle modifications involving dietary approaches, regular physical activity, and pharmacological options to achieve meaningful weight reduction that translates to clinically significant blood pressure improvement and cardiovascular risk reduction in at-risk populations.

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