

The Consequences of Hypertension and Obesity on Coronary Heart Disease

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ABSTRACT

Background: Coronary heart disease (CHD) is the leading cause of death worldwide. At present, more than 20 million adults suffer from CHD, and the number keeps increasing. Hypertension and obesity, known risk factors of CHD, are also inclining in prevalence. Given the diversities of world population, studies on correlation of hypertension and obesity in the incidence of CHD still have a lot to unveil. **Aim:** to analyze the association between hypertension and obesity to CHD as both independent and dependent risk factors. **Method:** A cross-sectional descriptive study on 100 hypertensive subjects was conducted from March to November 2022. Subjects were selected by simple random sampling technique. The data were collected by anamnesis, physical examination, laboratory examination, electrocardiography, and by checking on medical records if needed. Statistical analysis was conducted using SPSS. **Result:** The mean age of the study samples was 58.64 years old. The mean value of BMI was 25.77 ± 5.21 SD, and 53.6% of the participants was obese. 33% of the subjects were diagnosed with CHD. There was no correlation between obesity and CHD (OR = 0.582, 95% CI = 0.245-1.382); between hypertension on therapy or without therapy and CHD (OR= 0.729, 95% CI= 0.15-15.09). There was also no correlation between length of hypertension and CHD (R= 0.117; p = 0.27). **Conclusion:** This study shows no significant correlations between hypertension, obesity, and CHD. Whether socioeconomic and demographic profile play a part in the association of hypertension and obesity with CHD, specifically for Indonesian ethnicities, should be studied further.

Keywords: Coronary Heart Disease, Cardiovascular Disease, High Blood Pressure, Body Mass Index, Indonesia.

INTRODUCTION

Coronary heart disease (CHD), also known as coronary artery disease (CAD), is one the major leading causes of death worldwide. It is the most common cardiovascular disease (CVD). According to the Centers for Disease Control and Prevention (CDC), around 20.1 million adults over 20 have CHD. The mortality rate of CHD in adults below 65 years old is 2 in 10 deaths.¹ Aside from the mortality burden, CHD also has some deleterious effects on individual performance and the global economy. CHD tends to reduce productivity in the working-age population due to absence from work and reduced efficiency at work due to short-term disability.² The loss of productivity can affect the economic stability at the individual, company or even global level. The cost of direct CVD-related medical treatment is already high, reaching 273 billion USD in the US in 2010, and 7.4 trillion Rupiah in Indonesia in 2016. The advanced therapeutic breakthrough in the past years has reduced the mortality rate and increased the life-span by ± 30 years.³ However, the burden of CVD, including CHD keeps increasing in low-income to middle-income countries, and so does the disability it causes.⁴

The increasing case of CHD is closely related to the increment of risk factors. CHD risk factors were first coined by the Framingham heart study (FHS) in 1957. It demonstrated the relationship between blood pressure, cholesterol level, and cigarette smoking to CAD. This concept is evolving, and

now we understand several risk factors grouped as modifiable and conventional. The modifiable risk factors include high blood pressure, high blood cholesterol levels, smoking, diabetes, overweight or obesity, lack of exercise, poor diet, and high-stress level. The conventional risk factors comprise age, sex, family history (genetics), and race or ethnicity.^{5,6}

Hypertension is a significant risk factor for CVD and mortality, including CHD. The prevalence of hypertension has been increasing over the past years. By 2021, the World Health Organization (WHO) has estimated that around 1.28 billion adults over 30 are suffering from hypertension.⁷ Hypertension accounts for 7.5 million deaths worldwide and 5.7 million lost disability-adjusted life years (DALYs).⁴ Being a modifiable risk factor, blood pressure control is hoped to reduce the mortality and disability rate of cardiovascular disease. However, the definite correlation of blood pressure as the risk factor for CHD, especially in anti-hypertensive treatment, is still unclear. A study suggests that controlling systolic and diastolic blood pressure is proven to prevent coronary events in patients without clinical CHD. The same study also highlights that systolic control in patients with clinical CHD is beneficial.⁸

Obesity is reported to directly contribute to CVD risk factors (i.e., dyslipidemia, diabetes, hypertension). It is also an independent risk for the development of CHD, and the CVD-related mortality. However, trials of medical weight loss have not reported a reduction in CHD rates.^{9,10} In addition, a few studies also found no difference of obese and non-obese individuals in the prevalence of CHD.¹¹

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Being the major risk factor of CVD that is also modified by obesity, hypertension consequences towards CHD events still need further study. Given the variety of associations between obesity and CHD, it is a field needing further understanding. In this study, we sought to analyze the association between hypertension and obesity to CHD as both independent and dependent risk factors.

METHODS

Study Population

The current study was carried out in DR. Soetomo Hospital in Surabaya, Indonesia. It was a cross-sectional descriptive study conducted from March 2022 to November 2022. The sampling was part of the Indonesia Left Ventricle Geometric Pattern on the Hypertensive Patient Registry frame. We required 41 male and 41 female subjects equally distributed according to their age group (18-25, 26-35, 36-45, 46-55, 56-65, and 66-75 years). To compensate for the possible poor response rate and missing data, we collected 100 subjects. Patients were stratified according to their age group. Then, we selected our sample in a simple random sampling technique.

Over ten months, patients aged 18-75 who met our inclusion criteria and did not meet our exclusion criteria were invited to participate in our study. The inclusion criteria were: had a history of hypertension for at least 4 weeks; had blood pressure $\geq 140/90$ mmHg at least on two hospital visits; had been consuming antihypertensive drugs routinely. While the exclusion criteria were: patients unwilling to participate in the study; professional athletes; pregnant women; patients with moderate or severe valvular disease; patients with congenital heart disease; patients with pulmonary arterial hypertension.

Data Collection and Operational Definition

Data were collected through anamnesis, physical examination, laboratory examination during a hospital visit, and medical records if needed. Patients then underwent transthoracic echocardiography procedures performed by echocardiography consultants. During anamnesis, patients were asked for demographic characteristics, symptoms, history and period of hypertension, antihypertensive drugs consumed, comorbidities, heart medications, and alcohol intake history. On physical examination, vital signs, body height, body weight, body mass index (BMI), signs of heart disease, and electrocardiogram (ECG) were evaluated. Hypertension was defined as self-reported antihypertensive treatment for at least two weeks, or average systolic blood pressure (SBP) of ≥ 140 mmHg or diastolic blood pressure (DBP) ≥ 90 mmHg at a minimum of two outpatient appointments, or reported hypertension on the medical record.¹² The period in which patients were diagnosed with hypertension was described in months. Obesity was defined as BMI ≥ 25 kg/m².¹³

Statistical Analysis

Statistical analyses were conducted using IBM SPSS statistics version 26 (IBM Corporation, Armonk, New York, USA). First, we examined the descriptive statistics reported as percentage and frequency for the categorical variable, mean with standard deviation, and median with minimum and maximum value for normally distributed and abnormally distributed numerical variables, respectively. Kolmogorov-Smirnov test was used to analyze data normality. Next, we conducted a bivariate analysis followed by a multivariate analysis for hypertension, BMI or obesity, and coronary heart disease. Bivariate analysis, Chi-Square, was used. It was followed by a logistic regression predictive test to perform multivariate analysis if the bivariate analysis was significant. A two-sided p-value < 0.05 was considered significant.

RESULT

Patient Characteristics

One hundred subjects were recruited in this study, and 97 met the inclusion criteria following data collection and screening. Normally distributed parameters such as age, weight, BMI, diastolic blood pressure, and heart rate were presented in mean \pm standard deviation. The abnormally distributed data were presented in median (min-max). Subject demographic and characteristics are presented in Table 1. The mean age of the study samples was 58.64 years old. The proportion of males and females was 43.3% to 56.7%. 53.6% of the participants had an obese BMI (25 kg/m²). The mean value of BMI was 25.77 ± 5.21 SD. All participants were hypertensive patients, 4.1% of whom did not take anti-hypertensive medication routinely. The median (min-max) of the hypertension period was 60 (3-300) months. 33% of participants have coronary heart disease.

Correlation between Coronary Heart Disease, Hypertension and Obesity

Table 2 highlights the correlation between hypertension, obesity, and CHD. As all our samples were hypertensive, no statistical value can be retrieved. In our bivariate analysis, we found no significant correlation for obesity and hypertension medication toward the event of coronary heart disease.

Correlation Between Length of Hypertension and Coronary Heart Disease

Our in-depth bivariate analysis demonstrates that the correlation power of the period of hypertension towards the event of CHD is not adequate ($r < 0.3$) with an insignificant p-value ($p > 0.05$). Therefore, we conducted no further multivariate analysis (Table 3).

DISCUSSION

This study showed that most participants take antihypertensive medication regularly, and almost half were obese. The prevalence of CHD was higher in the hypertensive groups who take medication and in the obese groups.

Hypertension is known as a significant independent CVD risk factor associated with increased prevalence and severity of CHD.¹⁴⁻¹⁶ However, this study demonstrates that hypertension and the period of hypertension do not correlate with the event of CHD. There was a fragile association between periods of hypertension and CHD. However, the association was insignificant. A study from the Lebanese population suggests that hypertension and its treatment are significantly associated with late-onset CHD, with the mean age of CHD-onset being 61 years old (SD = ± 11).¹⁷ The study did not find a significant association between hypertension and early-onset CHD. The profile of our study participants whose mean age was 58 years old (SD = ± 11). More than half were females who are known to get CHD in later years compared to males. The considered 'young' study participants might explain the low cases of CHD and the poor association between hypertension and the event of CHD. However, we should consider any confounding factors affecting hypertension and CHD, such as socioeconomics, environmental, and genetic factors. These factors are known to play a role in the development of CVD. Low socioeconomic status is related to an increased risk of early CHD.¹⁸ Genetic profile uniqueness can be protective against atherosclerosis and, therefore, CHD.¹⁹

The second variable that we studied was obesity, a well-known independent risk factor for CHD. Even after the diagnosis of CHD, obesity is correlated to the acceleration of CHD progression. It also deteriorates the prognosis of CHD.²⁰ However, this study showed no correlation between obesity and the incidence of CHD. More than half

Table 1. Overall Study Demographic and Clinical Characteristics.

Subject Characteristics	n	%	Mean ± SD	Median (min-max)
Age			58.64 ± 11.75	
Gender				
Male	42	43.3		
Female	55	56.7		
Ethnicity				
Javanese	89	91.8		
Chinese	5	5.2		
Madura	3	3.1		
Body Height (cm)				158 (143 – 186)
Body Weight (kg)			65.16 ± 14.717	
Body Mass Index (BMI) (kg/m ²)			25.77 ± 5.21	
<18.5	4	4.1		
18.5 - 23	23	23.7		
23-25	18	18.6		
25	52	53.6		
Systolic blood pressure (mmHg)				137 (91 – 217)
Diastolic blood pressure (mmHg)			80.38 ± 15.18	
Heart Rate (bpm)			76.74 ± 13.29	
Hypertension				
Yes	97	100		
No	0	0		
Hypertension routine drug consumption				
Yes	93	95.9		
No	4	4.1		
Period of Hypertension (months)				60 (3-300)
LDL (g/dL)				106 (57-242)
HDL (g/dL)				45.25 (31-74)
Triglyceride (g/dL)				121.33 (43-360)
Total Cholesterol (g/dL)				177 (38-314)
Alcohol consumption				
Yes	0			
No	97	100		
Coronary Heart Disease				
Yes	32	33		
No	65	67		

Table 2. Coronary Heart Disease correlation with Obesity and Hypertension.

Variable	Coronary Heart Disease				p value	OR	95% CI
	yes		No				
	n	%	n	%			
BMI					0.22	0.582	0.245 – 1.382
Non-Obese (<25 kg/m ²)	12	37.5	33	50.8			
Obese (≥25 kg/m ²)	20	62.5	32	49.2			
Hypertension	32	33	65	67			
Hypertension therapy					0.729	1.5	0.15 – 15.09
Yes	31	33.3	62	66.7			
No	1	25	3	75			

Table 3. Coronary Heart Disease and Length of Hypertension.

Independent Variable	Total N / Mean ± SD / Median (min-max)	Correlation to Coronary Heart Disease (CHD)	
		r	p value
Hypertension Period (months)	60 (3-300)	0.117	0.27

study participants were obese, yet the incidence of CHD was diverse. Interestingly for Asians, the association between BMI and risks of CVD mortality was U-shaped. Despite the increasing prevalence of obesity in Asian countries, the number of underweight people in Asia is also the highest worldwide. Therefore, in Asia, the increased CVD risks were

associated with both lower and higher BMI.²¹ This might explain why in our study obesity did not correlate with CHD.

Another possible reason for these findings was the inadequate number of study participants, which only represented part of the population. Previous robust studies showing the positive association between

hypertension and CHD primarily include hundreds, even thousands of participants representing the population.^{16, 22-24} This particular reason explains why hypertension does not show an association with CHD. Another possible reason is confounding factors such as ethnicity, diet, exercise or physical activity, and smoking. Most of our subjects were Javanese, unlike Caucasian, African-American, or mongoloids are mainly studied. Ethnicity is a trait that may influence diet and exercise habits. Additionally, each ethnicity has a unique epigenetic profile that influences mortality and morbidity rates related to chronic diseases, especially CVD and its risk factors.^{25,26} Unfortunately, our study did not explore our subjects' exercise and smoking habits, which are also CVD risk factors.

This is a study on the association of CHD, hypertension, and obesity from a clinical registry. The data was only a portion of the bigger scale registry of hypertensive patients. However, there are some study limitations to be considered. First, this is a cross-sectional study; hence no causal relationship can be retrieved. Second, the subjects on this portion of the registry were only 100, with only 97 of the data can be included in this study. This small number of subjects may not depict the whole population, hence the outcomes of this study. Third, only hypertensive patients on appointment to the outpatient clinic were included in this study. This may initiate a selection bias, where most of the data would be patients under anti-hypertensive therapy.

CONCLUSION

CHD can manifest in hypertensive patients disregarding the period of hypertension disease. It can also manifest in patients with or without obesity. This study showed no significant correlations between hypertension, obesity, and CHD. Whether demographic profile plays a major part in CHD should be studied further with a bigger sample size. Study on association between hypertension, obesity, and CHD, specifically for Indonesian ethnicities is also encouraged.

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ETHICAL CONSIDERATION

Participants have given their written consent before participating in this study. The DR Soetomo Ethical Committee ethically approved this study with the following reference number: 0385/KEPK/III/2022.

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CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest to disclose.

AUTHOR CONTRIBUTOR'S

The authors contributed to this study, including literature review, data collection, data analysis, and manuscript preparation.

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