

## Association between Coronary Artery Disease and Body Mass Index

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### Abstract

**Background:** Coronary artery disease (CAD) is a prevalent cardiovascular condition. Body mass index (BMI), a measure of body fat based on height and weight, has been studied as a potential risk factor for CAD.

**Aim and Objective:** The Aim of this study was to evaluate the association between BMI and CAD in male patients undergoing Coronary Angiography and in matched controls.

**Materials and Methods:** This study was done at the Department of Physiology, Thanjavur Medical College Tamil Nadu. Participants (n = 80) were recruited from the population in and around Thanjavur. The Institute Ethical Committee approval was obtained for this study. Subjects who met inclusion and exclusion criteria were included in this study as control group: Group A (n = 40) and study group: Group B (n = 40). Informed, written consent was taken from both groups. BMI was measured for the 80 participants. Angiogram report was obtained from the cardiologist.

**Results:** The mean BMI of 25.5165 ±2.22366 for Group A and the mean BMI of 26.5113 ±2.25700 for Group B. The P value is 0.051(>0.05) which is not significant.

**Conclusion:** It has been concluded that the association between the Body Mass Index and Coronary Artery Stenosis in male patients undergoing coronary angiography is statistically not significant compared to the control group.

**Key Words:** Body Mass Index; Coronary Artery Disease; Coronary Artery Stenosis

### Introduction

Coronary Artery Disease has become the primary cause of death in the world.<sup>(1,2,3)</sup> Researchers claim that by 2030, death from Coronary Artery Disease will be more globally<sup>(2)</sup>. World Health Organisation had estimated that 17 million persons perished of Cardio Vascular Disease in 2004 and there will be nearly 20 million Cardio Vascular Deaths worldwide every year and 24 million deaths by 2030. Each year, there are about 5.8million new Coronary Artery Disease

cases and nearly 40 million persons with established Coronary Artery Disease are living today.<sup>(3)</sup>

Due to the numerous health implications associated with obesity, it is currently estimated to be the second leading cause of preventable death.<sup>(4)</sup> Obesity is associated with increased risk of hypertension (HTN), diabetes mellitus type 2 (DM), the metabolic syndrome (MetS), and dyslipidemia, all risk factors for coronary artery disease (CAD).<sup>(5,6)</sup>

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Obese individuals that are relatively insulin sensitive, labelled metabolically healthy obese (MHO), who have a more favourable cardiovascular (CV) profile compared to insulin-resistant obese (IRO) individuals<sup>(7-11)</sup>. A number of inflammatory responses are found to occur with obesity including increased clotting factors such as fibrinogen, von Willebrand factor, and factors VII and VIII; and increasing plasminogen activator inhibitor type-I that are associated with decreased fibrinolysis, all which may lead to increasing CAD.<sup>(12,13,14)</sup> Elevated levels of tumor necrosis factor alpha in obesity have also been implicated in the development of insulin resistance.<sup>(15)</sup>

In obesity, elevated leptin levels have been associated with negative outcomes in Coronary Artery Disease (CAD) and are linked to in-stent restenosis<sup>(16)</sup>. Additionally, other potential mechanisms contributing to the increased risk of CAD in individuals with diabetes mellitus (DM) include reduced insulin-mediated vasodilation, heightened insulin-mediated renal sodium reabsorption, stimulation of the sympathetic nervous system by insulin, and increased vasoconstriction due to elevated circulating free fatty acids.<sup>(12,13)</sup> Obesity not only increases the risk of hypertension (HTN) and diabetes mellitus (DM) but also raises the risk of dyslipidemia, all potentially increasing the risk for CAD.<sup>(17,18, 19, 20)</sup>

## Materials and Methods

This study was conducted at the Department of Physiology, Thanjavur Medical College, Thanjavur. The study included participants from the general community in and around Thanjavur. Ethical committee approval was obtained prior to conducting the study. It was a case-control study conducted between October 2015 and May 2016. The patient group consisted of 40 male patients who were admitted for diagnostic Coronary Angiography due to symptoms indicative of Coronary Artery Disease. Additionally, a control group of 40 individuals of the same age group and sex was included. The degree of Coronary Artery Stenosis in the patient group was greater than 50%. The results of the angiogram were reported by a Cardiologist.

### Inclusion criteria:

- Patients who were indicated for Diagnostic Coronary Angiography.
- Degree of Coronary Artery Stenosis > 50%
- CRP(<5mg/l)

### Exclusion criteria:

- Hypogonadism
- Hypopituitarism
- Taking drugs that might affect sex hormone level
- High CRP(>5mg/l)
- Previous Cardiovascular Event
- Coronary or Periphery Atherosclerosis.
- Degree of Coronary Artery Stenosis < 50%.

The presence of Coronary Artery Stenosis was established by Coronary Angiography. The angiography was performed in the catheterization laboratory under local anesthesia by a skilled Interventional Cardiologist and the outcome were reported by an Expert Cardiologist. Significant coronary stenosis and thus CAD was defined as a 50% or more narrowing of the lumen diameter in at least one major coronary artery.

Body Mass Index was calculated from the measured height and weight by using Quetelet's Index. BMI is measured by dividing weight in kilogram by height in meter square.<sup>(21)</sup>

The procedure was explained to all the people who participated in this study. Informed written consent was taken from both the controls and subjects.

### Statistical Analysis

The continuous variables are expressed as mean  $\pm$  standard deviation (SD) and compared using Student's t-test or Mann-Whitney U-test between Group A and Group B. Categorical variables were described through frequency and percentage and were compared amongst the above-mentioned groups.

P-value was derived from data analysis using statistical package SPSS version.16 and statistical analysis was performed by Student's "t"-test. If P  $\geq$

0.05, it is not statistically significant. If  $P < 0.05$ , it is considered as statistically significant.

### Results

The continuous variables are expressed as mean  $\pm$  standard deviation (SD) and compared using student's t test between CAD and non-CAD groups. Categorical variables were described through frequency and percentage and were compared among the above mentioned groups.

P value  $< 0.05$  was considered to be statistically significant.

Eighty male subjects(n=80) were participated in this case control study, which were divided into two groups, A (n=40) and B (n=40). Group A includes forty control subjects and Group B includes forty CAD subjects who were referred for diagnostic

Coronary Angiography.

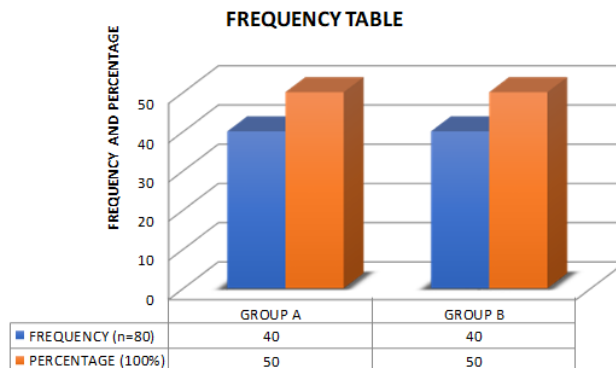


Figure 1

Figure 1 shows the Frequency and percentage of the subjects in this study. Group A has the frequency of n=40 and percentage of 50%. Group B has the frequency of n=40 and percentage of 50%.

### Descriptive Statistics:

#### Mean Value For Both The Groups

Table 1

PARAMETERS	All group (n=80)			
	Minimum	Maximum	Mean	Standard Deviation
BMI	19.33	30.96	26.0139	2.28173

Table 1 shows Mean and the Standard Deviation for BMI for both the groups. BMI shows the mean

value of  $26.0139 \pm 2.28173$  for both the groups.

#### T-Test for Group A and Group B:

Table 2

Group	Mean	S.D	T	DF	P Value
<b>BMI</b>					
A (n=40)	25.5165	2.22366	-1.986	78	.051>0.05 Not Significant
B (n=40)	26.5113	2.25700			

Table 2 shows t test for the parameter BMI for Group A and Group B. The mean BMI of  $25.5165 \pm 2.22366$  for Group A and the mean BMI of  $26.5113 \pm 2.25700$  for Group B. The T = -1.986 with 78 degrees of freedom. The P value is 0.051(>0.05) which is not significant.

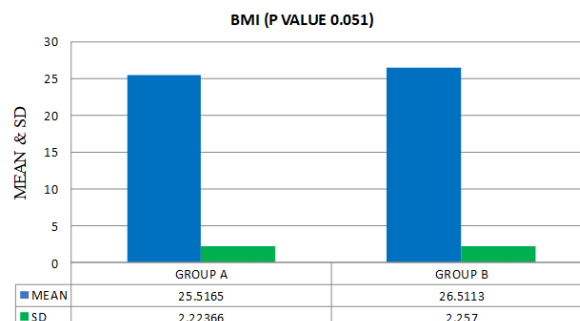


Figure 2

Figure 2 shows Mean and the Standard Deviation for BMI for Group A and Group B. The mean BMI of  $25.5165 \pm 2.22366$  for Group A and the mean BMI of  $26.5113 \pm 2.25700$  for Group B. The P value is  $0.051 (>0.05)$  which is not significant.

## Discussion

Coronary Artery Disease is the leading cause of mortality and morbidity worldwide. Men tend to develop Coronary Artery Disease at an earlier age compared to women. <sup>(2)</sup>

The study compared the Body Mass Index (BMI) results between patients with Coronary Artery Disease and healthy individuals of the same age and sex.

Our study reveals that BMI of Coronary Artery Disease patients are higher than the Healthy Individuals, but statistically insignificant.

### **Our study aligns with previous research that suggests there is no significant association between obesity and Coronary Artery Disease:**

- Our study findings are consistent with the research conducted by A. Alkamel et al <sup>(22)</sup> In their study, they compared the Body Mass Index (BMI) of normal coronary subjects ( $25.82 \pm 3.86$ ) with CAD patients ( $26.30 \pm 3.60$ ) and found that the difference was not statistically significant (p-value = 0.3)
- Our study findings are consistent with the research conducted by GMC Rosano et al <sup>(23)</sup>. In their study, they compared the BMI of CAD patients (26.2) with normal subjects (25.3) and found that the difference was not statistically significant.
- Our study findings align with the research conducted by Malkin et al <sup>(24)</sup>. In their study, they matched the BMI of individuals with normal coronaries (27.7) with those with Coronary Disease (28.0) and concluded that the difference was not statistically significant (p-value = 0.36).

### **Our study contradicts previous research suggesting that obesity increases the risk of Coronary Artery Disease:**

Our study results contradict the findings of the Hypogonadism in Males study <sup>(25)</sup>. According

to that study, which compared hypogonadal men (mean BMI of 31.5) with normal men (mean BMI of 28.5), the odds ratio for having hypogonadism was significantly higher in obese men. The study did not find a statistically significant relationship between total testosterone levels and BMI.

## Limitations

In our study, obesity was measured solely using BMI, and we acknowledge that additional measurements such as waist-to-hip ratio and estimated visceral adipose tissue were not included. These additional measurements can provide more specific information about fat distribution and visceral adiposity, which may be relevant factors in assessing the relationship between obesity and health outcomes.

## Conclusion

In this study involving 40 subjects and 40 patients, no significant association was found between body mass index (BMI) and coronary artery disease (CAD). These findings challenge the widely accepted notion of obesity as a major risk factor for CAD and highlight the complexity of the relationship between BMI and cardiovascular health.

While previous research has often reported a positive correlation between high BMI and CAD, the results of this study suggest that BMI may not be a reliable predictor of CAD risk in this specific sample. However, it is important to acknowledge the limitations of the study, including the small sample size, which may have limited the ability to detect a significant association.

Future research with larger sample sizes and diverse populations is warranted to further explore the relationship between BMI and CAD. It is also essential to consider other potential confounding factors such as genetics, lifestyle factors, and comorbidities that may influence the BMI-CAD relationship.

Understanding the underlying mechanisms and identifying reliable predictors of CAD risk are crucial for developing targeted interventions and effective management strategies. Further research in this area will help refine risk assessment models and contribute to improved CAD prevention and treatment approaches.

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**Conflicts of Interest:** None declared.

**Ethical Clearance:** Taken from Institutional Ethical Committee, Thanjavur Medical College, Thanjavur.

## References

1. Roger Detels, Robert Beaglehole, Mary Ann Lansang, Martin Gulliford. Oxford Textbook of Public Health. 5th. Oxford : Oxford University Press, 2009. pp. 7,186. Vol. 1.
2. Kanu Chatterjee, Mark Anderson, Donald Heistad, Richard E Kerber. Cardiology- An Illustrated Textbook. 1st Edition. New Delhi : Jaypee Brothers Medical Publishers (P) Ltd, 2011. pp. 844-845.
3. Valentin Fuster, Richard A Walsh, Robert A Harrington. Hurst's The Heart. 13th Edition. China : The Mc Graw Hill Companies, 2011. pp. 17-19,24.
4. Masters RK, Reither EN, Powers DA, Yang YC, Burger AE, Link BG. The impact of obesity on US mortality levels: the importance of age and cohort factors in population estimates. American journal of public health. 2013;103(10):1895-901.
5. Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity as an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. Circulation. 1983;67(5):968-77.
6. Todd Miller M, Lavie CJ, White CJ. Impact of obesity on the pathogenesis and prognosis of coronary heart disease. Journal of the cardiometabolic syndrome. 2008;3(3):162-7.
7. Ferrannini E, Natali A, Bell P, Cavallo-Perin P, Lalic N, Mingrone G. Insulin resistance and hypersecretion in obesity. European Group for the Study of Insulin Resistance (EGIR). The Journal of clinical investigation. 1997;100(5):1166-73.
8. Sims EA. Are there persons who are obese, but metabolically healthy? Metabolism: clinical and experimental. 2001;50(12):1499-504.
9. Brochu M, Tchernof A, Dionne IJ, Sites CK, Eltabbakh GH, Sims EA, et al. What are the physical characteristics associated with a normal metabolic profile despite a high level of obesity in postmenopausal women? The Journal of clinical endocrinology and metabolism. 2001;86(3):1020-5.
10. Karelis AD, St-Pierre DH, Conus F, Rabasa-Lhoret R, Poehlman ET. Metabolic and body composition factors in subgroups of obesity: what do we know? The Journal of clinical endocrinology and metabolism. 2004;89(6):2569-75.
11. Karelis AD, Faraj M, Bastard JP, St-Pierre DH, Brochu M, Prud'homme D, et al. The metabolically healthy but obese individual presents a favorable inflammation profile. The Journal of clinical endocrinology and metabolism. 2005;90(7):4145-50.
12. Rader DJ. Effect of insulin resistance, dyslipidemia, and intra-abdominal adiposity on the development of cardiovascular disease and diabetes mellitus. The American journal of medicine. 2007;120 (3 Suppl 1):S12-8.
13. Lee YH, Pratley RE. Abdominal obesity and cardiovascular disease risk: the emerging role of the adipocyte. Journal of cardiopulmonary rehabilitation and prevention. 2007;27(1):2-10.
14. Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet. 2005;365(9468):1415-28.
15. Kershaw EE, Flier JS. Adipose tissue as an endocrine organ. The Journal of clinical endocrinology and metabolism. 2004;89(6):2548-56.
16. Piatti P, Di Mario C, Monti LD, Fragasso G, Sgura F, Caumo A, et al. Association of insulin resistance, hyperleptinemia, and impaired nitric oxide release with in-stent restenosis in patients undergoing coronary stenting. Circulation. 2003;108(17):2074-81.
17. National Cholesterol Education Program Expert Panel on Detection E, Treatment of High Blood Cholesterol in A. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. Circulation. 2002;106(25):3143-421.
18. Grundy SM, Brewer HB, Jr., Cleeman JI, Smith SC, Jr., Lenfant C, American Heart A, et al. Definition of metabolic syndrome: Report of the National Heart, Lung, and Blood Institute/American Heart Association conference on scientific issues related to definition. Circulation. 2004;109(3):433-8.
19. Mokdad AH, Ford ES, Bowman BA, Nelson DE, Engelgau MM, Vinicor F, et al. Diabetes trends in the U.S.: 1990-1998. Diabetes care. 2000;23(9):1278-83.
20. Krauss RM, Winston M, Fletcher BJ, Grundy SM. Obesity: impact on cardiovascular disease. Circulation. 1998;98(14):1472-6.

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21. Menuet R, Lavie CJ, Milani RV. Importance and management of dyslipidemia in the metabolic syndrome. *The American journal of the medical sciences*. 2005;330(6):295-302.
  22. Shlomo Melmed, Kenneth S. Polonsky, P. Reed Larsen, Henry M. Kronenberg. *Williams Textbook of Endocrinology*. 12 Edition. Philadelphia : Elsevier, 2011. pp. 707-708.
  23. Alkamel A, Shafiee A, Jalali A, Boroumand M, Nozari Y. The Association between Premature Coronary Artery Disease and Level of Testosterone in Young Adult Males. *Arch Iran Med*. 2014; 17(8): 545 - 550
  24. Rosano G, Sheiban I, Massaro R, Pgnotta P, Marazzi G, Vitale C, Mercuro G, Volterrani M, Aversa A, Fini M. Low testosterone levels are associated with coronary artery disease in male patients with angina. *Int J Impot Res*. 2007;19:176-182.
  25. Chris J Malkin, Peter J Pugh, Paul D Morris, Sonia Asif, T Hugh Jones, Kevin S Channer. Low serum testosterone and increased mortality in men with coronary heart disease. *Heart* 2010;96:1821e1825. doi:10.1136/hrt.2010.195412
  26. Mulligan T, Frick MF, Zuraw QC, Stemhagen A, McWhirter C. Prevalence of hypogonadism in males aged at least 45 years: the HIM study. *Int J Clin Pract*. 2006;60:762-769.