

## Visceral Adiposity Index and its Correlation with other Anthropometric Indicators of Obesity in a group of Bangladeshi Patients with Acute Coronary Syndrome

Dr. Md. Mahfuzur Rahman<sup>1\*</sup>, Dr. Farid Uddin Ahmed<sup>2</sup><sup>1</sup>Assistant Professor, Department of Cardiology, Abdul Malek Ukil Medical College & Hospital, Noakhali, Bangladesh<sup>2</sup>Assistant Professor, Department of Community Medicine, Rangamati Medical College, Rangamati, BangladeshDOI: [10.36347/sjams.2021.v09i06.018](https://doi.org/10.36347/sjams.2021.v09i06.018)

| Received: 15.04.2021 | Accepted: 24.05.2021 | Published: 17.06.2021

\*Corresponding author: Dr. Md. Mahfuzur Rahman

## Abstract

## Original Research Article

**Background:** The visceral adiposity index (VAI) is proposed as a substitute marker of dysfunction and distribution of adipose tissue and is found to be independently correlated with cardiometabolic risk associated with visceral obesity. However, VAI is not well studied in Bangladeshi population. **Objectives:** This study was aimed to determine the visceral adipose dysfunction (VAD) based on VAI in a group of patients with Acute Coronary Syndrome (ACS) admitted in a tertiary care hospital of Bangladesh and also to determine the correlation of VAI with other anthropometric indicators of obesity. **Methods:** This descriptive cross-sectional study was conducted on 96 admitted patients of ACS at the Department of Cardiology, Abdul Malek Ukil Medical College & Hospital, Noakhali, Bangladesh from January 2019 to June 2019. Fasting serum lipid profile was obtained within 24 hours of hospitalization and demographic and anthropometric parameters [Body Mass Index (BMI), Waist Circumference (WC) and Waist to Height Ratio (WHtR)] were documented. VAI was calculated and VAD was determined for each patient. **Results:** The mean age of the subjects were  $57.7 \pm 14.4$  years with majority (71.9%) being male. Based on BMI 50% patients were obese. 69.8% and 81.3% had central obesity based on WC and WHtR criteria respectively. Overall, mean VAI was high ( $7.48 \pm 2.85$ ) and significantly higher in female than male ( $8.83 \pm 2.89$  versus  $6.95 \pm 2.67$ ;  $p=0.003$ ). All patients had VAD. WHtR had significant positive correlation with VAI ( $r=0.338$ ;  $p=0.001$ ). **Conclusion:** VAD was highly prevalent in this group of ACS patients and WHtR was better than WC and BMI to measure the visceral fat.

**Keywords:** Acute coronary syndrome, Body Mass Index, waist circumference, Waist to Height Ratio, visceral adiposity index.

Copyright © 2021 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

## INTRODUCTION

Obesity, defined as excess body fat, is a well-known risk factor for cardiovascular disease (CVD) [1]. Body mass index (BMI) is the most widely used measure of obesity. The prevalence of obesity, defined as a high BMI, has been increasing worldwide including Bangladesh along with obesity-related disorders, especially CVD [2, 3]. However, central or visceral obesity appear to be more strongly associated with cardiovascular risk [4]. Estimating the extent of visceral adiposity with waist circumference (WC) measurement is widely used CVD risk assessment. However, this method cannot distinguish between subcutaneous and visceral fat accumulation [5]. Computed tomography (CT) and magnetic resonance imaging (MRI) precisely and reliably quantify individual differences in abdominal fat distribution and distinguish visceral adipose tissue from subcutaneous

adiposity, but both methods are expensive and CT has a high risk of radiation exposure [6]. Thus, Amato et al. established the visceral adiposity index (VAI) model that was based on a Caucasian population; this multivariate model includes non-invasive, simple anthropometric data and lipid parameters (WC, BMI, serum triglycerides [TG], and high-density lipoprotein cholesterol [HDL-C] levels) to incorporate functionality in deriving a measure of dysfunctional adipose tissue that is not directly visceral adiposity [7]. VAI significantly correlates with metabolic syndrome and cardiovascular risk [7]. However, there are conflicting data, [8, 9] partly due to the study of different populations and ethnicity groups [10, 11]. The application of the VAI to non-Caucasian populations is limited, and there is little data regarding the Bangladeshi population. A more reliable and simple indicator for visceral obesity is essential for both

primary and secondary prevention of obesity related consequences. In this context, we aimed to determine the visceral adipose dysfunction (VAD) based on VAI in a group of Bangladeshi patients admitted with Acute coronary Syndrome (ACS) in a tertiary care hospital. We also assessed the correlation between different anthropometric indicators of obesity [BMI, WC, Waist-to-Height Ratio (WHtR)] and VAI.

## METHODOLOGY AND MATERIALS

This cross sectional observational study was conducted among 96 patients with a diagnosis of ACS admitted at Abdul Malek Ukil Medical College & Hospital, Noakhali, Bangladesh from January 2019 to June 2019 after taking Ethical clearance from the institutional Ethical Review Committee. All the participants had signed the consent for the study. History and clinical examination were performed as per the pre-structured proforma. Ninety six cases above the age of 18 years with a diagnosis of ACS were enrolled by a convenience sampling method. Patients with stable coronary artery disease were excluded. Height was measured to the closest 0.1 cm and the weight was

$$\text{For male: VAI} = \left( \frac{\text{WC}}{39.68 + (1.88 \times \text{BMI})} \right) \times \left( \frac{\text{TG}}{1.03} \right) \times \left( \frac{1.31}{\text{HDL}} \right)$$

$$\text{For female: VAI} = \left( \frac{\text{WC}}{39.58 + (1.89 \times \text{BMI})} \right) \times \left( \frac{\text{TG}}{0.81} \right) \times \left( \frac{1.52}{\text{HDL}} \right)$$

Optimal VAI cut-off points are: 2.52 (age < 30 years), 2.23 (age ≥30 and < 42 years), 1.92 (age ≥42 and < 52 years), 1.93 (age ≥52 and < 66 years) and 2.00 (age ≥ 66 years). Patients with VAI scores greater than these cut-off points were arbitrarily defined as having VAD. Statistical analyses were performed by SPSS version 23. Both descriptive and inferential data analysis methods were used. Descriptive statistics included means±standard deviations and frequency (percentage). Inferential methods included chi-square tests of association (for comparison of two categorical variables) and student's t test (to test the mean differences). Correlation between VAI and anthropometric indices (BMI, WC, WHtR) was determined by Pearson Correlation Coefficient. P <0.05 was considered as statistical significant.

## RESULTS

Out of 96 ACS patients there were 30 cases of UA, 25 cases of NSTEMI and 41 cases of STEMI. Age ranges from 21-95 years with mean age of 57.7±14.4 years. 69 (71.9%) of the patients were male and 27 (28.1%) were female with a male to female ratio of

measured to the closest 0.1 kg using an automatic height weight scale. BMI was estimated based on weight (kg) divided by height (m<sup>2</sup>). WC was measured at the umbilicus with the patient in the standing position. The same person acquired all measurements of the anthropometric parameters throughout the study in order to reduce variations. The cut-off point for obesity based on BMI was 25 kg/m<sup>2</sup> [12]. Abdominal Obesity was defined based on WC for male: ≥ 90 cm and female ≥ 80 cm [13]. WHtR was determined by the ratio between WC (cm) and Height (cm) and value of 0.50 was considered as central obese [5]. An electrocardiogram (ECG) at presentation was performed in all the patients and the cases were categorized as unstable angina (UA), non-ST elevation MI (NSTEMI) and ST-elevation MI (STEMI) based on the history, ECG changes and cardiac markers [14]. Fasting serum lipid profile was obtained within 24 hours of hospitalization. Lipid assay was done with Enzymatic Colorimetric Test for TC and TG with lipid Clearing Factor. LDL-C was determined by direct method and HDL-C by precipitation method. VAI was defined by the following formulas as described by Amato et al [7].

2.55:1. Most prevalent risk factor was smoking (55.2%), followed by hypertension (47.9%), diabetes mellitus (37.5%), dyslipidemia (27.1%) and family history of CAD (15.6%). Prevalence of smoking and family history of CAD was significantly higher and prevalence of hypertension and reported dyslipidemia was significantly lower in male compared to female (Table 1). Obesity was determined by BMI, WC, WHtR and VAI in this study. Table 2 shows that, VAI was the most sensitive (100%) in determining the obesity in these patients, followed by WHtR (81.3%), WC (69.8%) and BMI (50%). As per the VAI cutoff value the entire studied group had VAD. As per WC criteria, prevalence of abdominal obesity was significantly higher in male but mean VAI was significantly higher in female patients compared to their counterpart. Correlation between VAI and other anthropometric indicators were shown in Figure 1-3. Among the three anthropometric indices, WHtR had the strongest positive correlation with VAI (r=0.338; p=0.001) followed by WC (r=0.225; p=0.027). Though BMI had weak positive correlation (r=0.175) it was not statistically significant (p=0.087).

**Table-1: Demographic and risk factors of CAD in ACS patients stratified by gender**

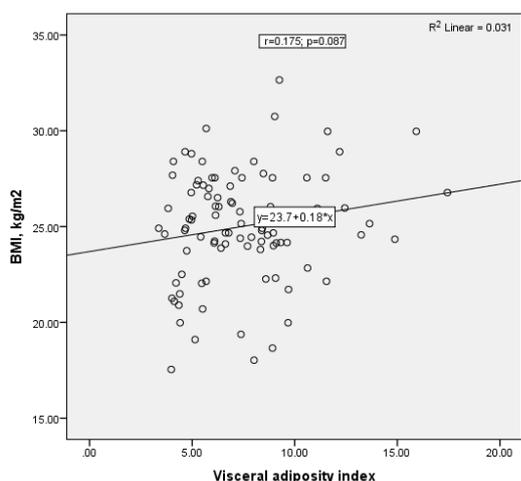
Variables	Total (n=96)	Male (n=69)	Female (n=27)	P value
Age, years	57.69±14.36	56.38±13.79	61.04±15.52	0.154 <sup>†</sup>
Smoking	53 (55.2)	52 (75.4)	1 (3.7)	<0.001*
Hypertension	46 (47.9)	28 (40.6)	18 (66.7)	0.021*
F/H of CAD	15 (15.6)	15 (21.7)	0 (0)	0.008*
Diabetes	36 (37.5)	25 (36.2)	11 (40.7)	0.682*
Dyslipidemia <sup>a</sup>	26 (27.1)	13 (18.8)	13 (48.1)	0.004*

Data were expressed as mean ±SD or frequency (percentage). <sup>a</sup>Reported by patient and under medication. P value was obtained from <sup>†</sup>independent sample t test or \*Chi-square test. CAD: Coronary artery diseases.

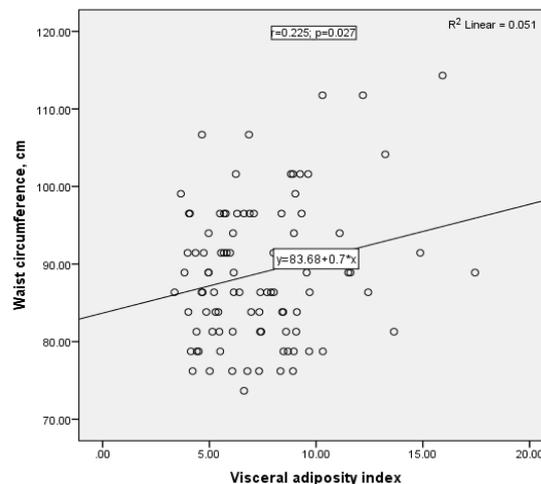
**Table-2: Obesity indicators in ACS patients stratified by gender**

Variables	Total (n=96)	Male (n=69)	Female (n=27)	P value
<b>BMI, kg/m<sup>2</sup></b>				
Mean ±SD	25.01±2.68	24.85±2.94	25.41±2.64	0.393 <sup>†</sup>
BMI ≥25 kg/m <sup>2</sup>	48 (50.0)	34 (49.3)	14 (51.9)	0.538*
<b>WC, cm</b>				
Mean ±SD	88.92±8.99	91.18±7.90	83.16±8.82	<0.001 <sup>†</sup>
Abdominal obesity	67 (69.8)	53 (76.8)	14 (51.9)	0.017*
<b>WHtR</b>				
Mean ±SD	0.55±0.06	0.55±0.6	0.55±0.6	0.582 <sup>†</sup>
Central obesity	78 (81.3)	58 (84.1)	20 (74.1)	0.262*
<b>VAI</b>				
Mean ±SD	7.48±2.85	6.95±2.67	8.83±2.89	0.003 <sup>†</sup>
VAD	96 (100.0)	69 (100.0)	27 (100.0)	NA

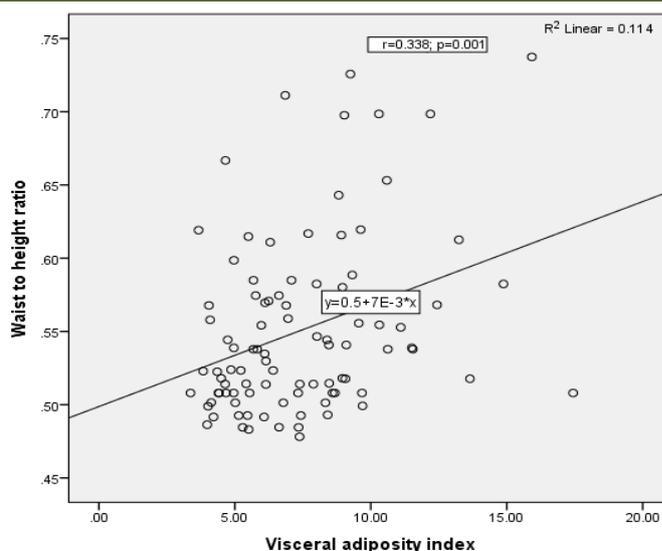
Data were expressed as mean ±SD or frequency (percentage). P value was obtained from <sup>†</sup>independent sample t test or \*Chi-square test. NA: Not applicable.



**Fig-1: Correlation between VAI and BMI**



**Fig-2: Correlation between VAI and WC**



**Fig-3: Correlation between VAI and WHtR**

## DISCUSSION

Visceral obesity is associated with an increased risk of CVD. The quantification of the visceral fat becomes necessary and advantageous in clinical practice, especially through accurate and precise methods in replacement of imaging methods as CT and MRI [5]. VAI is a simple, gender specific marker combining anthropometric data and lipid profiles and are reliable indicator of visceral fat dysfunction. Several studies found a strong association of VAI with cardiometabolic risk [16, 17]. In the current study mean VAI value of the 96 patients with ACS was higher than the recommended normal level and all of the included patients had VAD. BMI is the most widely used measure of obesity and the prevalence of obesity (defined as  $BMI \geq 25 \text{ kg/m}^2$ ) was 50% in the current study. Previous study conducted in this area of Bangladesh reported much lower frequency (<10%) of obesity in ACS patients [18]. But recent obesity trend is rising in Bangladesh as evident by Khanom et al. [13] who have reported that 20% of the adult general population of Bangladesh is obese as per BMI criteria. It is to be noted that, though obesity is an independent risk factor for CVD, in the present study half of the ACS patients were not obese. Although it seems logical that obesity or adiposity should be accompanied by more accumulation of fat cells everywhere in the body, including vascular walls (atherosclerotic plaques), it must be clarified that first of all, obesity per se is not adiposopathy, and second, the process of atherosclerosis is not a simple process of fat accumulation [19]. It seems that visceral adipose tissue is metabolically more active and pathological than subcutaneous adipose tissue, and induces immunity processes that contribute to atherosclerotic CVD [20]. The answer to the question raised from the obesity paradox is that atherosclerotic disease does not result from the accumulation of adipose tissue per se but is as a result of adipose tissue dysfunction, or 'sick fat' [20, 21]. In this regards, it

would be more beneficial to assess the visceral adiposity with other indices than BMI for primary and secondary prevention of CVD. Alternative methods for estimating visceral fat are WC and WHtR. In the present study, both of these indicators identified a higher proportion of patients with obesity compared to BMI (69.8% by WC and 81.3% by WHtR). Most important observation of the current study was that among the three anthropometric indicators WHtR has the strongest positive correlation with VAI. WHtR is considered a simple, fast, low cost trial tool superior than BMI for evaluating health risks and mortality by CVD and for all the causes [22, 23]. It is of great concern that, the entire ACS group of the current study had VAD, as assessed by VAI. A recent study from Bangladesh VAI was found to be a strong predictor of the severity of CAD [17]. Most recently, Bagyura et al. [24] reported that, VAI tertiles were associated with calcium scores and the highest VAI tertile was an independent predictor for the presence of Coronary artery calcium score >100. Our study has the limitations of a cross sectional analysis of a group of ACS patients without any control group. All the patients were selected from a single hospital and visceral adiposity was measured by VAI instead of CT and MRL. Determining the visceral adiposity for primary and secondary prevention of CVD related mortality might allow for an early risk stratification and prediction. In conclusion the VAD was highly prevalent in these ACS patients. VAI was the most sensitive anthropometric indicators for visceral adiposity among BMI, WC and WHtR. However, optimal cutoff margin of VAI need to be determined by large prospective studies in Bangladesh.

### Limitations of the study

This was a single center study with limited sample size. So, the result might not be reflecting the scenarios of the whole country.

## CONCLUSION AND RECOMMENDATIONS

VAD was highly prevalent in this group of ACS patients and WHtR was better than WC and BMI to measure the visceral fat.

**Funding:** No funding sources.

**Conflict of interest:** None declared.

**Ethical approval:** The study was approved by the Institutional Ethics Committee.

## REFERENCES

- Pagidipati NJ, Zheng Y, Green JB, McGuire DK, Mentz RJ, Shah S, et al. (2020). Association of obesity with cardiovascular outcomes in patients with type 2 diabetes and cardiovascular disease: Insights from TECOS. *Am Heart J.*, 219:47-57.
- Hruby A, Hu FB. (2015). The Epidemiology of Obesity: A Big Picture. *Pharmacoeconomics.*, 33(7):673-89.
- Khanam F, Hossain MB, Mistry SK, Afsana K, Rahman M. (2019). Prevalence and Risk Factors of Cardiovascular Diseases among Bangladeshi Adults: Findings from a Cross-sectional Study. *Journal of epidemiology and global health.*, 9(3): 176–184.
- Despres JP. (2012). Body fat distribution and risk of cardiovascular disease: an update. *Circulation*, 126(10):1301-1313.
- Roriz C, Karla A, Passos S, Carlos L, Oliveira C, De Almeida M, Barbosa R, et al. (2016). Anthropometric clinical indicators in the assessment of visceral obesity: an update. *Nutr. clín. diet. hosp.*, 36(2):168-179.
- Graffy PM, Pickhardt PJ. (2016). Quantification of hepatic and visceral fat by CT and MR imaging: relevance to the obesity epidemic, metabolic syndrome and NAFLD. *Br J Radiol.*, 89(1062):20151024.
- Amato MC, Giordano C, Galia M, Criscimanna A, Vitabile S, Midiri M, et al. (2010). Visceral Adiposity Index: a reliable indicator of visceral fat function associated with cardiometabolic risk. *Diabetes care.*, 33 (4):920–922.
- Mohammadreza B, Farzad H, Davoud K. (2012). Prognostic significance of the complex" visceral adiposity index" vs. simple anthropometric measures: Tehran lipid and glucose study. *Cardiovascular diabetology.*, 11(1):20.
- Elisha B, Messier V, Karelis A, Coderre L, Bernard S, Prud'homme D, et al. (2013). The visceral adiposity index: Relationship with cardiometabolic risk factors in obese and overweight postmenopausal women– A MONET group study. *Applied Physiology, Nutrition, and Metabolism.*, 38(8):892–899.
- Lear SA, Humphries KH, Kohli S, Chockalingam A, Frohlich JJ, Birmingham CL. (2007). Visceral adipose tissue accumulation differs according to ethnic background: results of the Multicultural Community Health Assessment Trial (M-CHAT)–. *The American journal of clinical nutrition.*, 86(2):353–359.
- Tanaka S, Horimai C, Katsukawa F. (2003). Ethnic differences in abdominal visceral fat accumulation between Japanese, African-Americans, and Caucasians: a meta-analysis. *Acta diabetologica.*, 40(1): s302–s4.
- WHO Expert Consultation. (2004). Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet.*, 363(9403):157-163.
- Misra A, Vikram NK, Gupta R, Pandey RM, Wasir JS, Gupta VP. (2006). Waist circumference cutoff points and action levels for Asian Indians for identification of abdominal obesity. *Int J Obes (Lond).*, 30(1):106-111.
- Findlay IN, Cunningham AD. (2005). Definition of acute coronary syndrome. *Heart.*, 91(7):857–859.
- Amato MC, Giordano C, Pitrone M, Galluzzo A. (2011). Cut-off points of the visceral adiposity index (VAI) identifying a visceral adipose dysfunction associated with cardiometabolic risk in a Caucasian Sicilian population. *Lipids Health Dis.*, 10:183.
- Kang YM, Jung CH, Cho YK, Jang JE, Hwang JY, Kim EH, et al. (2017). Visceral adiposity index predicts the conversion of metabolically healthy obesity to an unhealthy phenotype., *PloS One* 12(6):e0179635.
- Singha C, Biswas E, Choudhury A, Khalequzzaman M, Khanam R, Mahmood S, Paul N. (2017). Association of Visceral Adiposity Index Score with the Severity of Coronary Artery Disease in Patients with Ischemic Heart Disease. *BHJ.*, 32(1):36-39.
- Das PK, Kamal SM, Murshed M. (2015). Acute myocardial infarction in young Bangladeshis: A comparison with older patients. *Journal of Indian College of Cardiology.*, 5(1):20–24.
- Snijder MB, van Dam RM, Visser M, Seidell JC. (2006). What aspects of body fat are particularly hazardous and how do we measure them? *Int J Epidemiol.*, 35(1):83-92.
- Bays HE, González-Campoy JM, Bray GA, Kitabchi AE, Bergman DA, Schorr AB, Rodbard HW, Henry RR. (2008). Pathogenic potential of adipose tissue and metabolic consequences of adipocyte hypertrophy and increased visceral adiposity. *Expert Rev Cardiovasc Ther.*, 6(3):343-368.
- Bays HE. (2011). Adiposopathy is "sick fat" a cardiovascular disease? *J Am Coll Cardiol.*, 57(25):2461-73.
- Savva SC, Lamnisos D, Kafatos AG. (2013). Predicting cardiometabolic risk: waist-to-height ratio or BMI. A meta-analysis. *Diabetes Metab Syndr Obes.* 6:403-419.
- Schneider HJ, Friedrich N, Klotsche J, Pieper L, Nauck M, John U, et al. (2010). The predictive value of different measures of obesity for incident cardiovascular events and mortality. *J Clin Endocrinol Metab.*, 95(4):1777-85.
- Bagyura Z, Kiss L, Lux Á, Csobay-Novák C, Jermendy ÁL, Polgár L, Szélid Z, Soós P, Merkely B. (2020). Association between coronary atherosclerosis and visceral adiposity index. *Nutr Metab Cardiovasc Dis.* 30(5):796-803.