Research Article

Association of Visceral Fat Index with Coronary Collateral Circulation in Patients with Coronary Artery Disease

Shazia Nazar[1], Erum Afaq[*, 1], Shayan Zufishan[2], Nahida Baigam[3], Ghazala Masood Farrukh[1], Sumera Mustafa[1]

[1]*Department of Physiology, Dow University of Health Sciences, Karachi, Pakistan.*
[2]*Department of Biochemistry, Karachi Medical and Dental College, Karachi, Pakistan.*
[3]*Department of Medicine, Abbasi Shaheed Hospital, Karachi, Pakistan.*

Abstract: Background: Coronary collateral circulation (CCC) is a network of anastomosing channels, established by heart in response to ischemia of myocardium. Hypertension, diabetes mellitus, and BMI are well established risk factors for development of poor CCC. Good CCC minimizes symptoms of angina, reduce the size of infarcts and prevent adverse cardiac events. The current research study was designed to find out the association of visceral adiposity and development of CCC among patients with coronary artery disease.

Materials and Methods: The prospective study, conducted in Civil Hospital Karachi, comprised of 270 patients of coronary artery disease. According to the Rentrop Cohen categorization, patients were placed into two groups: the group with good collateral circulation having Rentrop grades i2-3 (n = 140) and the group with poor collateral circulation having Rentrop grades i0-1(n = 130). Rentrop score was determined by angiography. Segmental multi-frequency bioelectrical impedance analyzer was used to determine body fat mass and body muscle mass. Using Omron body fat and weight measurement systems, visceral fat index (VFI) was evaluated to determine the composition of visceral adipose tissue. SPSS was used for data analysis (Version 22). To assess the independent risk factor for poor CCC, logistic regression analysis was used. ROC curve was constructed to assess the efficacy of VFI in identifying CCC.

Results: Overall, good collateral circulations were observed in 51.9% (n=140) of CAD patients, whereas poor collateral circulations were found in 48.1% (n=130) of patients. Poor CCC was significantly associated with hypertension (OR=3, 95%CI= 0.111-8.231, p = .001) and VFI (OR=2, 95%CI= 1.451-3.567, p =.001). ROC analysis revealed a VFI > 9 to be a potential predictor of poor CCC with AUC=0.9, sensitivity of 95.00% and specificity of 86%.

Conclusion: The current study concluded that greater VFI and concomitant hypertension considerably increase the likelihood of having poor CCC, therefore, visceral adiposity can be considered as a potential target for preventing poor collateral circulation in patients with established cardiac disease.

Keywords: Coronary artery disease, Coronary collateral circulation, Visceral adiposity, Visceral fat index, Hypertension, Atherosclerosis.

INTRODUCTION

Coronary Artery Disease (CAD) is a one of the main causes of morbidity and mortality worldwide [1]. According to World Life Expectancy, Pakistan's recorded rate of CAD deaths in 2020 was 16.49%, ranking it 30th globally [2]. Atherosclerosis, characterize by coronary artery stenosis by fibrofatty plaque, is the major cause of developing CAD [3].

Coronary collateral circulation (CCC) is an anastomosing channel, established by heart in response to decrease oxygen supply to myocardium due to stenosis of major coronary arteries [4]. Development of good CCC leads to reduced symptoms and sequalae of ischemia by protecting the myocardium [5, 6].

Intact, healthy, and functioning endothelial lining is necessary for the establishment of coronary collateral circulation [7]. According to studies, aging, multi-vessel coronary stenosis, more than 80% obstruction of coronary arteries, physical inactivity, smoking, obesity, abnormal lipid profile, hereditary variables, and insulin resistance can all affect the development of good coronary collateral circulation [8, 9]. However, the exact mechanism of CCC is yet unknown.

Obesity is thought to be a distinct risk factor for development of coronary artery disease (CAD) [10]. Vascular permeability and ongoing inflammation have been related to a greater risk of cardio-vascular events in obese individuals [11]. Visceral obesity has been shown to have a greater connection with CAD risk because visceral adipose tissue (VAT) has higher pro-inflammatory tendencies than subcutaneous adipose tissue (SAT) [12].

The current research study was planned to find out the association of visceral adiposity and development of CCC among CAD patients.

MATERIALS AND METHODS

This is a prospective study, conducted from January 2018

*Address correspondence to this author at the Department of Physiology, Dow University of Health Sciences, Karachi, Pakistan.
Email: erum.afaq@duhs.edu.pk

to June 2018, at the Cardiac wards of Civil Hospital Karachi (CHK), Pakistan. The ethical committee of Dr. Abdul Qadeer Khan Institute of Biotechnology Genetic Engineering (KIBGE) permitted the study protocol. A total of 270 diagnosed CAD patients (males: 222, females: 48) were recruited in the current study. Prior to data collection, each participant gave signed consent after being fully informed. Angiography served as the diagnostic standard. CAD was defined as the obstruction of one or more coronary arteries by more than 50%.

According to the Rentrop Cohen categorization, patients were placed into two groups: the group with good collateral circulation having Rentrop grades i2-3 (n = 140) and the group with poor collateral circulation having Rentrop grades i0-1(n = 130).

The exclusion criteria were pregnancy, malignancy, coronary revascularization, liver or kidney disorders, and infections in previous 3 weeks. The patients with ascites, pleural effusion, and pretibial edema were not undergone the process.

Information about age, gender, smoking habits, physical activity, dietary habits, and family history of CAD, history of diabetes mellitus, history of hypertension, and concurrent medications were all collected using questionnaire. The experienced cardiologists performed coronary angiography, evaluating the total coronary obstruction to establish the CCC. Rentrop scoring was used to rate collateral blood flow in epicardial blood vessels [13].

Grade 0: No visible collaterals.

Grade 1: Side branch being filled via collateral vessels without the epicardial segment being visible.

Grade 2: Epicardial vessels are partially filled.

Grade 3: Epicardial vessels are fully filled.

Segmental multi-frequency bioelectrical impedance analyzer (BIA) (InBody I770) was utilized to calculate total body fat mass and total body muscle mass. On the morning of the first hospital day, measurements of bio-impedance were taken, paying special consideration to the degree of hydration, time, and attire (with standard clothing and no personal belongings). Using Omron body fat and weight measurement systems, visceral fat index (VFI) was evaluated to determine the composition of visceral adipose tissue (V-body HBF371; Omron, Kyoto, Japan). The normal visceral fat range should be about 10% of body fat. Age, gender, weight, and height of the study participants were entered into the device to obtain visceral fat index. Participants held the electrodes in both hands with the arms extended, while standing barefoot on the footplate.

Threshold values used to categorize visceral fat:

- 19: normal.
- 110-14: high.
- 115-30: very high.

C-reactive protein (CRP) in plasma was measured using an enzyme-linked immunosorbent assay (ELISA) kit (Catalog No. BTB-E1805Hu) made up of micro titer plates that were already coated with human hs-CRP antibodies.

An automated hematology analyzer (Sysmex XE-2100) was used to determine the absolute lymphocyte, platelet, and neutrophil count. The neutrophil to lymphocyte ratio (NLR), platelet to lymphocyte ratio (PLR) and neutrophil to platelet ratio (NPR) were calculated.

Sphygmomanometer was used to measure blood pressure in millimeters of mercury (mmHg). Values more than 140/90 mm of Hg were considered as hypertension. Using a portable stadiometer, the patients' height was estimated (Seca 213, USA). The weight of the participants was measured using a weighing scale (Camry-China-BR 9016), with accuracy set at 0.1 kilograms. To calculate the body mass index (BMI), weight and height were used. BMI as measured in kg/m^2 units.

STATISTICAL ANALYSIS

SPSS, Version 22, was used for data analysis (IBM Corp., Armonk, NY, USA). Categorical variables were subjected to a Pearson chi square analysis. Uneven distributed numerical data were analyzed using the Mann-Whitney U test, whereas regularly distributed data were analyzed using the Student's t test. To examine the correlations between the research variables, a Pearson's correlation test was used. To assess the independent risk factor for poor CCC, logistic regression analysis was used. By determining the area under the curve, the ROC curve was constructed to assess the efficacy of VFI in identifying CCC.

RESULTS

Table **1** displays the biochemical, anthropometric, and demographic details of CAD patients with good and poor collateral circulation. Overall, good collateral circulations was observed in 51.9% (n=140) of CAD patients, whereas poor collateral circulations were found in 48.1% (n=130) of patients. Right coronary artery blockage was found more prevalent in patients with single and double vessel diseases. Poor CCC was observed in older CAD patients (61.3 ± 9.8 vs. 50.7 ±13.2, p=.001). Poor CCC was significantly associated with hypertension (76.9 % vs. 53.9%, p = .001), diabetes mellitus (50.8% vs. 34.4%, p=.001), and use of beta blockers (80.0% vs. 62.9%, p = .001). A statistically significant difference was found between both groups in terms BMI (34.9 ± 7.9 vs. 26.1 ± 12.4, p = .001), fat mass (24.9± 2.8vs. 17.8± 3.9, p = .001) and VFI (123 vs. 31, p =.001).

However, the lipid profile and inflammatory biomarkers were similar in both the poor and good CCC groups. Neutrophils, lymphocytes, and platelet count were found to be decreased in the poor CCC group but found to be statistically insignificant (Table **1**).

Table 1. Demographic and Clinical Characteristics of Study Population.

Study Parameters	Good CCC (Rentrop=2-3) n=140	Poor CCC (Rentrop=0-1) n=130	*p-value*
Demographic Parameters			
Age (years)	50.7± 13.2	61.3± 9.8	.001
Gender			
Male	104(74.3%)	118(90.8%)	.006
Female	36(25.7%)	12 (9.2%)	
Comorbidities n (%)			
Diabetes mellitus	48 (34.4%)	66 (50.8%)	.001*
Hypertension	74 (52.9%)	100 (76.9 %)	.001*
Concomitant Medications n (%)			
Beta blockers drugs	88 (62.9%)	104 (80.0%)	.001*
Nitrates	8 (5.7%)	10 (7.7%)	.091
Anti-platelets	138 (98.6%)	120 (92.3%)	.007
Anti- hypertensives	126 (96.9%)	132 (94.3%)	.027
Characteristics of Occluded Coronary Vessels n (%)			
Right coronary artery	86 (61.4%)	106 (81.5%)	.001*
Left circumflex artery	20 (14.35%)	4 (3.1%)	.092
Left anterior descending artery	34 (24.2%)	20(15.3%)	.011
Single vessel disease	51 (36.4%)	52 (40.0%)	.045
Double vessel disease	60 (42.8%)	74 (56.9%)	.007
Multi vessel disease	29(20.7%)	4(3.07%)	.098
Anthropometrics			
BMI	26.1 ±12.4	34.9 ± 7.9	.001*
Muscle Mass	49.9± 2.9	52.7 ± 8.7	.098
Fat mass	17.8± 3.9	24.9± 2.8	.001*
Visceral Fat Index n (%)			
1-9	119(85%)	7(5.3%)	.001*
≥ 9	21(15%)	123(94%)	.001*
Hemogram			
WBCs ($\times 10^9$/ L)	9.18	7. 34	.023
Lymphocytes($\times 10^9$/ L)	1.2	0.9	.009
Platelets(× 103 /uL)	4.4	3.9	.031
Biochemical Parameters			
TC mg/dl	224. 61 ± 9.9	229± 12.3	.98
LDL-c mg/dl	160.09 ±4.8	172.9 ± 9.1	.009
HDL-c mg/dl	38.1 ±5.5	35.9± 0.2	.034
Inflammatory Markers			
CRP mg/dl	60.9± 18.7	69.8± 12.8	.091
NLR	.01	.01	.234
PLR	.02	.02	.087
NPR	.01	.01	.760

Data is expressed as the 1mean± standard 1deviation, frequency and percentages, *Significant 1p value ≥ .05, BMI: body mass index, TC: total cholesterol, HDL-c: high density lipoprotein cholesterol, LDL-c: low density lipoprotein cholesterol, CRP: C-reactive protein, NLR: neutrophil-leukocyte ratio, PLR: platelet-leukocyte ratio, NPR: neutrophil-platelet ratio, CCC: collateral coronary circulation.

ROC CURVE ANALYSIS

The relationship between visceral fat index and Rentrop scores was found to be significantly negative (r = -0.6, p= .001). ROC analysis showed that visceral fat index more than 9 might be a possible indicator of poor development of coronary collateral circulation (Fig. **1**).

UNI-VARIABLE AND MULTI-VARIABLE REGRESSION ANALYSIS TO DETERMINE THE INDEPENDENT RISK FACTOR FOR POOR CCC

A univariable regression analysis was performed to evaluate the risk assessment of clinical variables with poor CCC. VFI (OR=2, 95% CI= 1.451-3.567, p=.001), and hypertension (OR 3, 95% CI=1.111 to 8.231, p=.002) remained the strong predictors of poor CCC in CAD patients in multi variable logistic regression model adjusted for study variables including age, BMI, DM, body fat mass and body muscle mass (Table **2**).

Table 2. Logistic Regression Analysis.

Variables	Uni-variate Analysis		Multi-variate Analysis	
	OR(95%CI)	p^a	OR(95%CI)	p^b
Age (years)	1 (1.031-1.022)	.001	1 (0.899-1.23)	0.99
BMI ≥25Kg/m^2	1 (1.143-3.423)	.034	.889 (0.132-5.589)	.889
Diabetes Mellitus	1 (0.898-2.56)	.045	2 (0.777-3.68)	.171
Hypertension	2 (1.321-3.256)	.004	3 (1.111-8.231)	.001*
Body Muscle Mass	1 (0.901-1.231)	.091	1 (0.982-1.23)	.231
Body Fat mass	1 (1.023-1.142)	.002	0.7 (0.443-1.005)	.982
VFI	1.4 (1.347-1.887)	.001*	2 (1.451-3.567)	.001*

p^a= p for uni-variable analysis, p^b= p for multivariable analysis adjusted for age, BMI, BFM, BMM, diabetes mellitus. Odds ratio (95% CI) was expressed for the risk assessment, * Significant *p* value.

DISCUSSION

The physiological adaptation known as coronary collateral circulation (CCC) develops to restore blood flow to myocardium as

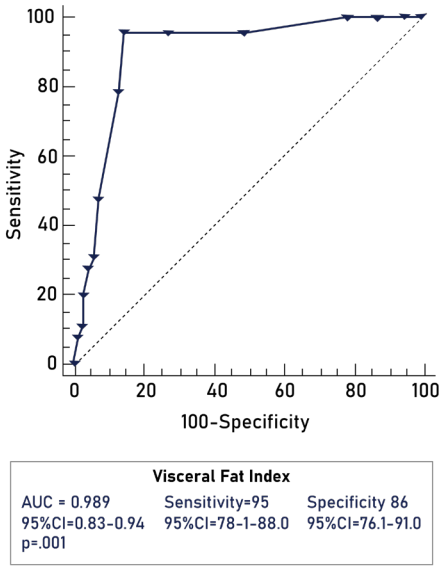

Fig. (1). ROC Curve Analysis Describing the Association of VFI and CCC, AUC= Area under Curve.

it receives less blood and oxygen owing to coronary artery stenosis [14]. CCC acts as a natural bypass system, and expected to improve survival after temporary or permanent coronary blockage and reduce the size of infarcts in CAD patients, restoring myocardial function [15].

The current study found that higher VFI (≥ 9) and hypertension are significant contributors of poor collateral circulation in CAD patients. In addition, aging, obesity, increased body fat mass, diabetes mellitus and smoking are also associated with increased possibility of developing impaired coronary collateral circulation in patients with CAD.

Literature has revealed that components of metabolic syndrome including old age, high blood pressure and increased BMI are associated with impaired CCC in obstructive CAD [16-18].

According to Koerselman *et al*., pack years of smoking were inversely linked with CCC but current smoking was positively associated (OR= 4.17; 95% CI= 1.79-9.71) [19].

Studies have produced conflicting results about the relationship between diabetes mellitus and the development of CCC. Akyuz *et al*., demonstrated that diabetes is a significant factor for poor CCC [20]. However, Zbinden *et al*., did not discover a link between diabetes and a higher probability of having bad CCC [21].

Our study found no association between TC, HDL-c, LDL-c and inflammatory biomarkers including CRP, NLR, and PLR with development of CCC.

İleri *et al*., discovered that neutrophil-lymphocyte ratio (NLR), along with the presence of diabetes mellitus, was an independent predictor of poor CCC, Asli *et al*., however, came to the opposite conclusion, finding that increased NLR was substantially related

with improved CCC formation in coronary artery disease [22, 23].

HDL-c levels were discovered to be less in the poor CCC class than in the good CCC group (37.3 9.8 vs. 44.1 8.6, p = .001), according to research by Yildirim *et al*., The group with poor CCC had higher LDL- c levels (185.7 39.2 vs. 132.8 28.1, p = .000). Moreover, he discovered that the poor CCC group had higher CRP levels (3.73 2.5 vs. 1.67 1.4, p =.001) [24].

Visceral adipose tissue (VAT) is now considered as an organ which is highly active metabolically. Unrelated to BMI, hypertrophy of visceral adipose tissue is regarded as an independent risk factor for insulin resistance and atherosclerosis In addition, it was found, that visceral fat posed a greater risk for cardiac events than BMI [25].

It has been demonstrated that VFI is a very relevant way for assessing visceral adiposity and that it is an accurate and reliable method. Sahinturk *et al*., found a significantly negative association between VFI and Rentrop scores (r = -0.668, p=.001). Sahinturk Y. *et al*., observed that higher VFI in the presence of hypertension were independent predictors of poor CCC (OR= 1.955, 95% CI= 1.342 -2.848, p=.001) [26]. These findings are similar to our results. Our research also revealed a considerable negative association between VFI and Rentrop scores, and it also shown that VFI (> 9) may accurately estimate the chance of a poor development of CCC.

The results of current study made known that increased visceral adiposity is a significant and more accurate predictor of poor collateral development rather than BMI. Similar findings were made in a community-based cross-sectional study on obesity and myocardial infarction, where it was discovered that visceral fat index is more sensitive indicator in determining cardiovascular events in both genders, than body mass index and waist circumference [27].

Another predictor for poor CCC observed in our study population was hypertension. This data appears to be consistent with research' findings of Tayebjee *et al*., who found that individuals with coronary blockage and hypertension, had a higher risk of poor collateral formation [28].

Shu *et al*., observed that the chance of poor CCC increased as diastolic blood pressure reached >95 mmHg. The major blood supply to the myocardium occurs during diastolic phase of cardiac cycle, hence the authors hypothesized that diastolic blood pressure would affect the development of CCC by altering the coronary artery blood flow velocity during diastole. They also demonstrated that high diastolic blood pressure may result in occlusion of the coronary vessel that supplies blood to the CCC, which would reduce the perfusion of the CCC [29].

To enhance the reliability and reproducibility of the study results we have used standardized angiography technique and bioelectrical impedance analysis has been used to assess VFI. Robust statistical framework has also been used for evaluating the research question. There are few limitations of this study as this is a single centered study involved small sample size and further studies should be conducted on larger scale to conform the conclusion.

CONCLUSION

The current study concluded that greater VFI and concomitant hypertension considerably increase the likelihood of having poor CCC, therefore, visceral adiposity can be considered as a potential target for preventing poor collateral circulation in patients with established cardiac disease.

ETHICAL APPROVAL

The study was approved by ethical committee of KIBGE (Ref. No. KIBGE/ICE/06/30/05/16).

PATIENTS' CONSENT

All patients gave inform written consent prior to data collection.

AUTHORS' CONTRIBUTION

- **Shazia Nazar**: Main idea, Study design, Data collection, Manuscript writing, Literature review.
- **Erum Afaq**: Manuscript writing, Statistics, Data analysis.
- **Shayan Zufishan**: Manuscript writing, Literature review.
- **Nahida Baigam**: Manuscript writing, Data analysis, Final approval.
- **Ghazala Masood Farrukh**: Literature review, Data interpretation.
- **Sumera Mustafa**: Data collection, Data interpretation, Proof reading.

CONFLICT OF INTEREST

Declared none.

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