Journal of Cardiovascular Disease Research

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

A case control study to assess heart carotid pulse wave velocity and pulse transit time in triple vessel disease when compared to individuals without coronary artery disease

[1]DR.RAVINANDANA GOWDA H V, [2] Dr. Sriranga Rangashamaiah [3]Dr. NAGAMANI.A.C

[1 ,2] Assistant professor department of Cardiology Sri Jayadeva Institute of Cardiovascular Sciences & Research, Bengaluru

[3] Professor, Department of Cardiology Sri Jayadeva Institute of Cardiovascular Sciences & Research Bengaluru

Corresponding Author: [1]DR.RAVINANDANA GOWDA H V

Abstract:

Introduction: Coronary heart disease (CAD) is the leading cause of morbidity and mortality worldwide, with India being the top cause. The Asian subcontinent accounts for 20% of the world's population, but 60% of the cardiovascular disease burden is due to genetic predisposition and environmental factors. Arterial stiffness increases with aging, accelerated by cardiovascular risk factors like hypertension and diabetes. Endothelial dysfunction in coronary arteries affects arterial stiffness and potentially reversible in atherogenesis. Pressure wave velocity (PWV) is a useful index for assessing endothelial status and large artery stiffness. Large artery stiffening can increase systolic blood pressure, left ventricular afterload, and left ventricular hypertrophy, potentially limiting coronary perfusion.

Material and Methods: A prospective cross-sectional study was conducted at Sri Jayadeva Institute of Cardiovascular Sciences and Research Centre in Bangalore, India, from 1/6/2018 to 31/01/2019. Thirty patients with triple vessel disease and 30 age-matched controls underwent angiography at the institute. Echocardiography and Doppler evaluation assessed heart carotid pulse wave velocity and transit time. The study included patients with triple vessel disease and normal epicardial coronary arteries. The study excluded patients over 70 years old, having a heart rate above 100, having severe LV dysfunction below 30%, having chronic kidney disease, or having coronary artery stenosis. Demographic details, family history, medical history, clinical presentation, diagnostic tests, and blood tests were collected from patients. The study used descriptive and inferential statistical analysis, with continuous measurements presented as Mean ± SD and categorical measurements as Number (%).

Results: The study involved 30 cases and 30 controls, with the majority of patients aged 50-70 years and controls aged 40-60 years. The gender distribution was predominantly males. Diagnosis distribution was similar between the two groups, with patients with EA, UA, or TMT positive being more likely to undergo angiography. BMI (kg/m2) distribution was similar between the two groups, with the majority of patients still overweight or obese but having normal BMIs. Pulse transit time (PTT) was significantly lower in the triple vessel disease case group compared to the control group with normal coronary angiography. Pulse wave velocity (PWV) increased in the case group compared to the control group. In contrast to the control group, 66% of the patients in the case group showed some kind of LV dysfunction due to more patients with myocardial infarction. Pulse wave velocity was significantly lower in the case group, with 20% and 63% shorter. LMCA distribution was also similar between the two groups, with patients with LMCA having lower pulse transit time (20%) and (63% shorter) compared to the control group. The study highlights the importance of assessing patients' angiography and LMCA distribution in determining the appropriate treatment for patients with LV dysfunction.

Conclusion: Aortic PWV predicts future cardiovascular risk by accounting for known risk variables. It could be a valuable biomarker for intermediate risk, but randomized controlled studies are needed to prove its therapeutic utility before implementation.

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

Keywords: heart carotid pulse, pulse transit time, triple vessel disease, coronary artery disease

INTRODUCTION

Coronary heart disease (CAD) is the leading cause of morbidity and mortality, worldwide both in developing as well as developed countries.

In 2010, CAD was the leading cause of death globally, with India being the top cause. Mortality from cardiovascular disease is predicted to reach 23.4 million by 2030, with males more affected.[1,2]

Asian subcontinent accounts for 20% of the world's population, but 60% of cardiovascular disease burden is due to genetic predisposition and environmental factors, including hypertension, diabetes, dyslipidemia, smoking, unhealthy diet, low physical activity, alcohol consumption, and psychosocial stress.[3,4]

Arterial stiffness increases with aging, accelerated by cardiovascular risk factors like hypertension and diabetes, and predicts adverse cardiovascular outcomes like mortality and stroke.[5,6] Endothelial dysfunction in coronary arteries is a systemic vascular abnormality, affecting arterial stiffness and potentially reversible in atherogenesis.

Numerous methods assess endothelial status and large artery stiffness, including pressure wave velocity (PWV). PWV is a useful and robust index of arterial stiffness, as it doesn't rely on pressure-volume, pressure-diameter, or pressure-strain relationships. Aortic PWV helps differentiate patients at low and high risk of adverse cardiovascular outcomes. Carotid-femoral PWV is considered the "gold standard" for arterial stiffness and subclinical target organ damage in hypertensive patients.[4]

PWV is measured using tonometry, impedance, or Doppler. It is determined by a pulse wave generating during systole, dilates the aortic wall, and reflects off the aorta's bifurcation, creating a second wave. The difference between the first and reflected waves is related to the aorta's stiffness. The R-wave of the electrocardiogram (ECG) is better used as the starting point due to its proximity to the aortic valve opening.[7,8]

Large artery stiffening increases systolic blood pressure, left ventricular afterload, and left ventricular hypertrophy, potentially limiting coronary perfusion.[9,10]

AIMS AND OBJECTIVES

- To study the correlation between heart carotid pulse wave velocity and pulse transit time in patients who undergo coronary angiogram and are diagnosed to have triple vessel disease when compared to age and sex matched controls without any epicardial coronary artery disease.

MATERIALS AND METHODS

A prospective cross-sectional study at Sri Jayadeva Institute of Cardiovascular Sciences and Research Centre, Bangalore, collected demographic, medical, diet, substance use, and hospitalization data from patients.

Selection of Study Population

Doppler study is conducted on patients with triple vessel disease diagnosed during coronary angiogram at Sri Jayadeva Institute of Cardiovascular Sciences & Research from 1/6/2018 to 31/01/2019.

Study design

It is a prospective study of patients who were admitted to Sri Jayadeva hospital

SAMPLE SIZE - 60

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

STUDY PERIOD - 6 months

Thirty patients with triple vessel disease and 30 age-matched controls underwent angiography at Sri Jayadeva Institute of Cardiology. Echocardiography and Doppler evaluation assessed heart carotid pulse wave velocity and transit time.

The PWV method involves dividing pulse wave propagation distance by transit time between two waveforms. The R-wave of the electrocardiogram (ECG) is used as a starting point due to motion artifacts and noise contamination.

The markers of arterial stiffness, hcPWV and PTT, are measured 1 cm from the right carotid bifurcation using an I33 Philips ultrasound device under ECG gating. The measurements are taken from the peak of the R wave to the foot of the carotid upstroke on pulse Doppler.

Inclusion Criteria

1)Coronary angiogram demonstrating triple vessel disease for the case group.

2)Coronary angiogram demonstrating normal epicardial coronary arteries for control groups.

Exclusion Criteria

1)Age>70 years

2)Heart rate>100

3)Severe LV dysfunction EF<30%

4)Chronic kidney disease

5)Carotid artery stenosis

Study Procedures

Data collection in hospital involved surveying patients for coronary angiograms, focusing on prepared coronary artery fibroblasts.

The following information was collected from the patients who satisfied the inclusion criteria and were enrolled in the study.

- **Demographic details**: The demographic details of the patient such as age (in years), height (in centimeters), weight (in kilograms), and Body mass index was collected.
- **Family History**: Occurrence of CAD, Hypertension, and Diabetes Mellitus among family members was recorded.
- **Medical History**:

❖ The occurrence of Diabetes Mellitus, Hypertension, Chronic kidney disease, Chronic obstructive pulmonary disease, Hypothyroidism, Cerebrovascular accidents were recorded.

❖ Details of clinical presentation were recorded.

❖ List of diagnostic tests performed during the hospital stay such as ECG, Echocardiography and coronary angiography.

❖ List of blood tests performed during the hospital stay as per the standard practice (e.g. cardiac enzymes - CKMB and cardiac Troponin T), Hemoglobin, Total leucocyte count, differential count, platelets, serum creatinine, blood urea and lipid profile.

Statistical Analysis

Statistical Methods: The study uses descriptive and inferential statistical analysis, with continuous measurements presented as Mean ± SD and categorical measurements as Number (%). Significance is assessed at a 5% level. The assumptions include normally distributed dependent variables, random sample draws, and independent cases of samples.

Student t test was used to determine study parameters' significance in inter-group analysis, while Leven's test assessed variance homogeneity.

Chi-square/Fisher Exact test is utilized for categorical and non-parametric analysis of study parameters in qualitative data, particularly when cell samples are small.

Significant figures

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

+ Suggestive significance (P value: 0.05<P<0.10)
* Moderately significant (P value:0.01<P ≤ 0.05)
** Strongly significant (P value : P≤0.01)

Statistical software: The Statistical software namely SPSS 18.0, and R environment ver.3.2.2 were used for the analysis of the data and Microsoft word and Excel have been used to generate graphs, tables etc**.**

Ethical declaration

Study received ethical approval from Clinical Ethics Committee, Sri Jayadeva Institute of Cardiology, Bangalore, and Rajiv Gandhi University of Health Sciences, and patient consent was obtained before assessment.

RESULTS AND OBSERVATIONS

Study design: A Comparative case-Controls Clinical study 30 casese and 30 controls were selected based on the angiography.

Table 1: Age distribution of patients studied

Age in years	Cases	Controls	Total
<40	1(3.3%)	1(3.3%)	2(3.3%)
40-50	5(16.7%)	10(33.3%)	15(25%)
51-60	10(33.3%)	11(36.7%)	21(35%)
61-70	14(46.7%)	8(26.7%)	22(36.7%)
Total	30(100%)	30(100%)	60(100%)
Mean ± SD	57.03±8.54	54.20±7.64	55.62±8.16

Samples are age matched with P=0.181, student t test

Most patients in the case group were 50-70 years old, while controls were younger and 40-60 years old.

Table 2: Gender distribution of patients studied

Gender	Cases	Controls	Total
Female	5(16.7%)	12(40%)	17(28.3%)
Male	25(83.3%)	18(60%)	43(71.7%)
Total	30(100%)	30(100%)	60(100%)

P=0.045*, significant, Chi-Square test

In both case and control group the gender distribution was predominantly males when compared to females

Table 3: Diagnosis distribution in two groups of patients studied

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

Diagnosis	Cases	Controls	Total
UA	1(3.3%)	8(26.7%)	9(13.3%)
AWMI	7(23.3%)	0(0%)	7(11.7%)
EA,TMT -	0(0%)	7(23.3%)	7(11.7%)
IWMI	6(20%)	1(3.3%)	7(11.7%)
NSTEMI	5(16.7%)	2(6.7%)	7(11.7%)
EA,TMT+	3(10%)	3(10%)	6(10%)
CHEST PAIN	0(0%)	4(13.3%)	4(6.7%)
CSA	0(0%)	2(6.7%)	2(3.3%)
EV IWMI	2(6.7%)	0(0%)	2(3.3%)
IW+PWMI	1(3.3%)	1(3.3%)	2(3.3%)
UA,TMT+	2(6.7%)	0(0%)	2(3.3%)
DCM	0(0%)	1(3.3%)	1(1.7%)
HLWMI	1(3.3%)	0(0%)	1(1.7%)
IMWI	1(3.3%)	0(0%)	1(1.7%)
IW+RVMI	1(3.3%)	0(0%)	1(1.7%)
VT	0(0%)	1(3.3%)	1(1.7%)
Total	30(100%)	30(100%)	60(100%)

(AWMI-anterior, IWMI-inferior, PWMI-posterior, HLWMI- high lateral wall myocardial infarction, NSTEMI-non ST segment elevation MI, EA- effort angina, UA-unstable angina, CSA- chronic stable angina, TMT - treadmill test, DCM - dilated cardiomyopathy, VT-ventricular tachycardia)

Most patients underwent angiography for acute myocardial infarction, evolved MI, or unstable angina. Patients with EA, UA, or TMT positive were more likely to undergo angiography. Patients with DCM or VT were more likely to be TMT negative.

Table 4: BMI (kg/m^2) distribution in two groups of patients studied

BMI (kg/m^2)	Cases	Controls	Total
<18.5	0(0%)	0(0%)	0(0%)
18.5-25	21(70%)	21(70%)	42(70%)
25-30	6(20%)	9(30%)	15(25%)
>30	3(10%)	0(0%)	3(5%)
Total	30(100%)	30(100%)	60(100%)

P=0.186, Not Significant, Fisher Exact Test

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

In both the case and control groups, the majority of patients were still overweight or obese but had normal BMIs (body mass index range).

Table 5: Comparison of Height/Weight /BMI in two groups of patients studied

variables	Cases	Controls	Total	P value
Height (cm)	167.70±7.25	168.17±7.30	167.93±7.22	0.805
Weight (kg)	66.73±9.15	66.87±6.00	66.80±7.67	0.947
BMI (kg/m^2)	23.88±4.06	23.69±2.15	23.78±3.22	0.823

Student t test(Two tailed, independent)

Height, weight, and BMI did not significantly differ between the case and control groups.

Table 6: Pulse transit time/Pulse wave/EF in two groups of patients studied

variables	Cases	Controls	Total	P value
Pulse transit time	106.53±1.91	110.77±2.45	108.65±3.05	<0.001**
Pulse wave velocity	492.90±3.44	488.60±1.45	490.75±3.40	<0.001**
EF%	50.27±6.99	56.33±3.86	53.30±6.38	<0.001**

Student t test(Two tailed, independent)

Pulse transit time (PTT) was significantly lower in the triple vessel disease case group compared to the control group with normal coronary angiography. Additionally, pulse wave velocity (PWV) increased in the case group compared to the control group.

Table 7: EF distribution in two groups of patients studied

EF	Cases	Controls	Total
41-45	11(36.7%)	2(6.7%)	13(21.7%)
46-50	5(16.7%)	1(3.3%)	6(10%)
51-55	3(10%)	2(6.7%)	5(8.3%)
56-60	11(36.7%)	25(83.3%)	36(60%)
Total	30(100%)	30(100%)	60(100%)

P<0.001**, Significant, Fisher Exact Test

(EF-ejection fraction)

In contrast to the control group, where 83% of the patients had normal LV systolic function, 66% of the patients in the case group showed some kind of LV dysfunction since there were more patients with myocardial infarction.

Table 8: Pulse Wave distribution in two groups of patients studied

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

Pulse Wave velocity	Cases	Controls	Total
<490	6(20%)	20(66.7%)	26(43.3%)
>490	24(80%)	10(33.3%)	34(56.7%)
Total	30(100%)	30(100%)	60(100%)

P<0.001**, Significant, Chi-Square Test

Patients in the case group had pulse wave velocity >490cm/s, while in the control group, 33% had PWV above 490cm/s, 20% had PWV below 490cm/s.

Table 9: Pulse transit time distribution in two groups of patients studied

Pulse transit time	cases	controls	Total
100-104	6(20%)	0(0%)	6(10%)
105-108	19(63.3%)	6(20%)	25(41.7%)
109-113	5(16.7%)	19(63.3%)	24(40%)
114-117	0(0%)	1(3.3%)	1(1.7%)
Total	30(100%)	30(100%)	60(100%)

P<0.001**, Significant, Fisher Exact Test

Case group had lower pulse transit time (20%) and (63%) compared to control groups, with 20% and 63% shorter.

Table 10: LMCA distribution in two groups of patients studied

LMCA	Cases (n=30)	Controls (n=30)	Total (n=60)
No	21(70%)	30(100%)	51(85%)
Yes	9(30%)	0(0%)	9(15%)
● D60	1(3.3%)	0(0%)	1(1.7%)
● D30	2(6.7%)	0(0%)	2(3.3%)
● M40	2(6.7%)	0(0%)	2(3.3%)
● D40	1(3.3%)	0(0%)	1(1.7%)
● D70	1(3.3%)	0(0%)	1(1.7%)
● D80	1(3.3%)	0(0%)	1(1.7%)
● DIFF 90	1(3.3%)	0(0%)	1(1.7%)

P=0.002**, Significant, Fisher Exact Test

(LMCA-left main coronary artery, DIFF-diffuse, D-distal,M-mid)

The left main coronary artery disease (LMCA) affected 21 individuals in the case group (70%) in some way, and they were more likely to have considerably shorter pulse transit times and faster pulse wave velocities (P value 0.001).

Table11: LAD distribution in two groups of patients studied

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

LAD	Cases	Controls	Total
No	0(0%)	30(100%)	30(50%)
Yes	30(100%)	0(0%)	30(50%)
Total	30(100%)	30(100%)	60(100%)

P<0.001**, Significant, Chi-Square Test

All patients in the case group exhibited substantial LAD disease of some kind, with proximal and mid segment lesions predominating.

Table 12: RAMUS distribution in two groups of patients studied

RAMUS	Cases	Controls	Total
No	29(96.7%)	30(100%)	59(98.3%)
Yes	1(3.3%)	0(0%)	1(1.7%)
Total	30(100%)	30(100%)	60(100%)

P=1.000, Not Significant, Fisher Exact Test

Only one of the patients in the case group had a ramus intermedius branch.

Table 13: Left Circumflex distribution in two groups of patients studied

Left Circumflex	Cases	Controls	Total
No	0(0%)	30(100%)	30(50%)
Yes	30(100%)	0(0%)	30(50%)
Total	30(100%)	30(100%)	60(100%)

P<0.001**, Significant, Chi-Square Test

Left circumflex involved in all cases, dominant circumflex in 7%.

Table 14: RCA distribution in two groups of patients studied

RCA	Cases	Controls	Total
N	0(0%)	30(100%)	30(50%)
ND diseased	2(6.7%)	0(0%)	2(3.3%)
D diseased	28(93.3%)	0(0%)	28(46.7%)
Total	30(100%)	30(100%)	60(100%)

P<0.001**, Significant, Fisher Exact Test

(RCA-right coronary artery,N-normal.D-dominant, ND- non dominant)

RCA was involved in all patients in the case group. RCA was dominant in 93% of the patients

Table 15: HbA1c% distribution in two groups of patients studied

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

HbA1c%	Cases	Controls	Total
<6	7(23.3%)	27(90%)	34(56.7%)
6-9	16(53.3%)	3(10%)	19(31.7%)
>9	7(23.3%)	0(0%)	7(11.7%)
Total	30(100%)	30(100%)	60(100%)

P<0.001**, Significant, Fisher Exact Test

(HbA1c- glycosylated hemoglobin A1c)

Most case group patients had poor glycemic control compared to the control group.

Table 16: Lipid parameters distribution in two groups of patients studied

variables	Cases (n=30)	Controls (n=30)	Total (n=60)	P value
Total Cholesterol (mg/dl)				
● <200	21(70%)	29(96.7%)	50(83.3%)	0.012*
● 200-280	8(26.7%)	1(3.3%)	9(15%)	
● >280	1(3.3%)	0(0%)	1(1.7%)	
HDL (mg/dl)				
● <35	26(86.7%)	3(10%)	29(48.3%)	<0.001**
● 35-60	3(10%)	27(90%)	30(50%)	
● >60	1(3.3%)	0(0%)	1(1.7%)	
LDL (mg/dl)				
● <70	9(30%)	5(16.7%)	14(23.3%)	0.360
● 70-190	21(70%)	25(83.3%)	46(76.7%)	
● >190	0(0%)	0(0%)	0(0%)	
TG (mg/dl)				
● <150	9(30%)	28(93.3%)	37(61.7%)	<0.001**
● 150-500	21(70%)	2(6.7%)	23(38.3%)	
● >500	0(0%)	0(0%)	0(0%)	

Chi-Square/Fisher Exact Test

(HDL- high density , LDL- low density lipoprotein cholesterol, TG- triglycerides)

Case group patients had higher total cholesterol, LDL, TG, and lower HDL levels compared to control group (P <0.001).

Table 17: Comparison of lipids in two groups of patients studied

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

variables	Cases	Controls	Total	P value
Total Cholesterol (mg/dl)	168.40±52.08	146.37±24.58	157.38±41.88	0.041*
HDL (mg/dl)	30.02±10.66	38.32±3.19	34.17±8.85	<0.001**
LDL (mg/dl)	93.20±34.69	82.93±15.14	88.07±27.04	0.143
TGL (mg/dl)	214.63±85.96	103.57±25.00	159.10±84.12	<0.001**

Student t test(Two tailed, independent)

The table gives the median distribution of the total cholesterol, HDL, LDL, TG in the case and control group.

Table 18: Hemoglobin (g/dl) distribution in two groups of patients studied

Hemoglobin (g/dl)	Cases	Controls	Total
<12	3(10%)	1(3.3%)	4(6.7%)
12-16	24(80%)	28(93.3%)	52(86.7%)
>16	3(10%)	1(3.3%)	4(6.7%)
Total	30(100%)	30(100%)	60(100%)

P=0.387, Not Significant, Fisher Exact Test

Both the case and control groups' haemoglobin levels were within normal limits, with 6.7% of the patients having anaemia (12 gm/dl for men and 11 gm/dl for women).

Table 19: RFT distribution in two groups of patients studied

Sr. Creatinine	Cases	Controls	Total
<1	12(40%)	26(86.7%)	38(63.3%)
1-1.2	15(50%)	4(13.3%)	19(31.7%)
>1.2	3(10%)	0(0%)	3(5%)
Total	30(100%)	30(100%)	60(100%)

P<0.001**, Significant, Fisher Exact Test

All patients had normal renal function tests, including serum creatinine and blood urea nitrogen.

Table 20: ELECTROLYES distribution in two groups of patients studied

S.r Electrolytes	Cases	Controls	Total
Normal	30(100%)	30(100%)	60(100%)
Abnormal	0(0%)	0(0%)	0(0%)
Total	30(100%)	30(100%)	60(100%)

P=1.000, Not Significant, Fisher Exact Test

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

All patients in both groups had normal serum levels of sodium, potassium, and chlorides.

Table 21: Other Investigations

	Cases (n=30)	Controls (n=30)	Total (n=60)	P value
LFT	0(0%)	0(0%)	0(0%)	1.000
TFT	0(0%)	0(0%)	0(0%)	1.000
CAROTID DOPPLER	0(0%)	0(0%)	0(0%)	1.000

Chi-Square/Fisher Exact Test

(LFT-liver function test, TFT- thyroid function test/thyroid profile)

All patients in the case and control groups had normal liver function tests, thyroid function tests, and carotid doppler results.

DISCUSSION

Large artery stiffening increases left ventricular afterload and hypertrophy, leading to impaired coronary perfusion and unbalanced coronary perfusion/myocardial demand equilibrium. Arterial stiffness may integrate cumulative damage from CV risk factors over time. Aortic pulse wave velocity (aPWV) may represent a surrogate end point, indicating patients' traditional CV risk factors translating into real risk. Arterial stiffness is a promising biometric for predicting major cardiovascular events and death.[11,12]

Aortic stiffness can impair pressure buffering abilities, leading to high pulsatile pressure and increased flow into the coronary and brain microcirculation. This increases vascular damage and small-vessel damage in the kidneys, potentially causing albuminuria.

In our investigation, coronary artery disease was associated with an increase in hcPWV velocity (P 0.001), with greater velocities seen in individuals with triple vascular disease and lower velocities in normal epicardial coronary angiography. In contrast to the control group, which had longer pulse transit times, there was a reciprocal reduction in pulse transit time in the case group in the presence of triple vessel disease (P value 0.001).

hcPWV does not correlate with atherosclerosis severity or grade, but is increased in left main disease or multi vessel disease. Patients with this condition are more likely to have myocardial infarction, LV dysfunction, and abnormal lipid profiles, including low HDL and high LDL levels. They are also more diabetic and have poor glycemic controls compared to the control group.

In our study, triple vascular disease patients had higher pulse wave velocities and shorter pulse transit times, which can be utilised as a non-invasive predictor of triple vessel disease's most likely occurrence.

Coutinho et al. found that aPWV was independently associated with subclinical target organ damage in coronary, cerebral, and peripheral arterial beds, and predicted coronary and lower extremity atherosclerosis, marking the first study to report simultaneous associations.[13]

Terai et al.[14] found that aPWV predicted myocardial infarction or stroke in 676 patients with essential hypertension. The study used objective measures of vascular disease and assessed the association with subclinical large and small vessel target organ damage.

In a 2010 meta-analysis research, information on 15,877 individuals was examined. According to this analysis, for people with high vs. low aortic PWV, the combined relative risks for all CV events, CV mortality, and all-cause mortality were 2.26 (95% CI 1.89 to 2.70, 14 studies), 2.02 (95% CI 1.68 to 2.42, 10 studies), and 1.90 (95% CI 1.61 to 2.24, 11 studies), respectively[15].

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

A systematic review and meta-analysis of 15,877 subjects found that an increase in aortic PWV of 1 m/s or 1 SD increases the risk of coronary artery disease by almost twice as much. This risk is significantly associated with adverse cardiovascular events and mortality, as well as all-cause mortality. The predictive value of aPWV was stronger in younger subjects, possibly due to the "healthy survivor effect" and other risk factors attenuating its effects at older ages.[15]

Chunyue et al's[16] study found that rcPWV (R wave carotid pulse wave velocity) could be a useful index of atherosclerosis in hypertensive patients. They found that thicker carotid intimal thickness (CIMT) had a positive association with increased plaque prevalence. RcPWV was significantly associated with CIMT in thickened CIMT hypertensive patients, independently correlated with CIMT after adjusting for clinical confounders. This suggests that CIMT and hcPWV correlate with each other in thickened CIMT hypertensive patients.

Signals were collected from 28 participants in a research to evaluate aortic and arterial pulse wave velocity (PWV) in coronary heart disease (CHD) patients with healthy young volunteers. Coronary angiography confirmed severe coronary heart disease. The Arteriograph was used to measure the aortic PWV. The main finding of this study's four arterial PWVs was that individuals with CHD had higher aortic PWVs than healthy people.Study results, however, indicated that there was no statistically significant difference between CHD patients with different grades of atherosclerosis in terms of aortic and arterial PWV.

American Heart Association's 2015 statement emphasizes arterial stiffness importance for predicting future cardiovascular events beyond risk factors.[17]

aPWV attenuation may benefit novel risk reduction strategies, as attenuation is linked to improved survival. Current drugs don't lower aPWV in blood pressure-independent ways, but novel agents targeting elastic fiber cross-linking or calcification may offer some benefits.[18]

The study's limited statistical power suggests future studies using uniform techniques and analytical approaches to improve comparison and implementation of aortic PWV in clinical practice.

CONCLUSION

By accounting for known risk variables, aortic PWV predicts future cardiovascular risk and enhances risk categorization. It may be possible to employ aPWV as a valuable biomarker to enhance cardiovascular risk prediction for people at intermediate risk because it is now consistently and simply monitored. Coronary artery disease can be detected by the speed of the heart's carotid pulse waves.However, randomised controlled studies employing aPWV to direct risk stratification and/or therapy are necessary to give persuasive proof that this approach has therapeutic utility before its implementation may be advised.

LIMITATIONS OF THE STUDY

The sample size of our study is too small to extend any findings to the broader population, which is one of its drawbacks. Other restrictions include the user-dependent evaluation of PWV and inconsistent definitions.

BIBLIOGRAPHY

1. Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, Abraham J, Adair T, Aggarwal R, Ahn SY, et al. "Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010". Lancet. 380 (9859): 2095–2128.

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

2. Finegold JA, Asaria P, Francis DP . "Mortality from ischaemic heart disease by country, region, and age: Statistics from World Health Organisation and United Nations". International Journal of Cardiology. 168 (2): 934–45.
3. Liu, Y., Wang, D. H., Liao, X. X. and Xu, M. G. Increased brachial-ankle pulse wave velocity is associated with impaired endothelial function in patients with coronary artery disease.Chin. Med. J., 2006, 119, 1866–1870.
4. Yusuf S, Hawken S, Ounpuu S, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet. 2004;364:937–952.
5. Ben-Shlomo Y, Spears M, Boustred C, et al. Aortic pulse wave velocity improves cardiovascular event prediction: an individual participant meta-analysis of prospective observational data from 17,635 subjects. J Am CollCardiol. 2014;63:636–646.
6. Ishisone T, Koeda Y, Tanaka F, et al. Comparison of utility of arterial stiffness parameters for predicting cardiovascular events in the general population. Int Heart J. 2013;54:160–165.
7. Nagasaki T, Yamada S, Wakita Y, et al. Clinical utility of heart carotid pulse wave velocity in healthy Japanese subjects. Biomed Aging Pathol. 2011;1:107–111.
8. Li C, Xiong H, Wu W, et al. The relationship between heart-carotid pulse transit time and carotid intima-media thickness in hypertensive patients. J Hum Hypertens. 2015;29:663–668.
9. VanPopele, N. and Grobbee, D. Association between arterial stiffness and atherosclerosis: the Rotterdam study. Stroke, 2001, 32, 454–460
10. Vlachopoulos C, Aznaouridis K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with arterial stiffness: a systematic review and meta-analysis. J Am CollCardiol. 2010;55:1318–1327.
11. Laurent S, Cockcroft J, Van Bortel L, et al. European Network for Non-invasive Investigation of Large Arteries. Expert consensus document on arterial stiffness: methodological issues and clinical applications. Eur Heart J 2006;27:2588 – 605.
12. Zieman SJ, Melenovsky V, Kass DA. Mechanisms, pathophysiology,and therapy of arterial stiffness. Arterioscler Thromb Vasc Biol 2005;25:932– 43.
13. 43)Coutinho T, Turner S, Iftikhar J. Kullo.Aortic Pulse Wave Velocity Is Associated With Measures of Subclinical Target Organ Damage.JACC: VOL. 4, NO. 7, 2011
14. Mitchell GF. Arterial stiffness and wave reflection in hypertension: pathophysiologic and therapeutic implications. Curr Hypertens Rep 2004;6:436–41.
15. Vlachopoulos C, Aznaouridis K, Stefanadis C. Prediction of cardiovascular events and all-cause mortality with arterial stiffness: a systematic review and meta-analysis. J Am Coll Cardiol 2010;55:1318–27.
16. Chunyue Li,Huahua Xiong, Sandeep Pirbhulal.Heart-Carotid Pulse Wave Velocity a Useful Index of Atherosclerosis in Chinese Hypertensive Patients Medicine.Volume 94, Number 51, December 2015
17. Townsend RR, Wilkinson IB, Schiffrin EL, Avolio AP, et al. Recommendations for Improving and Standardizing Vascular Research on Arterial Stiffness: A Scientific Statement from the American Heart Association. Hypertension. 2015 July 9; 66:698-722. http://hyper.ahajournals.org/content/66/3/698.long.

ISSN: 0975-3583, 0976-2833 VOL14, ISSUE 07, 2023

18. Kass D, Shapiro EP, Kawaguchi M, et al. Improved arterial compliance by a novel advanced glycation end-product crosslink breaker. Circulation 2001;104:1464–70.