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AN OBSERVATIONAL STUDY TO COMPARE CAROTID FEMORAL PULSE WAVE VELOCITY IN SUBJECTS WITH OR WITHOUT METABOLIC SYNDROME IN JODHPUR CITY

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ABSTRACT **Background-** Metabolic syndrome is an assemblage of physiological and biochemical abnormalities characterized by diabetes, central obesity, abnormal cholesterol and triglyceride levels and hypertension. "Arterial Stiffness" refers to the physical characteristics of blood arteries, including their distensibility capacity, compliance, and elasticity. Arterial stiffness caused by artery stenosis is a standalone predictor of cardiovascular morbidity and mortality. **Materials and Methods**: Cross sectional analytical observational study including 80 subjects, 40 metabolic syndromes and 40 normal subjects aged 35-65 years (53.05 ± 9.20) of either gender. Anthropometric parameters (weight, height, waist circumference, hip circumference, waist to hip ratio, body mass index) and cardiovascular parameters (blood pressure and resting heart rate) were measured. Fasting Plasma Glucose, HbA1c and lipid profile tests (total cholesterol, triglycerides, HDL-c were performed by Robonik automatic biochemical auto-analyzer. Arterial stiffness was measured by pulse wave velocity using Periscope based on oscillometric technique. **Results:** T-test result depicts significant increase in C-F PWV for MS group ($p<0.05$) shows raised pulse wave velocity is an indicator of increased CVD risk.

KEYWORDS : Metabolic Syndrome, Pulse Wave Velocity, Arterial Stiffness, Visceral Adiposity Index

INTRODUCTION

Coronary artery disease (CAD) prevails as a major causative factor of death and disability, globally. The pathophysiology underneath CAD attributes to Atherosclerosis, a complex process governed by multiple factors. Hypertension, diabetes mellitus, dyslipidemia, cigarette smoking and obesity, significantly contribute to the development of atherosclerosis and CAD.[1] **The Metabolic Syndrome (Syndrome X)**, is a state of chronic low-grade inflammation due to complex interplay between genetic and environmental factors. Overall, it predicts a two-fold increase in the risk of CVD mortality, stroke and a 1.5-fold increase in risk of all-cause mortality.[2]

Metabolic syndrome, a pre-atherosclerotic state, includes multiple factors, such as abdominal obesity, arterial hypertension, dyslipidemia and hyperglycemia. MS not only accelerates central arterial aging but is also an independent predictor of cardiovascular events in elderly people.[3]

With age advancement, alterations in arterial structure and function, progressively lead to arterial stiffening along with other deteriorations like Hypertension. **Arterial stiffness** reflects structural damage of the arterial wall by gradual fragmentation and loss of elastin fibers and accumulation of stiffer collagen fibers in the large arteries followed by functional alterations of vascular smooth muscle tone.[4,5] Essentially, arterial stiffness can predict adverse cardiovascular events beyond classic risk factors, in patient groups with different diseases and in the general population. Therefore, the assessment of arterial stiffness is highly advantageous in early detection and prevention of vascular disease.[6] The pulse wave travels through the artery system, and its speed is inversely proportional to the arterial wall's distensibility: the higher the velocity, the lower the vascular distensibility.[7]

MATERIALS AND METHODS-

Research work was commenced after the approval of the synopsis by the ethical committee, SNMC, Jodhpur.

Study Design: Cross sectional analytical observational study.

Study Area: Department of General Medicine, Department of Biochemistry and Department of Physiology, Dr. S.N. Medical College, Jodhpur, Rajasthan.

Study Duration: From July 2021 to July 2022.

Sample size: 80 subjects.

Sample Size Calculation: The sample size was calculated at alpha error 0.05 and study power 90% using the formula for hypothesis testing for two population mean -

$$n = \frac{2 \times \left(Z_{1-\alpha/2} + Z_{1-\beta}\right)^2 \times \sigma^2}{(\mu_1 - \mu_2)^2}$$

n = Sample size

($Z_{1-\alpha/2}$) = Standard normal deviate for alpha error (taken as 1.96 for alpha error 0.05)

($Z_{1-\beta}$)= Standard normal deviate for beta error (taken as 1.28 for 90% study power)

σ = pooled variance of pulse wave velocity of the two population. As it is not known, it is replaced by S_p^2 –

$$S_p^2 = \frac{S_1^2 + S_2^2}{2}$$

Sample size was calculated to be a minimum of 37 subjects in each group, which then round off to 40 subjects in each group.

Inclusion Criteria (With MetS)

1. Individuals aging between 35 to 65 years, of either gender.
2. Obese individual with below mentioned two or three criteria was considered as **Metabolic Syndrome** subjects.
   Obese individuals (≥ 90 cm in males and ≥ 80 cm in females) with high Triglyceride level (≥ 150 mg/dl), low HDL-c (<40 mg/dl in males and <50 mg/dl in females), high blood pressure (≥ 130 mm Hg Systolic or ≥ 85 mmHg Diastolic) and fasting plasma glucose (> 100 mg/dl)

Inclusion Criteria (Without MetS)

1. Individuals aging between 35 to 65 years, of either gender.
2. Obese individuals (≥ 90 cm in males and ≥ 80 cm in females) with normal lipid profile, normal blood pressure level and normal fasting blood sugar level was considered as **Non-Metabolic Syndrome** subjects.

Exclusion Criteria

1. Pregnancy, postpartum period, lactating females, hormonal replacement therapy.
2. Subjects with chronic diseases like congestive heart failure, coronary artery disease and chronic kidney disease.
3. Use of any antipsychotic drug, antiepileptic drug and weight reduction medications.

Procedure Methodology

In the present study, anthropometric measurements (weight, height, waist circumference, hip circumference, waist-to-hip ratio, BMI), cardiovascular parameters (blood pressure, resting heart rate), Fasting Plasma Glucose, HbA1c and lipid profile tests (total cholesterol,

triglycerides, HDL-c were performed by Robonik automatic biochemical auto-analyzer. Low density lipoprotein (LDL) was calculated as follows: -

Low Density Lipoprotein (LDL) cholesterol

$$LDL = TC - HDL - LDL$$

Visceral Adiposity Index - VAI is a model of adipose distribution with fat function. It was calculated for both male and female by the following formula –

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

$$\text{Females: VAI} = \left(\frac{WC}{36.58 + (1.89 \times BMI)}\right) \times \left(\frac{TG}{0.81}\right) \times \left(\frac{1.52}{HDL}\right)$$

Arterial Stiffness was assessed by PERISCOPE [RMS India]. PERISCOPE is a computer-based pulse waveform analysis system that simultaneously calculates vital parameters like **Pulse Wave Velocity (PWV)**, **Arterial Stiffness Index (ASI)**, **Central Aortic Pressure Values**, and **Ejection Slopes** using non-invasive blood pressure measurements from all four limbs and ECG waveforms. We collected right and left brachial ankle (BaPWV), carotid femoral (CF PWV) pulse wave velocity and their reference value for age from report generated by this device.

Statistical analysis- Data were expressed as mean ± SD. Statistic calculations were done using Analysis Tool Pak Add-in in Microsoft Office excel® 2016 and IBM ® SPSS ® Statistics Subscription. Data were compared in subjects with or without metabolic syndrome groups by independent Student-T test.

OBSERVATIONS AND RESULTS

Table 1- Comparison Of C-F PWV In Subjects With Or Without Metabolic Syndrome

Parameters	Group (N=80)	Mean	Std. Deviation	Std. Error Mean
C-F PWV (cm/sec)	Control Group (N=40)	893.37	158.38	25.04
	Study Group (N=40)	1131.91	212.66	33.62

Note: 1. Control Group= Without Metabolic Syndrome 2. Study Group= With Metabolic Syndrome

Table 2- Independent Student T Test

Parameters		Levene's Test for Equality of Variances		t-test for Equality of Means					95% Confidence Interval of the Difference	
C-F PWV (cm/sec)		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
	Equal variances assumed	1.895	0.173	-5.689	78	0.000	-238.540	41.926	-322.01	-155.07
	Equal variances not assumed			-5.689	72.08	0.000	-238.540	41.926	-322.11	-154.97

*NS = Not Significant (p>0.05), S = Significant (p < 0.05)

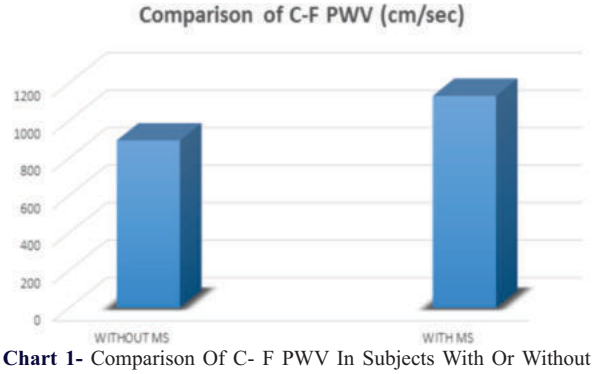

Chart 1- Comparison Of C- F PWV In Subjects With Or Without Metabolic Syndrome

As seen in above Table: - 1 & Chart: - 1 C-F PWV is higher is study group. T test analysis shows significant p-value for C-F PWV in study group. **(p<0.05)**

DISCUSSION-

Metabolic Syndrome is a well-known central arterial ageing accelerator and is linked to a higher risk of cardiovascular events.[8] The main cause of arterial stiffness, which results from structural and functional changes to the vascular smooth muscle tone and the arterial wall, is the gradual fragmentation and loss of elastin fibers as well as the build-up of stiffer collagen fibers in the larger arteries.[9]

Metabolic syndrome subjects presented higher values for Carotid Femoral Pulse Wave velocity. Carotid Femoral Pulse Wave velocity was significantly increased in metabolic syndrome subjects as compared to subjects without metabolic syndrome. (p<0.05) [Table No. 1]

One of the potential causes of elevated aPWV in persons with MetS is arterial structural alterations brought on by long-term hyperglycemia, increase in the expression of angiotensin II (AT II) and the local activity of the renin-angiotensin-aldosterone system. Through the AT1 receptor, AT II plays a significant role in VSMC proliferation and collagen formation. The development of arterial stiffness may be caused by these alterations in the vascular wall. Impaired glucose tolerance increases non-enzymatic advanced glycation end products on the vascular matrix proteins, which causes an increase in the production of collagen fibers in the vascular wall and ultimately,[10] the production of oxidative stress or the interaction of membrane receptors, which in turn, causes the inflammatory response.[11]

Endothelial dysfunction, inflammation, and oxidative stress are additional characteristics of arterial stiffness associated with MetS.[12] Evidence suggests that the MetS's three main components fat accumulation, dyslipidemia, and insulin resistance are linked to an increase in oxidative stress and, as a result, a rise in lipid peroxidation.[13] Several studies have also reported that metabolic syndrome and all its components are associated with increased arterial stiffness, leading to increased aPWV. Egidija Rinkuniene et al concluded higher TG are associated with C-F PWV in patients with MS as the test results revealed significantly increased in C-F PWV for the study (MS+HTG) group.[14]

Gerard Blasco et al concluded the association between MetS and its component with carotid femoral pulse wave velocity (cf PWV) in asymptomatic subjects without diabetes. They observed that C-F PWV is significantly increased in middle-aged subjects with MetS than in subjects without MetS. (p<0.05)[15]

CONCLUSION-

Pulse wave velocity is a well-established and widely used index of arterial stiffness for non-invasive evaluation of subclinical atherosclerotic changes. Raised arterial stiffness or decreased vascular compliance is indicated by an increase in PWV. The use of PWV to assess the vascular phenotype allows for the quantification of subclinical vascular disease even before the onset of systemic hypertension. The T-test result depicts significant increase in C-F PWV for study group (p<0.05) shows raised pulse wave velocity is an indicator of increased CVD risk.

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