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ASSOCIATION OF VISCERAL AND GENERAL OBESITY INDICATORS WITH PULSE WAVE VELOCITY FOR PREDICTION OF ARTERIAL STIFFNESS.

Physiology

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ABSTRACT

Adiposity of either type whether central (visceral) or generalized (subcutaneous) more or less contributes in arterial stiffness. The aim of the study was to analyse the relationship of general and visceral adiposity indices with arterial stiffness in general population of Jodhpur city.150 adults both males and females aged 25-65 years (50.85±11.06) were enrolled for this cross sectional study. Anthropometric parameters like weight, height, WC and BMI were measured Biochemical tests were performed to obtain VAI values by calculations. Total body fat percentage was derived from skinfold thickness of various body sites. Arterial stiffness was measured by pulse wave velocity using Periscope based on oscillometric technique. Pearson correlation and linear regression analysis were performed to assess the strength of relationships between variables. The results infer that central obesity correlate with characteristics of central arteries more as compared to generalized obesity.

KEYWORDS

adipocytes, skin fold thickness, body fat percentage, pulse wave velocity, arterial stiffness.

INTRODUCTION

"*Obesity is a disorder of the energy homeostasis system, rather than simply arising from the passive accumulation of excess weight*". (1)

A.Generalised / Gynoid -Abnormal or excessive subcutaneous fat accumulation causes 'generalized', ('gynoid', 'pear-shaped') obesity and presents a risk to health.(2) Subcutaneous fat which is found in places like thighs, buttocks and outer abdomen, serves as energy reservoir.(3) A crude population measure of obesity is the body mass index (BMI).which is closely associated with both total body fat and body fat percentage(4) and but it does not take into account the proportion of weight related to increased muscle mass or the distribution of excess fat within the body, (5) both of which affect health risks associated with obesity.(6)Individuals with a similar BMI can vary considerably in their abdominal-fat mass. (7) Skinfold measurement, also known as pinch test, measured by a caliper at several precise points on the body, allows to determine subcutaneous fat layer thickness and body fat percentage (8)

B.Abdominal / Android-Abdominal obesity is increased accumulation of fat surrounding the intra- abdominal organs,which causes central ('abdominal', 'visceral',/android', apple-shaped',) obesity, that is more common with metabolic syndrome and cardiovascular disease (9). Adipose tissue in the form of triglycerides is stored as the reservoir of fuel in adipocytes that controls glucose homeostasis and lipid metabolism.10.Visceral adipose tissue is the main fat store responsible for insulin resistance; thus, elements of the insulin resistance syndrome may serve as part of the link between increased visceral adipose tissue and stiffening of the aorta.11 Common surrogate measures of abdominal obesity are WC, WHR and WHtR. 12.Magnetic resonance imaging (MRI) and computed tomography (CT) (13) are the gold standard for determining visceral obesity. However, their use is limited in clinical practices for general population because they are time-consuming, costly and harmful to some extent as well. Visceral adiposity index (VAI), based on WC, BMI, triglyceride (TG) and high density lipoprotein cholesterol (HDL-C), is a newly proposed sex specific indicator of fat distribution and function associated with cardiometabolism[14].

Vascular disease is the leading cause of morbidity and mortality in the Western world,(15) and vascular function is determined by structural and functional properties of the arterial vascular wall. (16) Cardiorenal metabolic syndrome such as obesity, diabetes, hypertension, kidney disease and aging are conditions that predispose to arterial stiffening, which is a pathological alteration of the vascular wall and ultimately results in target organ damage in heart and kidney (17) Arterial stiffness, indirectly measured through non-invasive pulse wave velocity (PWV), (18)is one of the pathological states of vascular damage, contributing to both cardiovascular morbidity and mortality.(19) Although it is well established fact, that fat distribution has different effects on the cardiovascular system,(20) very few studies have analyzed the association between markers of arterial stiffness and obesity according to the location of body fat. So the present study was conducted to explore new insights and information on effects of obesity on vascular compliance.

MATERIALS AND METHODS

This is a cross sectional observational study, conducted in Department of Physiology, Dr. S.N.Medical College, Jodhpur, Rajasthan. The research work was commenced after the approval of the synopsis by the ethical committee,SNMC 150 individuals aging between 25 to 65 years, (50.85±11.06) of either gender who are native residents of Jodhpur city were included after obtaining the consent. Subjects with history of smoking, Chronic illness (Malignancy, Tuberculosis, Interstilial Lung Disease, Arthritis , Psychiatric disorders) , Acute infections (Covid -19 , Influenza, Typhoid) and Metabolic disorders (Thyroid disease, Cushing syndrome) were excluded. Anthropometric parameters like weight, height, WC and BMI were measured Biochemical tests were performed to obtain VAI values by calculations.

Body Fat percentage (BF %) was calculated by the 'Durnin and Womersley' four-site skinfold test.(21).The four skinfold measurements of biceps, triceps, subscapular and suprailiac by Digital Skinfold Caliper were used in this equation.

The Siri's equation BF% = ([4.95/bodydensity] −4.5) ×100 (22)

Table 3: Body Density Constants

		17-19 yrs	20-29 yrs	30-39 yrs	40-49 yrs	50+yrs
MALE	C	1.162	1.1631	1.1422	1.162	1.1715
	M	0.063	0.0632	0.0544	0.07	0.0779
FEMALE	C	1.1549	1.1599	1.1423	1.1333	1.1339
	M	0.0678	0.0717	0.0632	0.0612	0.0645

Body density = C (M [log10 sum of all four skinfolds). C, M = Predicted constant values of various age groups for body density

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

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

Visceral Adiposity Index- In the present study, skewness of data is observed for Visceral adiposity index so log transformed variable was used for analysis of data. Visceral adiposity index was calculated using sex specific equations.23.

Arterial Stiffness was assessed by PERISCOPE [RMS India], a Computer based pulse waveform analysis system that uses simultaneous Noninvasive Blood Pressure measurements and wave form from four limbs and ECG waveforms to calculate Pulse Wave Velocity (24) We collected right and left brachial ankle (ba PWV) and carotid femoral (cf PWV) pulse wave velocity25. 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 ToolPak Add-in in Microsoft Office excel® 2007 and IBM ® SPSS ® Statistics Subscription. Correlation analyses were performed between each and every variable by Pearson correlation analysis

Table 1- Pearson Correlation Analysis Obesity Indices Vs Arterial Stiffness Indices

Variables	Body Fat %		log VAI	
N 150	r value	p value	r value	p value
Rt baPWV (cm/sec)	0.018	0.828 NS	0.067	0.419 NS
Lt baPWV (cm/sec)	0.158	0.050 S	0.193	0.018 S
C-F PWV (cm/sec)	0.360	0.000 HS	0.153	0.05 S

*NS = Not Significant, S = Significant ($p < 0.05$) HS = Highly Significant ($p < 0.01$)

Significant associations of both body fat % and log VAI with pulse wave velocities were observed.

To obtain precise outcome of above relation analysis, independent association of body fat% and log VAI with arterial stiffness indices was done by linear regression analysis .

Table 2- Linear Regression Between Arterial Stiffness Indices And Log VAI

Variables	R^2	F	β	t	p value	CI 95% For β	
						Upper	Lower
Lt Ba PVW	0.037	0.193	5.696	2.387	0.018 S	30.084	319.721
CF-PVW	0.054	8.474	0.233	2.911	0.004 S	0.088	0.461

S = Significant ($p < 0.05$)

Beta coefficients represent 1 standard deviation (SD) proportional change in the outcome for each 1 SD increase in adiposity.

When dependent variable, PWV was regressed on predictive variable, log VAI, to assess the strength of relationship, significant p values denote positive effect of VAI on pulse wave velocity. R^2 indicates 3.7% and 5.4% variance in Lt Ba PVW and CF-PVW by log VAI.

Table 3-linear Regression Between Arterial Stiffness Indices And Body Fat %

Variables	R^2	F	β	t	p value	CI 95% For β	
Lt Ba PVW	0.025	3.787	0.158	1.946	0.054 NS	-0.170	22.098
CF-PVW	0.007	1.103	0.086	1.050	0.295 NS	-3.898	12.738

*NS = Not Significant, S = Significant ($p < 0.05$) HS = Highly Significant ($p < 0.01$)

Beta coefficients represent 1 standard deviation (SD) proportional change in the outcome for each 1 SD increase in adiposity.

No corelation could be established when dependent variable CF-PVW was regressed on predictive variable BF% , as non-significant p values for the regression analysis were observed.

DISCUSSION-

Obesity is a determinant factor in the development of cardiovascular diseases, and is associated to an increased incidence of hypertension.26. In addition to the increased total fat mass, cardiovascular risk is influenced by the distribution of fat deposits. Both type of obesity is inter-related and increases simultaneously but increase in fat deposition on thighs, arm and chest may not be as harmful as deposition of intra abdominal fat for risk of arteriosclerosis.As compared to subcutaneous fat, visceral obesity more closely predicts cardiovascular and metabolic diseases.27. Visceral adipocytes show elevated lipolytic activity that results in increased free fatty acids release in the portal vein with an accumulation in liver, pancreas, and muscles thus contributing to insulin resistance.28. Insulin stimulates the sympathetic nervous system, leading to higher values of heart rate and BP, both of which add to the mechanical strain on the vascular system. 29.In addition, insulin may cause hypertrophy of the vascular wall, resulting in increased number and size of monocytes, increased collagen, and proliferation of smooth muscle cells. 30.Another contributing factor is an increase in non-esterified fatty acids, which have been found to be associated with central obesity. 31.Non esterified fatty acids increase vascular stiffness by increasing α-adrenergic reactivity, vascular tone, and BP.32 Increases in the expression of pro inflammatory cytokines , interleukin-6 and tumor necrosis factor-α are expressed in adipose tissue and have been linked specifically to central obesity.33These pro inflammatory cytokines have been hypothesized to induce a low-grade systemic inflammation in persons with excess body fat and central fat specifically.34 Measures of inflammation have been found to be positively associated with PWV in various experimental studies.35.

Similar results for VAI were observed by Kim –Sutton Tyrell (2001),35 Hirokazu Morigami1, Tomoaki Morioka1 (2016) (36),Bin Wu , Jingshan Huang (2018), (37)Hee Seon Choi (2019) 38,Da-Hye Son (2021). (39)

In contrary to present study , B. Strasser M. Arvandi Austria 2015.(40) study revealed both carotid-femoral PWV and brachial-ankle PWV were significantly associated with BMI (both $P < 0.05$) but not with body fat percentage. .A. Ishida 2017(41), univariate analyses, VFA and WC showed a weak positive correlation; followed by weak negative association in multivariate analyses after adjustment for sex, age, systolic pressure, and heart rate. Visceral fat obesity was not systolic pressure-independently associated with increased aortic stiffness.

Miguel M. Fernandes-Silva1, Amil M. Shah1 USA 2018 (42) body fat percentage demonstrated,significant non-linear relationships with cfPWV (P=0.033).

SUMMARY AND CONCLUSION -

These results imply that arterial stiffness is more strongly associated with visceral adiposity (VAI) than overall obesity assessed by Body fat %. Although both general and visceral obesity were related to aortic stiffening in univariate analysis, it was clear from the regression analysis that the visceral fat depot was the most important factor for determining risk of arterial stiffness. Considering that the measurement of BF% requires specific equipment and technology, it is recommended to use VAI as an indicator of obesity in clinical practice. Further comprehensive research needs to be done to analyse the modified effect of age on the relationship between body composition and arterial stiffness.

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