THE ASSOCIATION BETWEEN ARTERIAL STIFFNESS AND OBESITY IN YOUNG ADULT SUBJECTS : A CASE -CONTROL STUDY

Sharma Ankit ¹, Jain Manila², Jee Kanhaiya³, Amir Hasan Ameerul ⁴

¹PhD Scholar, Department of Physiology, Index medical college, Malwanchal University, Indore M.P.

²Professor and Head, Department of Physiology, Index medical college, Malwanchal University,Indore M.P.

³ Assistant Professor, Department of Anatomy, Mayo Institute of Medical Sciences ,Barabanki, Uttar Pradesh-225001

4 Demonstrator, Department of Biochemistry, Government Medical College, Jalaun (Orai).

Correspondence: Dr. Ankit Sharma

ABSTRACT

<u>AIM</u>: the aim of the present study is to assess the levels of arterial stiffness among obese subject and healthy subjects.

<u>METHODS</u>: A total of 80 participants were included with age groups between 18-30 yrs. participants are divided into two groups ,group I-obese (n=40) and group II non obese (n=40). Arterial stiffness was assessed by pulse wave velocity(Brachial ankle pulse wave velocity) and Augmentation index(ALX).

<u>RESULTS</u>: Pulse wave velocity and Augmentation Index shows significantly positive correlation with BMI.

<u>**CONCLUSION:**</u> increase in arterial stiffness was closely correlated to the body mass index, which indicating that adult obesity has a adverse impact on vascular adaptation.

KEYWORDS: pwv (pulse wave velocity), Augmentation index, body mass index(BMI).

INTRODUCTION

Overweight is defined as having a weight that is greater than what is considered a healthy weight for a given height. It is determined by the body mass index (BMI). Obesity is defined as the accumulation of excess body fat to the point where it poses a health risk [1]. It is a complicated condition with multiple causes. Obesity is caused by genetic and Neuroendocrine factors, as well as an excessive caloric intake, sedentary lifestyle and other environmental factors [2]. In 2016, approximately 39% and 13% of the global adult population were overweight (BMI 25.0-29.9 kg/m2) and obese (BMI > 29.9 kg/m2) respectively [3].

The world is concerned about the rapid increase in overweight and obesity prevalence and the rising burden of disease, particularly in developing nations like India. By 2040, it is expected that the prevalence of obesity among Indian individuals aged 20 to 69 will have tripled [4].

Obesity is frequently linked to changes in the population and epidemiology, which have an impact on mortality and fertility rates. The metabolic syndrome, which includes type 2 diabetes, hypertension, and dyslipidemia, has become more prevalent as a result of rising obesity. These conditions significantly raise cardiovascular risk in addition to obesity [5].

Exercise and physical activity resulting high degree of cardiorespiratory fitness .All lower cardiovascular and all-causes of mortality, myocardial infarction and heart failure risk, as well as age-related arterial and cardiac stiffness. Over the last three decades, a lot of information on the pathophysiological mechanisms underlying atherosclerosis and obesity has come to light. Both conditions were formerly thought of as illnesses of lipid storage, with triglyceride buildup in fat tissue and cholesterol esters in atherosclerotic plaques. These days, both atherosclerosis and obesity are regarded as chronic inflammatory diseases, with a considerable role given to the activation of both innate and adaptive immunological mechanisms [6].

Numerous variables are shared in the aetiology of both atherosclerosis and obesity. Lipids, oxidised LDL particles, and free fatty acids both cause the condition by promoting inflammation. Obesity, insulin resistance, and type 2 diabetes are all linked to inflammation, which is also responsible for all the stages of atherosclerosis, from early endothelial dysfunction to the difficulties caused by atherosclerotic plaques. The atherosclerotic process is aided by the release of

adipocytokines from the adipose tissue, which cause insulin resistance, endothelial dysfunction, hypercoagulability, and systemic inflammation. [7]

Due to the rising prevalence of obesity and its shift toward younger ages, metabolic and cardiovascular problems are increasingly frequently observed in juvenile populations.[8] Childhood cardiovascular risk assessment knowledge is crucial since cardiovascular problems are the main cause of mortality worldwide.[9] However, assessments for early circulatory alterations in obese children are infrequently carried out during routine clinical care, thereby underestimating and undertreating cardiovascular problems linked to obesity.[10]

This is crucial, though, as it is known that cardiac abnormalities may be reversible as a result of early recognition, examination, and treatment of cardiovascular issues .An early indication of cardiovascular disease is arterial stiffness (AS).[11] Aging triggers a physiological process that causes the arteries to stiffen .It occurs when the amount of elastin fibres in the artery wall decreases and aberrant collagen is overproduced as a result of an inflammatory environment.[12]

AIM OF THE STUDY

AIM: The aim of the study is to compare the changes in arterial stiffness by evaluation of pulse wave velocity in young adult obese and compare with the control subject.

METHODOLOGY

<u>PLACE OF STUDY</u>: The present study was conducted in Department of physiology, Index Medical College, Malwanchal university, indore, (M.P).

SAMPLE SIZE: 80

Which were divided into 2 groups, **GROUP I**: obese n=40, **GROUP II**: control group n=40

INCLUSION AND EXCLUSION CRITERIA

INCLUSION CRITERIA (STUDY GROUP)

- obese adult of age (18-35yrs),BMI should be >30kg/m² INCLUSION CRITERIA (CONTROL GROUP)
- adult of age (18-35yrs),BMI should be 18.5-25 kg/m²

EXCLUSION CRITERIA (STUDY GROUP & CONTROL GROUP)

- History of diabetes mellitus, Gastestional diabetes .
- History of cardiovascular disease, History of hypertension, History of peripheral vascular diasease, Any disease that can alter the cardio vascular health.
- Any current and past aspirin or hormone replcements therapy, autoimmune diasease ,acute and chronic infections, hepatic diseases was also excluded .

PROCEDURE:

- <u>Patient Preparation</u> By thoroughly describing the study protocol to the participants for obtaining their signed informed consent. To exclude potential subjects, a complete history will be gathered along with a general physical assessment. After detailing the precise experimental protocol. The first step is to take all anthropometric measurements. Arterial stiffness is assessed using a periscope after a five-minute break.
- **BMI Assessment:** BMI was calculated as: BMI= Weight(kg) /Height(m)2

Classification of BMI is (kg/m^2)

♦ Underweight-<18.5

- ✤ Normal- 18.5-24.9
- ✤ Overweight 25-29.9
 - ✤ Obese >30.

<u>Arterial Stiffness Parameters</u> :-The standardised procedure includes employing periscope, a noninvasive automatic equipment based on oscillometric technique, to measure the parameters of arterial stiffness (Periscope, Genesis Medical Systems, India). Every recording was done while lying down . Because the device is totally automated and shows the data on its own, operator bias was avoided. Each limb's vascular parameters were printed.

STATISTICAL ANALYSIS

The result are presented in Mean±SD. All the physiological parameters were compared by using independent t-test between cases and control. All the analysis was carried out by using Statistical Package for Social Sciences (SPSS) version 22.

RESULT:

Table 1: Descriptive statistics of Age, gender and smoking variables of young adult in Obese and Non-obese group

VARIABLE	AGE	GEN	NDER SMC		OKING
	Mean ± SD	MALE	FEMALE	YES	NO
		(Frequency)	(Frequency)	(Frequency)	(Frequency)
OBESE (n=40)	25.28 ± 3.105	21	19	17	23
NON-OBESE (n=40)	26.00 ± 2.909	22	18	17	23

Table 1 show that the mean age of obese patients was 25.28 years and mean age of non-obese patients was 26 years. The majority of 52.5% patients in obese and 55% patients in non-obese was male and 47.5% patients in obese and 45% patients in non-obese was female. The majority of 57.5% patients in obese and non-obese both was non smoker and rest patients were smoker in both groups in this study.



Fig.no.1 distribution of gender and smoking in obese and non-obese subjects

Object: To investigate the changes in arterial stiffness by evaluation of arterial stiffness index and pulse wave velocity in young adult obese and compare with the non obese subject.

Table 2: Compare between obese and non-obese patients in arterial stiffness by evaluation of augmentation index and pulse wave velocity in young adult

Arterial stiffness	Obese group (Mean \pm SD)	Non-Obese Group (Mean \pm SD)	P-value*
BMI (Kgm ²)	31.545 ± 1.069	22.195 ± 1.584	0.001
RT BAPWV	1310.347 ± 197.203	1170.987 ± 114.374	0.001
LT BAPWV	1389.857 ± 234.654	1241.337 ± 55.484	0.001
ALX	11.78 ± 7.141	5.18 ± 2.659	0.001

*Independent t test used for two group comparison



Fig.no 2. Comparison between RTBAPWV and LTBAPWV with BMI in obese and non obese subjects.



Fig.no 3. Relationship between BMI and Augmentation index(ALX) in obese and non obese subjects.

Table 2 show that compare between obese and non-obese patients in arterial stiffness by evaluation of arterial stiffness index and pulse wave velocity in young adult. There was statistical significance difference obese and non-obese patients in BMI with P=0.001; Right brachial pulse wave velocity with P=0.001; Left brachial pulse wave velocity with P=0.001; Augmentation index with P=0.001. Obese patients had greater mean value than Non-obese patients.

Dissussion:

An earlier study looked into the possibility of a relationship between arterial stiffening, as measured by baPWV, and peripheral skeletal muscle mass. It found that arterial stiffness was linked to a higher rate of muscle mass loss over time, regardless of age, total body fat, peripheral arterial disease, chronic inflammation, or cardiac disease. Age-related muscle loss and atherosclerosis are thought to interact and share comparable pathogenic mechanisms, according to Ochi et al. In actuality, the scientists found a direct link between baPWV and thigh muscle sarcopenia in males, but they could not find a similar link in women.[13]

Furthermore, Kohara et al. discovered that sarcopenic obese men had greater baPWV levels than men who were neither sarcopenic, obese, or even normal weight. The relationship between changes in arterial stiffness and cardiovascular risk may, in theory, be mediated. It is not apparent, though, how the loss of muscle mass and arterial stiffness are related. The authors hypothesised that disruption in blood vessel dynamics would play a prognostic role in muscle mass loss because basal limb blood flow reduces with ageing, in part because of arterial stiffening.[14]

Arterial compliance is the capacity of an artery to change in size and shape in response to the heart's contraction and relaxation. This enables the blood to change from its intermittent and pulsatile state to a continuous laminar flow. The results of the current investigation indicate a statistically significant association between arterial stiffness and obesity. A higher AS causes the left ventricle to work harder and experience increased blood flow resistance. As a result, atherosclerosis progressed more quickly and blood pressure increased.[15]

In this study, it was discovered that obese individuals' mean pulse wave velocities are higher than those of non-obese patients. Greenland et al. also found the similar association. The main causes of AS include advancing age and obesity. The artery stiffens as a result of the arterial wall's higher collagen content and lower elastin tissue levels. A rise in local vasoconstrictors like endothelin-1 (ET-1) or a decline in vasodilators like nitric oxide, in addition to structural changes (NO).We would like to emphasize the significance of physical activity, specifically the combination of progressive resistance exercise and aerobic exercise, in accordance with well-balanced nutrition in relation to low Skeletal muscle mass index and arterial stiffness, particularly because the participants with low Skeletal muscle mass index in the present study had lower physical performance, lower BMI, and a higher risk for malnutrition.[16]

Conclusion:

Increase in pulse wave velocity and augmentation index in adult with obesity were observed, both indicating an increased arterial stiffness. Consequently, measurement of arterial stiffness should be considered in adult with obesity as part of cardiovascular risk assessment.

REFERENCE:-

1. WHO (2020) Obesity. World Health Organization.

2. Ranjani H, Mehreen TS, Pradeepa R, Anjana RM, Garg R, et al. (2016) Epidemiology of childhood overweight & obesity in India: A systematic review. Indian J Med Res 143(2): 160-174.

3. WHO (2020) Overweight and obesity. World Health Organization.

4. Luhar S, Timæus IM, Jones R, Cunningham S, Patel SA, et al. (2020) Forecasting the prevalence of overweight and obesity in India to 2040. PLoS ONE 15(2): e0229438.

5. Akil L, Ahmad HA (2011) Relationships between obesity and cardiovascular diseases in four southern states and Colorado. J Health Care Poor Underserved 22(S4): 61-72.

6. V. Z. Rocha and P. Libby, "Obesity, inflammation, and atherosclerosis," Nature Reviews Cardiology, vol. 6, no. 6, pp. 399–409, 2009.

7. R. Ross, "Atherosclerosis–an inflammatory disease," The New England Journal of Medicine, vol. 340, no. 2, pp. 115–126, 1999.

8. Han JC, Lawlor DA, Kimm SY. Childhood obesity. Lancet. 2010;375:1737-1748.

9. Expert Panel on Integrated Guidelines for Cardiovascular Health and Risk Reduction in Children and Adolescents; National Heart, Lung, and Blood Institute. Expert panel on integrated guidelines for cardiovascular health and risk reduction in children and adolescents: summary report. *Pediatrics*. 2011;128(suppl 5):S213-S256.

10. Cote AT, Harris KC, Panagiotopoulos C, Sandor GG, Devlin AM. Childhood obesity and cardiovascular dysfunction. *J Am Coll Cardiol*. 2013;62:1309-1319.

11. Mozos I, Malainer C, Horbańczuk J, et al. Inflammatory markers for arterial stiffness in cardiovascular diseases. *Front Immunol*. 2017;8:1058.

12. Zieman SJ, Melenovsky V, Kass DA. Mechanisms, pathophysiology, and therapy of arterial stiffness. *Arterioscler Thromb Vasc Biol*. 2005;25:932-943.

13. Ochi M, Kohara K, Tabara Y *et al*. Arterial stiffness is associated with low thigh muscle mass in middle-aged to elderly men. *Atherosclerosis* 2010; **212**: 327–332.

14. Abbatecola AM, Chiodini P, Gallo C *et al*. Pulse wave velocity is associated with muscle mass decline: Health ABC study. *Age (Dordrecht, Netherlands)* 2012; **34**: 469–478.

15.Sherwood, L. (2008). Human Physiology: From Cells to Systems. Boston: Cengage Learning.

16. Sakuma K, Yamaguchi A. Sarcopenic obesity and endocrinal adaptations with age. *Int J Endocrinol* 2013; **2013**: 1–12. Available from: <u>http://dx.doi.org/10.1155/</u> 2013/204164.