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THE ASSOCIATION BETWEEN ARTERIAL STIFFNESS AND OBESITY IN YOUNG ADULTS: A CASE CONTROL STUDY

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Abstract

Background: The world is concerned about the rapid increase in overweight and obesity prevalence and the rising burden of disease, particularly in developing nations like Pakistan.

Objective: The objective of the current study was to find out the he levels of arterial stiffness among obese subject and healthy subjects.

Material and Methods: A total of 80 participants were included with age groups between 18-30 years and participants were divided into two groups. The group I-obese (n=40) and group II non-obese (n=40). Arterial stiffness was investigated by pulse wave velocity (Brachial ankle pulse wave velocity) and Augmentation index (ALX).

Results: Pulse wave velocity and Augmentation Index shows significantly positive correlation with BMI.

Conclusion: The increase in arterial stiffness was closely correlated to the body mass index, which indicating that adult obesity has an adverse impact on vascular adaptation.

Keywords: Arterial Stiffness, Obesity, Young Adults, BMI

1. INTRODUCTION

Being overweight is described as weighing more than what is deemed healthy for a person of a particular height. The body mass index (BMI) determines it. The buildup of excess body fat to the point where it becomes dangerous for one's health is known as obesity. [1] It is a complex ailment with several underlying causes. In addition to an excessive calorie intake, sedentary lifestyle, and other environmental variables, obesity is caused by hereditary and neuroendocrine factors. [2] In

2016, the proportion of overweight adults (BMI 25.0-29.9 kg/m2) and obese adults (BMI > 29.9 kg/m2) worldwide were roughly 39% and 13%, respectively. [3]
The world is worried about the sharp rise in the prevalence of overweight and obesity as well as the rising cost of illness, especially in developing countries like Pakistan. It is anticipated that the percentage of Indians aged 20 to 69 who are obese would have tripled by 2040. [4]

Changes in the population and epidemiology, which affect mortality and fertility rates, are often associated with obesity. The increased obesity rate has led to an increase in the metabolic syndrome, which encompasses type 2 diabetes, hypertension, and dyslipidemia. In addition to obesity, these disorders markedly increase the risk of cardiovascular disease. [5]

Physical activity and exercise lead to a high level of cardiorespiratory fitness. Age-related arterial and cardiac stiffness, myocardial infarction and heart failure risk, and all reduce cardiovascular and all causes of death. A wealth of knowledge regarding the pathophysiological pathways driving obesity and atherosclerosis has emerged within the past thirty years. Prior to this, both disorders were believed to be disorders of lipid storage, with cholesterol esters in atherosclerotic plaques and triglyceride accumulation in adipose tissue. These days, the activation of both innate and adaptive immune pathways is considered to play a significant role in the development of both obesity and atherosclerosis, which are classified as chronic inflammatory disorders. [6]

The etiology of obesity and atherosclerosis shares many commonalities. The disorder is brought on by oxidized LDL particles, free fatty acids, and lipids all of which increase inflammation. Inflammation is associated with obesity, insulin resistance, and type 2 diabetes. It is also the cause of all stages of atherosclerosis, from early endothelial dysfunction to the problems brought on by atherosclerotic plaques. Adipocytokine are released from adipose tissue and contribute to the atherosclerotic process by inducing insulin resistance, endothelial dysfunction, hypercoagulability, and systemic inflammation. [7]

Juvenile populations are increasingly experiencing metabolic and cardiovascular issues as a result of the growing incidence of obesity and its shift toward younger ages.[8] Given that cardiovascular issues are the leading cause of death globally, understanding childhood cardiovascular risk assessment is essential.[9] Unfortunately, evaluations for early circulatory changes in obese children are rarely performed as part of standard clinical care, which leads to an underestimation and under treatment of obesity-related cardiovascular issues.[10]

This is important, though, because it is well recognized that early detection, testing, and treatment of cardiovascular problems may result in reversible cardiac abnormalities. Arterial stiffness (AS) is a precursor to cardiovascular disease.[11] The arteries stiffen as a result of a physiological process brought on by aging. It happens when the arterial wall's elastin fiber content drops and abnormal collagen is overproduced as a result of an inflammatory milieu.[12]

Objective of the Study
The objective of the current study was to find out the he levels of arterial stiffness among obese subject and healthy subjects.

2. MATERIAL AND METHODS
The present study was conducted in Department of physiology of Jinnah Hospital and Mayo Hospital, Lahore. The sample size of the study was 80 participants. The sample size of consisted of two groups (I) Obese group consist of 40 participants (II) Control group also consisted of 40 participants. The inclusion criteria of the study was that obese adult of age (18-35yrs), BMI should be >30kg/m^2. The inclusion criteria of the control group was adult of age (18-35yrs), BMI should be 18.5-25 kg/m^2. The exclusion criteria for the both groups (Study and Control) was individual

with the history of diabetes mellitus or Gestational diabetes, individual with history of cardiovascular disease, individual with history of hypertension, individual with history of peripheral vascular disease, and any disease that can alter the cardio vascular health. The individual was also excluded with any current and past aspirin or hormone replacements therapy, autoimmune disease, acute and chronic infections, hepatic diseases.

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

The classification of BMI is (kg/m^2):
- Underweight-<18.5
- Normal- 18.5-24.9
- Overweight – 25-29.9
- Obese - >30.

The standardized procedure includes employing periscope, a non- invasive automatic equipment based on oscillometric technique, to measure the parameters of arterial stiffness (Periscope, Genesis Medical Systems). Every recording was done while lying down. Because the device was totally automated and showed the data on its own, operator bias was avoided. Each limb's vascular parameters were printed.

The findings were 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 25.

3. RESULTS

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

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

The average age of non-obesity patients was 26 years, while the average age of obese patients was 25.28 years, as indicated by Table 1. Male patients made up 52.5% of the obese and 55% of the non-obese patients, while female patients made up 47.5% of the obese and 45% of the non-obesity patients. In this study, the majority of patients—57.5% of the non-obese and obese patients as well as the remaining patients—did not smoke.

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 ± SD)**	**Non-Obese Group (Mean ± 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

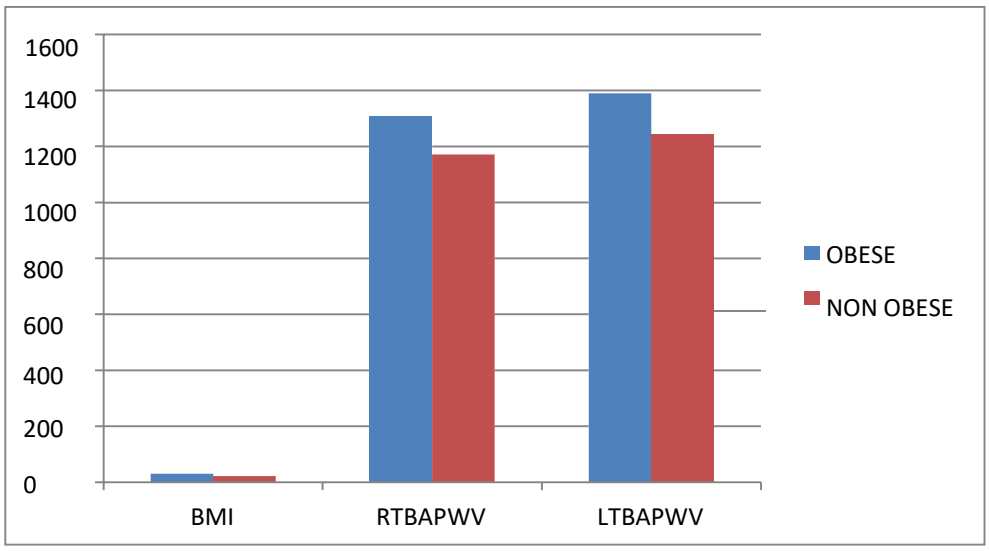

Figure 1: Comparison between RTBAPWV and LTBAPWV with BMI in obese and non-obese subjects.

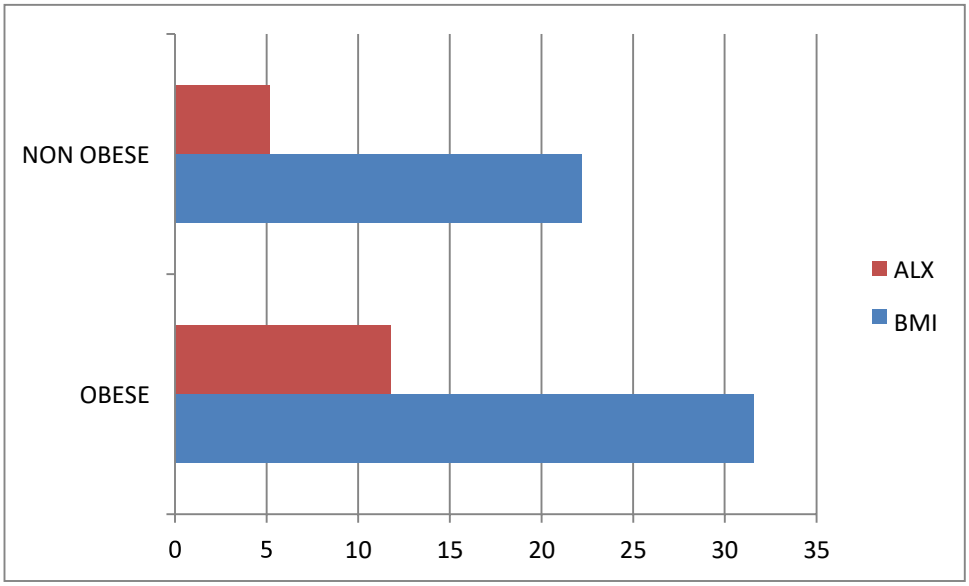

Figure 2: Relationship between BMI and Augmentation index (ALX) in obese and non-obese subjects.

Table 2 compares the arterial stiffness of obese and non-obese patients using the young adult's pulse wave velocity and arterial stiffness index. Patients who were obese or not showed statistically significant differences in their BMI (P = 0.001), left and right brachial pulse wave velocities (P = 0.001), and augmentation index (P = 0.001). Compared to non-obese patients, obese patients had a higher mean value.

4. DISCUSSION

An earlier study investigated the potential of a connection between peripheral skeletal muscle mass and arterial stiffness as evaluated by baPWV. It was discovered that, independent of age, total body fat, peripheral arterial illness, chronic inflammation, or heart disease, arterial stiffness was associated with a faster rate of muscle mass loss over time. According to Ochi et al., age-related muscle loss and atherosclerosis are hypothesized to interact and share similar pathogenic processes. Actually, the researchers discovered a direct correlation between baPWV and thigh muscle sarcopenia in males, but not in females. [13]

Moreover, sarcopenic obese men exhibited higher baPWV values than neither sarcopenic nor obese nor even normal weight men, according to Kohara et al. theoretically, there could be a mediating effect between alterations in arterial stiffness and cardiovascular risk. The relationship between arterial stiffness and muscle mass loss is unclear, though. The authors postulated that because basal limb blood flow decreases with aging, partly due to arterial stiffening, disruption in blood vessel dynamics would have a predictive effect on muscle mass loss. [14]

The ability of an artery to alter in size and form in response to the contraction and relaxation of the heart is known as arterial compliance. As a result, the blood can transition from a pulsating, intermittent state to a continuous, laminar flow. The current study's findings show a statistically significant correlation between obesity and arterial stiffness. The left ventricle must work harder and encounter more blood flow resistance when the AS is higher. As a result, blood pressure rose and atherosclerosis advanced more swiftly. [15]

This study found that the mean pulse wave velocities of obese patients are higher than those of non-obese people. Greenland et al. discovered the same correlation. The two main causes of AS are obesity and aging. Because there is less elastin tissue and more collagen in the arterial wall, the artery stiffens. Together with structural alterations, there may also be a decrease in vasodilators like nitric oxide or an increase in local vasoconstrictors such endothelin-1 (ET-1). In relation to low skeletal muscle mass index and arterial stiffness, we would like to emphasize the importance of physical activity, specifically the combination of progressive resistance exercise and aerobic exercise, in accordance with well-balanced nutrition. This is especially important because the participants in the current study who had low skeletal muscle mass index also had lower BMIs, lower physical performance, and a higher risk of malnutrition. [16]

5. CONCLUSION

All the above discussion can be concluded that adult obesity was associated with a rise in both the augmentation index and pulse wave velocity, which are markers of increased arterial stiffness. Therefore, as part of the evaluation of cardiovascular risk in adults with obesity, arterial stiffness measurement should be taken into account.

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