Journal of
Obesity & Weight Loss Therapy

Short Communication Open Access

Obesity: A Crucial Risk Factor for Underlying Cause of Cardiovascular Diseases

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Introduction

Obesity has become more common around the world, which is a cause for concern because the detrimental effects of obesity can begin as early as childhood. The body mass index (BMI) is the most extensively used anthropometric technique to determine relative weight and define obesity; BMI alone indicates a U- or J-shaped relationship with clinical outcomes and death. Such an inverse association has sparked a debate in the literature known as the 'obesity paradox,' which claims that individuals with high BMI and chronic conditions have a better survival rate and have fewer cardiovascular (CV) events than non-obese patients. BMI, on the other hand, is unable to distinguish between an increased body weight caused by high levels of lean vs. fat body mass. In general, metabolic problems are more typically related with an excess of body fat (BF) than with a high amount of lean body mass.

Adipose tissue is now thought to be a key organ in the fate of excess dietary lipids, determining whether body homeostasis is maintained (metabolically healthy obesity) or a state of inflammation/insulin resistance is produced, both of which have negative cardiovascular consequences. Obesity, particularly visceral obesity, causes a number of anatomical changes in the structure and function of the cardiovascular system. Adipose tissue is currently thought of as an endocrine organ that orchestrates critical interactions with important organs and tissues such the brain, liver, skeletal muscle, heart, and blood vessels [1].

Obesity and CVD: Obesity has a number of negative effects on the CV system. Excess body fat accumulates over time, causing a number of metabolic changes that increase the incidence of CVD risk factors while also altering systems that control inflammation. Obesity increases changes in other intermediate risk factors such as dyslipidemia, hypertension, glucose intolerance, inflammation, obstructive sleep apnea/hypoventilation, and a prothrombotic state, as well as possibly many more unknown processes.

Cardiac adaptations to obesity: The CV system adapts to maintain whole-body homeostasis as a result of chronic excessive body fat buildup. In this adaptative condition, increased cardiac output and decreased peripheral resistance are important. The increase in circulating blood volume increases stroke volume, which is a primary predictor of higher cardiac output in obese patients. Increased heart preload is aided by increased blood volume, which causes the Franck-Starling curves to move to the left [2]. Long-term, increasing cardiac burden causes ventricular remodelling, including expansion of the heart cavities and increased wall tension, which can lead to left ventricular hypertrophy (LVH).

Ventricle thickening is associated with a reduction in diastolic chamber compliance, which leads to an increase in LV filling pressure and LV diastolic dysfunction, which can be corrected with weight loss or aerobic exercise training. LVH adapts to LV chamber enlargement early in the disease's progression, and systolic function is retained. When LVH becomes more essential than LV dilatation, however, systolic function will eventually be affected. Muscle degradation, increased total blood volume, and diastolic and systolic dysfunctions are the major antecedents of HF in obesity, in addition to LVH. Furthermore, various obesity-related co-morbidities, such as hypertension, sleep apnea, and diabetes, may worsen or predispose obese patients to HF. Obese children have more epicardial fat, left atrial, and LV enlargement than lean controls, according to clinical investigations [3].

Along with increasing adiposity, several other changes are seen in the heart itself. Epicardial fat deposit is found on the surface of the heart, close to the coronary arteries, in healthy people. A bigger amount of extracellular fat deposition builds up in the epicardium with obesity, in addition to the intracellular fat accumulation. The closeness of epicardial fat and coronary arteries may be linked to the severity of atherosclerosis. Furthermore, the amount of visceral fat is linked to epicardial fat deposition. The potential link between fat buildup on the surface of the heart and the risk of cardiovascular disease is still unknown. Epicardial fat, on the other hand, appears to create pro-inflammatory adipo(cyto)kines and macrophage signals that may have a role in the development of CHD.

Obesity's Role in Cardiovascular Disease: Atherosclerosis is a degenerative disease that begins early in life and continues throughout one's life. Atherosclerosis progresses with age, but numerous chronic inflammatory diseases, including as obesity and diabetes, can hasten its onset. Obesity and the development of cardiovascular disease are now undeniably linked [4].

Obesity is linked to the development of atherosclerosis in young individuals as indicated by carotid intimalmedial thickness with time. This study lends credence to the idea that childhood obesity may have a cumulative cardiovascular effect on adult CV outcomes. Young people with visceral obesity appear to have more infiltrating macrophages (macrophages/mm^2) in their atherosclerotic lesions from a pathophysiological standpoint. Endothelial dysfunction is more common in young obese people, according to research.

Even in the absence of established CVD risk factors, obesity, particularly abdominal obesity, has been linked to reduced endothelium dependent vasodilation. Endothelium-dependent vasodilation is thought to be an early indicator of atherosclerosis due to its connection to nitric oxide. Aside from endothelial dysfunction, the resistance of blood arteries and their inflammation are likely to play a role in the early development of atherosclerosis in obese people.

Obesity and processes leading to accelerated atherosclerosis share common pathophysiological pathways, which both involve inflammation and changes in lipid metabolism. In contrast to atherosclerosis, the pathophysiology of obesity involves free fatty acids (FFAs) and triglycerides rather than LDL cholesterol. Chronic caloric excess in obesity causes the buildup of dietary fatty acids in adipose tissue until its storage capacity is reached, resulting in lipid

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Received November 08, 2021; **Accepted** November 22, 2021; **Published** November 29, 2021

Citation: Michaelson K (2021) Obesity: A Crucial Risk Factor for Underlying Cause of Cardiovascular Diseases. J Obes Weight Loss Ther 11: 474.

spillover into typically lean tissues such as the liver, muscles, and intra-abdominal or visceral adipose depots. The saturation of lipid storage capacity in subcutaneous adipose tissue, as well as the ectopic fat deposition that results, causes inflammation and insulin resistance. Adipo(cyto)kines are also implicated in modifying processes that promote atherosclerosis, such as endothelial vasomotor dysfunction, hypercoagulability, and dyslipidemia, and are released by adipose tissue. Obesity changes the levels of several inflammatory mediators. First, levels of circulating Creactive protein (CRP) and tumour necrosis factor (TNF) (produced by adipose tissue) are elevated, but other mediators (such as Il-6 and 1B, and monocyte chemoattractant protein 1) and hormones (such as adiponectin and leptin) are also known to play a role in the inflammatory profile seen in obesity, particularly abdominal obesity [5].

Raised blood supply is required for excessive adipose tissue growth, as seen in obesity, and total adipose tissue blood flow is increased globally. Perfusion per unit of adipose tissue, on the other hand, diminishes as adiposity rises. When an obese person is compared to a nonobese control, the difference in perfusion may imply a 35 percent reduction in relative perfusion. This misalignment in perfusion reduces the oxygen supply to adipocytes, contributing to cellular hypoxia, organ stress and malfunction, pro-inflammatory responses, and metabolic illness.

References

1. Mathieu P, Poirier P, Pibarot P, Lemieux I, Despres JP (2009) Visceral obesity: the link among inflammation, hypertension, and cardiovascular disease. Hypertension 53: 577-84.
2. Wormser D, Kaptoge S, Di AE (2011) Separate and combined associations of body-mass index and abdominal adiposity with cardiovascular disease: collaborative analysis of 58 prospective studies. Lancet 377: 1085-95
3. Messerli FH, Nunez BD, Ventura HO, Snyder DW (1987) Overweight and sudden death. Increased ventricular ectopy in cardiopathy of obesity. Arch Intern Med 147: 1725-28.
4. Wilson PW, D'Agostino RB, Sullivan L, Parise H, Kannel WB (2002) Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. Arch Intern Med 162: 1867-72.
5. Freedman DS, Dietz WH, Tang R, Mensah GA, Bond MG, et al. (2004) The relation of obesity throughout life to carotid intima-media thickness in adulthood: the Bogalusa Heart Study. Int J Obes Relat Metab Disord 28: 159-66.