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IP Journal of Nutrition, Metabolism and Health Science

Journal homepage: https://www.jnmhs.com/

Review Article

Impact of obesity on hypertension: A review

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ARTICLE INFO

Article history:
Received 21-05-2021
Accepted 10-06-2021
Available online 22-06-2021

Keywords:
Body mass
Sugar
kidney
Metabolic disorder
Heart disease
Hypertension

ABSTRACT

The primary cause of extreme obesity and overweight is a disparity between energy consumption and expenditure. Obesity is technically described as the excess deposition of 20% or more body fat over a person's ideal weight of body. The latter is an individual's maximum healthy value, which is measured primarily based on age, build, height, and degree of muscular growth. Obesity, on the other hand, is diagnosed by comparing an individual's weight to his or her height and calculating the BMI. The NIH has set a maximum of 30 kg/m2 as the threshold for being considered obese. As a result, amid World Health Organization warnings, obesity is on the rise in children and adults around the world. Obesity's rise, as well as the scope of related health problems, has significant ramifications for both people and public healthcare systems. Obesity is linked to increased chances of injury, sickness, and death, and it is one of the world's most overlooked public health problems. Obesity is linked to cardiac problems, which are the primary cause of death globally especially hypertension and diabetes. However, the mechanisms underlying obesity-related hypertension and other metabolic disorders have yet to be thoroughly studied. We looked at the connection among obese and heart disease, specifically the biological mechanisms that link obese and hypertension, in this study.

1. Introduction

Obesity is historically described as a weight that is 20% higher than the average weight, that correlates to the smallest mortality rate for people of a particular age, gender, and Height.[1] More current obesity recommendations have included the usage of a formula called (BMI), which is determined by multiplying an personal's weight by 703 and dividing by twice their height in inches. The BMI value is now utilized to determine the phase of overweight and obesity, with a BMI of 25.9–29 being deemed overweight and a BMI of >30 being considered obese.[2] In 2005, nearly 1.6 billion people above the age of 15 were obese, as per the World Health Organization (WHO).[3] At estimated 390 million individuals were overweight, with a further 20 million children under the age of five being obese. For the existing year (2021), it is estimated that there are nearly 2.3 billion obese adults and over 700 million obese ones.[4] The World Health Organization describes 'globesity' as a global obesity epidemic that is constantly on the rise. The condition is critical because the disorders that can result from overweight are becoming more common, especially heart disease, which are now the leading cause of death globally.[5] Obesity can lead to health problems, such as high blood pressure, diabetes mellitus, an elevated incidence of coronary artery disease, inexplicable heart failure, hyperlipidemia, fertility problems, and an increased proportion of colon, ovarian, uterine, and prostate cancer.[6,7] While the connection between overweight and diabetes in adults and children is well-known.[8–11] The process through which overweight induces obesity is being researched.[12,13] The thoracolumbar division activation, the quantity of intra-abdominal and intravascular fat, sodium retention, which leads to a rise in renal reabsorption, and the adrenergic system are all thought to play a role in the pathophysiology

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https://doi.org/10.18231/j.ijnmhs.2021.007

of obesity-related hypertension,a long-term clinical problem wherein blood pressure remains consistently at or above 140/90 mm Hg, but not at the usual range of 100–140 and 60–90 mm Hg for systolic and diastolic pressure, generally. As a result, obesity is a complicated and severe health condition that necessitates a thorough comprehensive understanding especially of the events that contribute to hypertension, in order to develop effective therapeutic interventions.The current review looks at the pathways through which overweight can contribute to high blood pressure and how obesity control can be used as a clinical tool for hypertension care.

2. Hypertension and Obesity

2.1. Obesity: causes and effects around the world

There is no common reason for all instances of overweight around the world. Socioeconomic, status race, residence area, season, and city living are all external influences linked to obesity.[14] Obesity, on the other hand, is caused by a mismatch betwixt energy consumption & expenditure.[15] While genetic predisposition can be a determining factor of weight gain, previous research has demonstrated that genetic predisposition does not always contribute to the progression of obesity, as dietary habits and physical activity patterns may play a larger role in the extent of calories obtained.[16,17] Sedentary behaviour, as well as behavioural factors like depression, low self-esteem, and lack of night sleep, may all lead to weight gain.[18] While the precise cause of gaining weight is unknown, it is thought to be caused by a complicated confluence of things, including genetic factors that influence how the body controls hunger and the level at which food is converted into energy, defined as the metabolic rate.[19,20], Excess weight is clearly obtained by consuming more calories than the body uses, with the extra calories being retained as fat tissue.[15] As a result, the amount of fat in a person's diet may have a bigger effect on their weight than the amount of calories. The bulk of calories and fat are contained in fat cells, that grow and multiply, adding to the body's weight and girth.[21] Carbs, such as cereals, breads, fruits, and vegetables, as well as proteins, are transformed to fuel almost immediately after intake.[22] Furthermore, high-fat diets cause elevated and unusually high blood cholesterol levels (hypercholesterolemia).[23] Fat in the bloodstream may instantly impact some organs such as the liver and kidney, as well as have debilitating local effects within the arteries throughout the development of atherosclerosis depending on the balance between the fractions of saturated and unsaturated fatty acids[24] Visceral fat is an accumulation of fat within the arterial wall that causes occlusion of the medium and broad arteries. The latter is primarily responsible for metabolic syndrome.[25]

Highly high-carbohydrate diets and excessive alcohol consumption encourage a rise in the lipid product found in the blood, triglycerides, that is another risk factor in the development of atherosclerosis.[24] Physical, lack of activity, smoking, tension, inflammation, and some bacteria are all factors that promote the development of atherosclerosis[26] However, fat continued to lodge in branch vessels, obstructing the flow of blood through the broad arteries. Enhanced carotid intima-media width has been found in obese subjects regardless of blood pressure levels, including in the normotensive blood pressure range, which is consistent with these findings.[27] This finding points to an early stage of the atherosclerotic phase in obese people. Accumulation builds up over time, forming a type of plaque that can continue to develop until the vascular wall narrows or stenosis occurs[28] that can begin to escalate until significant blockages of the arterial lumen are caused, resulting in downstream local diseases that damage organs contributing to morbidity[29,30] and even death.[31] Another negative consequence of excessive calorie consumption is a rise in norepinephrine turnover in peripheral tissues, which raises the resting plasma norepinephrine level, that is an indirect measure of SNS activity, and amplifies the increased plasma **Norepinephrine** in response to triggers like upright posture.[32] Thus, a high fat and carbohydrate intake in the diets has been proposed to acutely activate peripheral 1 and adrenergic receptors, resulting in an increase in sympathetic activation and hypertension.[33] In overweight hypertensive rats up — regulated hypothalamic tyrosine hydroxylase and hypothalamic adrenoceptor gene expression of the 2B receptor were discovered.[34] Furthermore, in model systems, pharmacological blockade of adrenergic activity significantly reduced the increase in blood pressure in animals fed a high-fat diet.[10] In humans, combined and adrenergic blockade substantially reduced blood pressure in overweight patients with critical hypertension compared to lean patients,[35] though increased heart rate appears to be the result of reduced parasympathetic function.[36] Obesity is a disorder that alters the body's structure, causing organ damage and modification. The heart,[37] liver[38], kidney,[39] lungs,[40] colon,[41] skin,[42] vessels,[43] and brain are among the major organs that bear the brunt of obesity.[44] Although the impact on each organ is linked to collateral defects that may pose severe health risks, renal injury appears to be the most closely linked to body mass, as dietary fat restriction improves renal histology significantly.[45] Obesity-related structural alterations in the kidneys are notable, as fat deposits across the kidneys, in combination with elevated abdominal pressure caused by central obesity, have been proposed as another trigger of increased renal re - absorption disorder. Obesity triggers renal vasodilation and glomerular hyper filtration as a corrective process to retain sodium balance despite the elevated tubular re absorption, which,

along with increased arterial blood pressure and metabolic disturbances, as well as many other factors including inflammation, oxidative stress and lipotoxicity, may lead to the exacerbation of the condition.[45–47] Proteinuria is normally detected clinically before the glomerular filtration rate declines over many years as a result of these factors.[48,49] The involvement of insulin and angiotensin II in the development of glomerular hyper filtration have been shown in obese animals[42] in which, leptin, a hormone produced in adipose tissue, contributes to the development of renal injury through the induction of cytokines.[50] Adipose tissues, especially that of abdomen fat have been shown to have systemic effects by secreting a variety of hormones and cytokines, resulting in obese-associated glomerulopathy.[51] According to the evidence, high-fat, high-carbohydrate diets cause hyperglycemia and free fatty acids in the blood[23] that, in combination with free fatty acids emitted from phospholipids, may focus independently on ion channels of smooth muscle cells and other tissues.[52] It can also function as effective modulators of the phosphorylation of protein kinase C's calcium-independent isoenzyme,[53] which is essential for signaling pathways and cell regulation. The binding of free fatty acids to NA/K-ATPase causes changes in interactions between the enzyme and neighbouring membrane proteins, resulting in the development of multiple signaling modules and the activation and output of the epidermal growth factor receptor, which results in an increase in reactive oxygen species.[54] Endothelial function is reduced primarily as a consequence of a decline in nitric oxide (NO) as a result of increased oxidative stress or as a result of pro-inflammatory cytokines.[55] Overall, cytokines, oxidative stress, and a drop in NO cause vascular constriction and arterial stiffness, which are harmful and predisposed to venous insufficiency, venous thrombosis, and pulmonary embolus, heart disease, and hypertension in general.[37]

According to physiological, biochemical, and operational studies, the renin-angiotensin model is directly engaged in the creation of high blood pressure through two systems: tissue and circulating blood.[55] Adipose tissue-derived angiotensinogen, the main site where all constituents of the RAS are produced, can travel through the bloodstream, confirming this finding. The RAS in the tissue was constantly interacting with the blood. Angiotensinogen, angiotensin (Ang) I, and angiotensin (Ang) II are developed local and picked up by cells with overexpressed AngII receptors at the same time. Angiotensinogen development is both a trigger and an effect of adipocyte hypertrophy, and it raises blood pressure by inducing systematic vascular constriction, immediate sodium and water retention, and enhanced aldosterone development through operation of AngII.[56] As a result, since AngII is formed at increased levels and is not reduced by volume expansion, it defines an elevated salt-sensitive hypertension disorder in obesity.A sustained increase of sympathetic tone, which causes renal vascular constriction and renin-dependent chronic hypertension may be another cause of RAS activation According to the theory of infinite feedback gain, the arterial blood pressure regulation system of diuresis and natriuresis appears to be elevated near the higher blood pressure value in obese people.Abnormalities in such pathways appear to enhance blood pressure, sodium and water excretion through pressure natriuresis and diuresis,[57] resulting in extracellular fluid volume expansion and a hypertensive change of the pressure natriuresis,[10,11] A hypertensive resetting of the kidney fluid system, associated with the model of hypertension caused by volume overload. The risk of shifts in intrarenal forces induced by histological changes in the renal medulla, which may tighten the loops of Henle and vasa recta, is another important factor of pressure natriuresis shifting towards higher blood pressure levels in obesity.[58] Besides that, in human obese,[59] extreme amounts of plasma renin production, plasma AngII, and aldosterone were found, with a presynaptic potentiating impact on sympathetic neurotransmission in patients on sodium restriction,[60] Hypothetical pathways include reduced baroreceptor sensitivity and elevated concentrations of circulating free-fatty acids, AngII, insulin, and leptin, among others.[61]

Increased rates and unusually dispersed free-fatty acids were found in obese hypertensive patients, which increased vascular adrenergic sensitivity and, as a result, adrenergic sound (62). Despite the fact that the pathways were endogenous, the effect of free-fatty acids has yet to be studied. Free fatty acids have been shown to suppress Na+, K+ ATPase, and the sodium pump, increasing vascular smooth muscle tone and resistance.[54]

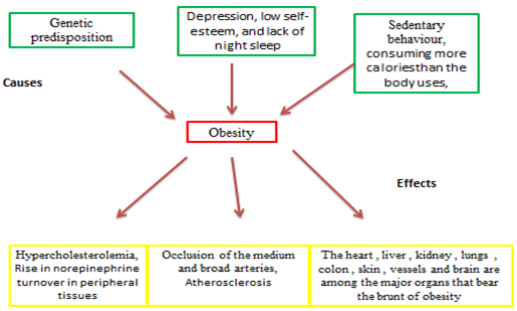

Fig. 1: summarization of cause and effects of obesity

2.2. *Obesity-induced hypertension treatment*

Antihypertensive medications, such as RAS blockers, -Beta blockers, and diuretic drugs, are appropriate for overweight patients with hypertension, but they have serious complications such as hyperglycemia, hyperlipidemia, and hyperuricemia, particularly at high doses. The treatment

of hypertension-induced obesity necessitated the control of the component obesity. Obesity treatment includes recommendations that recommend significant dietary changes aimed at reducing body weight, such as eating a low-calorie diet of 500–1,500 calories for men and 500–1,200 calories for women. This may include limiting salt intake and lowering saturated fat and cholesterol intake by increasing water, fruits, fresh and raw veggies, fish, lean meats, whole grains, and/or moderate and consistent physical activity as well as sufficient night sleep.[62,63] The aim of these behaviours and activities is to build and improve muscle mass while reducing fat mass.

3. Conclusion

Obesity enhances tubular reabsorption, hampering pressure natriuresis, and triggering volume widening through stimulation of the RAS and SNS , making it a potential contributing factor for essential diabetes, hypertension, and other comorbidities that contribute to kidneys disorder development.

Obesity also triggers renal and cardiovascular diseases via a number of different mechanisms namely, hypertension, inflammation, inflammation, dyslipidemia, & atherosclerosis, that are abnormalities that can exist side by side, particularly when excessive visceral fat is present to activate metabolic syndrome[64] and are associated with changes in fat metabolism induced by lipid aggregation. Regular diet rich in fiber, omega-3 fats, nutritious veggies and protein sources, less fat and sugar, antioxidants, vitamins, and exercising regularly are thus healthy habits that allow the body 's nutritional signalling pathways to reach an allowable limit of equilibrium.

4. Source of Funding

None.

5. Conflict of Interest

None.

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Cite this article: Abdussalam. Impact of obesity on hypertension: A review. *IP J Nutr Metab Health Sci* 2021;4(2):41-46.