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Obesity is A Chronic Disease in all Ages

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Abstract

Obesity is a long-lasting condition marked by an excessive build-up of fat in the body, elevating the possibility of numerous health issues like heart disease, type 2 diabetes, hypertension, and certain cancers. This condition arises from an imbalance in energy, meaning an individual consumes more calories through their diet than what they expend via physical activity. Preventive measures involve maintaining a nutritious diet, engaging in regular physical exercises, and making lifestyle adjustments, while treatment options may consist of medication and surgical procedures.

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Introduction

An increase in caloric intake coupled with decreased physical activity leading to insufficient burning of these calories leads to an energy imbalance that causes obesity and excess weight [1]. Numerous factors contribute to this situation, including societal and environmental shifts, inadequate infrastructure and policies, and personal neglect. Generally, the rising consumption of energy-dense foods (high in fats, sugars, and salt but lacking essential minerals, vitamins, and micronutrients); the easy access to low-cost processed foods; reductions in physical activity; growing urbanization, globalization, environmental and industrialization providing broader market access; and contemporary lifestyles elevate the risk of being overweight and obese. This issue has also become critical for the younger population; particularly in affluent nations and among higher social classes, there is a noticeable rise in obesity rates.

Obesity is a condition characterized by a heightened level of body fat and is linked to various metabolic disorders [2]. The global incidence of obesity is on the rise, carrying serious health repercussions. Numerous factors contribute to obesity, including genetics, metabolic conditions, neuroendocrine imbalances, alongside

dietary and lifestyle choices. Approaches for addressing this disorder encompass preventive measures, alterations in diet and lifestyle, pharmacological treatments, and surgical options.

Globally, the incidence of obesity keeps increasing unchecked due to swift urban expansion in both affluent and developing countries, along with unhealthy lifestyle choices [3]. The condition is also linked with the emergence of significant cardiovascular diseases, diabetes, joint problems, behavioral issues, depression, cancers, and liver diseases. Hence, it is crucial to counteract the rise in obesity by introducing behavioral modifications aimed at reducing the intake of calorie-rich foods and encouraging regular physical activity. Ironically, these strategies often do not assist the larger population suffering from severe obesity. Additionally, it is important to highlight that recent clinical research shows that existing pharmacological treatments may not adequately lower the risk of adverse cardiovascular events in obese patients who also have coexisting conditions such as diabetes, hypertension, or liver diseases like non-alcoholic fatty liver disease, non-alcoholic steatohepatitis, or alcoholic steatohepatitis.

The widespread issue of obesity nationally creates distinct challenges for surgical procedures and anesthetic practices [4]. The body mass index, calculated by dividing weight in kilograms by height in meters squared, provides insight into the extent of obesity. A standard BMI is approximately 21.6 kg/m2; individuals classified as overweight fall between 25 and 30 kg/m2, while those considered obese have a BMI from 30 to 35 kg/m2, and those with extreme obesity exceed 35 kg/m2. Patients with extreme obesity encounter numerous perioperative complications and necessitate assessment in a pre-anesthetic consultation. Key areas of focus should include the upper airway as well as evaluations of cardiovascular, respiratory, metabolic, and gastrointestinal health. Patients with abnormal BMI experience cardiovascular complications, including issues with venous access, high blood pressure, enlarged heart, reduced left ventricular function, and cor pulmonale, and their likelihood of developing ischemic heart disease is double that of individuals with a normal weight. Significant pulmonary complications are linked to extreme obesity, such as limited lung volumes, obstructive sleep apnea, low blood oxygen levels, elevated Paco2, increased hematocrit, and right heart failure. The airway of extremely obese patients can be challenging to secure via mask ventilation due to restricted neck movement and excess fat, necessitating thorough preoperative evaluation. Nearly all major endocrine issues with severe obesity relate to diabetes mellitus effects and require an assessment of blood sugar management prior to surgery. Obesity can also result in atypical fat accumulation in the liver, which leads to increased breakdown of inhaled anesthetics. Morbidly obese individuals might face a greater likelihood of gastric content aspiration and subsequent aspiration pneumonia. Lastly, careful consideration of pain management after surgery is essential.

Metabolic Syndrome

Metabolic syndrome consists of a set of identifiable features that connect insulin resistance and central obesity to a heightened risk of cardiovascular events along with various other health concerns [5]. Central obesity is characterized by an enlarged waistline that corresponds to a higher waist-to-hip ratio. The fat distribution associated with central obesity includes both subcutaneous and intra-abdominal fat, with the intra-abdominal fat (such as visceral or omental fat) being most directly associated with insulin resistance and negative health outcomes.

Numerous labels have been used to describe metabolic syndrome, including insulin resistance syndrome, syndrome X, diabesity syndrome, morbesity syndrome, cardiometabolic syndrome, and dysmetabolic syndrome X (ICD-10 code: E88. 81). The National Institutes of Health defines metabolic syndrome as a collection of risk factors associated with being overweight or obese that elevates a person's likelihood of experiencing heart disease and other health issues like diabetes and stroke.

There are at least four primary theories connecting obesity to insulin resistance.

- In metabolic syndrome, the levels of free fatty acids in the bloodstream are heightened. In cases of obesity, high free fatty acid levels can result in the accumulation of triglycerides in the liver, muscle tissue, and pancreatic beta cells. This accumulation of fat in both the liver and muscles disrupts the signaling of insulin, leading

to a diminished insulin response in these areas. The tissues' absorption of free fatty acids may generate diacylglycerol, which could influence the insulin receptor's secondary messenger signaling. The fat buildup can harm beta cells, resulting in a relative lack of insulin in type 2 diabetes.

- In obesity, muscle becomes infiltrated with a greater amount of fat cells, or "marbled. " These fat cells (adipocytes) produce tumor necrosis factor-alpha (TNF-α) in that localized area, contributing to insulin resistance in skeletal muscle. It is noteworthy that skeletal muscle is the primary site for glucose uptake.
- Adipose tissue does not merely act as a reservoir or transporter for fats and fatty acids. Instead, it plays an active role in modulating insulin sensitivity systemically by generating various adipokines, like adiponectin. A deficit of adiponectin in cases of metabolic syndrome results in decreased insulin sensitivity and promotes both inflammation and atherogenesis.
- The conversion of circulating steroids to cortisol in the adipose tissue via the enzyme 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1) may contribute to hypercortisolism within metabolic syndrome. Despite this evidence, an increasing amount of research suggests that hyperresponsiveness of beta cells could be a driving force behind metabolic syndrome.

BMI

Body weight is controlled physiologically; thus, obesity represents a disturbance within this regulatory mechanism [2]. Obesity is a significant contributor to the risk of developing diabetes mellitus, heart disease, and some cancers. The condition results from an ongoing positive energy balance, often resulting from excessive caloric intake and a rise in sedentary behavior against a backdrop of genetic and epigenetic tendencies.

Obesity is defined as a body mass index (BMI) of 30. 0 or above, and it is categorized as follows:

- Class 1: BMI of 30 to <35
- Class 2: BMI of 35 to <40
- • Class 3: BMI of 40 or greater, also referred to as ‘severe’ obesity

Trigger

Obesity serves as a significant catalyst for a range of metabolic disorders, including type 2 diabetes mellitus (T2DM), cardiovascular conditions, liver dysfunction, and dyslipidemia [6]. Excess weight and obesity contribute to the malfunction of adipose tissue. This tissue's role includes storing and releasing triglycerides to supply energy during fasting, regulating body temperature, and safeguarding mechanical organs. Adipose tissue is recognized as an endocrine gland. The secretion of adipokines, which include bioactive peptides, lipids, and diverse metabolites, happens through structures formed by adipocytes, immune cells, and fibroblasts. Adipokines play a vital part in managing inflammatory responses, maintaining homeostasis, regulating insulin sensitivity and secretion, and overseeing energy storage and consumption, as well as appetite control. Adjustments to immune cell movement toward adipose tissue also occur.

Adipogenesis and the metabolism of adipocytes represent the primary roles of adipokines within adipose tissues. Adipokines influence numerous biological mechanisms, which include adiponectin, leptin, fibroblast growth factor 21 (FGF21), dipeptidyl peptidase 4 (DPP-4), and resistin (related to glucose metabolism); retinol-binding protein 4 (RBP4), chemerin, and omentin (pertaining to insulin sensitivity); tumor necrosis factor-α (TNFα), progranulin, resistin, C-reactive protein (CrP), and interleukin (IL)-1b, (IL)-6, and (IL)-10 (linked to inflammation); insulin-like growth factor-1 (IGF-1), fibronectin, and transforming growth factor-β (TGFβ) (associated with growth); adipsin, serum amyloid A3 (SAA3), and acylation-stimulating protein (ASP) (related to the immune response); as well as various other essential biological activities. Recently, a serpin derived from visceral adipose tissue, known as serpin A12 (vaspin), has gained attention within clinical trials and studies focusing on obesity and diabetes, categorizing it as a novel adipokine in the adipose tissue-derived serpin group. This research centers on the connection between levels of vaspin and type 2 diabetes. In contrast to type 1 diabetes, which is characterized by an autoimmune attack on pancreatic beta cells by T cells leading to an inability to produce insulin and resulting in insulin deficit from early life, vaspin enhances the sensitivity of cells to insulin and does not exhibit a significant correlation with the impairment of insulin production.

Recent cellular and clinical research provides strong evidence supporting a relationship between vaspin concentrations and the occurrence of type 2 diabetes related to insulin resistance.

Disease

Obesity is defined as a medical condition marked by a prolonged imbalance where energy consumption exceeds energy usage [2]. Hormonal imbalance is crucial to both the onset and persistence of excessive body weight. The body's set point is adjusted to a higher level, creating challenges in sustaining weight loss. Caloric intake is the most significant contributor to obesity's development. Diets that are high in calories and energy density lead to significant weight gain. Various environmental elements, including socioeconomic conditions, exposure to endocrine disruptors, and sedentary behavior, heighten the likelihood of developing obesity. Ongoing research aims to uncover the epigenetic, genetic, and developmental factors that contribute to this condition.

The global issue of obesity is on the rise, significantly affecting young adults diagnosed with Type 1 diabetes (T1D) as well [7]. Until recently, the influence of obesity on the onset of T1D had not been a primary research topic, but that is changing with growing interest in the subject. A follow-up study covering 18 years revealed a 47% rise in the rate of overweight individuals, alongside a seven-fold surge in obesity prevalence. Obesity heightens the likelihood of developing T1D and may lead to an earlier onset in those individuals who are predisposed, as evidenced by a recent study using mendelian randomization that identified associations between 23 SNPs (single nucleotide polymorphisms) and the onset of T1D in childhood. Increased body mass, obesity, and insulin resistance elevate the chances of developing T1D; however, there are no long-term studies that have concurrently explored these relationships prior to the clinical onset of diabetes. A significant connection between inflammatory cytokines and adipokines appears to exist between obesity and T1D. It has been observed that obese individuals possess elevated levels of IL-17, IL-23, and leptin, and higher production of IL-17 is noted in the initial stages of T1D. Numerous studies indicate that adipokines such as leptin and resistin may contribute to T1D development, as resistin diminishes the viability of beta cells and is and is found in increased amounts in T1D patients. In similar research involving mouse models, leptin has demonstrated the ability to damage beta cells through its inflammation-promoting effects. Proinflammatory cytokines from pancreatic adipocytes exhibit direct cytotoxicity towards pancreatic islets, while also facilitating the infiltration of Th1 and Th17 cells, which promote ongoing inflammation within the islets by increasing the expression of chemokine ligand (CCL) 5. Obesity raises the likelihood of comorbidities like metabolic syndrome, as well as macrovascular and microvascular diseases in T1D patients; hence, addressing obesity could potentially reduce the onset of T1D and prevent its later complications.

Weight

A substantial amount of research indicates that experiences during pregnancy and early life play a significant role in obesity development [2]. Various studies propose that endocrine-disrupting chemicals (EDCs) may interfere with hormonal and metabolic processes, particularly during early life stages, leading to weight gain tendencies even when calorie consumption is restricted and physical activity is increased. A perinatal environment that promotes obesity plays a role in adult obesity, with specific maternal characteristics, including significant weight gain during pregnancy or pre-existing maternal obesity and diabetes, potentially increasing the risk of obesity in later life.

Many women undergo gestational weight gain along with incremental weight increases in subsequent pregnancies. Those who have been pregnant exhibit a threefold higher increase in the accumulation of visceral fat compared to women who have never borne children. For some women, this trajectory can result in obesity; those who start out overweight or obese are more likely to experience excessive weight gain throughout their pregnancies.

Evidence indicates that the gut microbiome might be involved in the development of obesity [8]. The fibers in the intestines that humans cannot digest are fermented by these microbes into short-chain fatty acids, including butyrate, propionate, and acetate. These fatty acids engage with G protein-coupled receptors known as GPR41 and GPR43 found in enteroendocrine cells, enteric neurons, and enteric leukocytes, promoting positive impacts on glucose regulation and

energy equilibrium, as well as providing local anti-inflammatory benefits. The production of short-chain fatty acids may aid in enhancing the metabolic syndrome by stimulating the release of peptide hormones like peptide YY and glucagon-like peptide-1 (GLP-1), which work to suppress hunger and boost insulin secretion, respectively. In response to a diet low in calories and fiber, the gut microbiota proliferate, disrupting the mucus barrier and triggering metabolic inflammation and increased permeability of the intestines, increasing the risk for obesity.

Gene-environment interactions create a complicated network that manages energy balance and physiological functions associated with body weight. Two neuronal groups within the arcuate nucleus of the hypothalamus, influenced by circulating neuropeptide hormones, regulate energy balance by managing food consumption and energy use. The coordination of short-term and long-term energy balance involves a network of central mechanisms and peripheral signals from the microbiome as well as cells in adipose tissue, stomach, pancreas, and various other organs. Regions of the brain outside the hypothalamus contribute to energy balance regulation by processing sensory inputs, cognitive functions, the pleasurable aspects of eating, memory, and focus.

Treatment

For numerous patients dealing with obesity, losing 5 to 10 percent of body weight can lead to significant clinical improvements in various risk factors, with the reduction in risk seemingly related to the amount of weight lost [9]. The extent of weight loss after one year is closely linked to enhancements in multiple measures, such as blood glucose levels, blood pressure, triglycerides, and HDL cholesterol.

Successful management of obesity necessitates a comprehensive strategy to overcome the body's resistance to shedding pounds. Central to effective weight control are diet, exercise, and changes in behavior. Various eating plans may contribute positively to weight reduction. It is crucial for these suggestions to align with an individual's tastes, as a better adherence to a diet correlates with more significant weight loss outcomes. Guidelines should highlight the consumption of primarily "unprocessed" foods while paying close attention to minimizing items that offer high caloric content without essential nutrients, such as heavily processed foods, sweetened beverages, fast food, snack foods, and desserts. The Mediterranean dietary pattern is favorable for individuals at increased cardiovascular risk because research indicates it lowers the likelihood of major heart-related incidents. A diet with a low glycemic index can help manage appetite and reduce cravings by stabilizing blood sugar levels. Meal replacement plans can enhance weight loss but may lack sustainability over time. Nutrition experts can offer dietary guidance and personalize meal plans.

Sustained modifications in eating habits are essential for weight loss retention, and strategies for behavior modification aid in this process. Focusing on meal planning and self-monitoring is vital, which includes weighing oneself regularly. Some individuals keep a diary of their food consumption to monitor calorie intake. Self-tracking supports behavioral transformation and gives health professionals more information to inform their suggestions. Patients can learn to identify "eating triggers" such as emotional and environmental factors and how to manage or steer clear of them. Maintaining weight can be more difficult than losing it initially, so ongoing follow-up remains important to ensure adherence to the prescribed treatment plan.

Bariatric procedures should be an option for individuals facing severe obesity (BMI ≥40 kg/m2) or moderate obesity (BMI ≥35 kg/m2) with significant health issues, particularly after unsuccessful attempts at various other treatments, maintained at a qualified weight for over three years, able to endure surgery, and without any addictions or significant mental health disorders [10]. Weight-loss surgical options are either restrictive (which reduce the volume of food the stomach can accommodate and slow gastric emptying), such as laparoscopic adjustable silicone gastric banding, or restrictive-malabsorptive, such as Roux-en-Y gastric bypass. Generally, these surgeries result in a 30–35% weight reduction, which can be sustained by about 40% of patients after four years. For many individuals, there is a considerable improvement in accompanying health conditions like type 2 diabetes, hypertension, sleep apnea, dyslipidemia, and cardiovascular issues. The metabolic advantages appear to be a result of both weight loss and physiological changes in gut hormones and fat tissue metabolism.

Possible complications include stenosis at the stoma, ulcers at the margin, and dumping syndrome. Procedures that involve malabsorption necessitate lifelong micronutrient supplementation (including iron, folate, calcium, as well as vitamins B12 and D) and carry a risk of islet cell hyperplasia and low blood sugar.

Prevention

Strategies designed to avert obesity encompass interventions in educational institutions and workplaces that emphasize the adoption of healthy habits related to nutrition and exercise [2]. Effective initiatives include decreasing the intake of foods that contribute to weight gain and updating the traditional food pyramid to promote better plate proportions, such as implementing a Healthy Plate model where half of the plate consists of fruits and vegetables, while the remaining half is allocated to proteins and carbohydrate-rich fibers. Nonetheless, efforts to tackle the obesity crisis through dietary measures have encountered numerous obstacles, particularly the high cost and limited availability of nutritious food choices. Additional methods focus on enhancing access to healthy food alternatives and promoting a more active lifestyle

Conclusion

Obesity is a long-term condition characterized by an excess buildup of fat in the body, along with a rise in body weight. A weight increase of 10% beyond the recommended level is classified as obesity. The prevalence of this condition is escalating and ranks among the foremost health concerns of contemporary society. It impacts numerous organs and systems, heightening the likelihood of heart-related diseases, while also diminishing quality of life. Obesity can occur at any age, being equally prevalent in both genders during puberty but more frequently observed in women post-puberty. In children prior to puberty, obesity is less common, but it can still occur. The primary contributors to obesity include overeating and insufficient physical activity.

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