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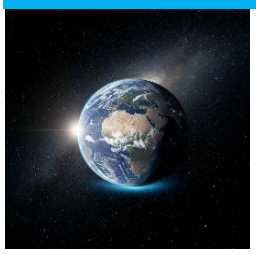

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Obesity — the major groups of its complications

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Abstract

Background: Obesity has evolved from a simple metabolic condition into a complex, chronic systemic disease that has reached pandemic proportions in the 21st century. It serves as a primary driver for a multitude of chronic pathologies across virtually every human organ system.

Aim: This narrative review aims to provide a comprehensive analysis of the major groups of obesity complications, exploring their pathophysiological mechanisms and epidemiological impact across the lifespan, with a focus on pediatric development and geriatric well-being.

Material and methods: A systematic search of PubMed, MEDLINE, Google Scholar. Selection criteria prioritized peer-reviewed articles, meta-analyses, and cohort studies published between 2014 and 2026, focusing on the mechanisms of metabolic, cardiovascular, respiratory, musculoskeletal, and psychosocial impairment.

Results: Metabolic complications of obesity include type 2 diabetes and hyperuricemia, while cardiovascular effects range from hypertension to coronary artery disease. Respiratory health is compromised by mechanical and inflammatory pathways, leading to obstructive sleep apnea and hypoventilation. Pediatric studies demonstrate that excessive weight significantly accelerates skeletal and dental maturation, while geriatric data highlight severe declines in self-esteem and social integration.

Conclusions: The complications of obesity are intrinsically linked to chronic low-grade inflammation and hormonal dysregulation. Management must move beyond simple weight loss to address the systemic inflammatory environment and the varied psychosocial consequences.

Key words: obesity, metabolic syndrome, pediatric development, postural balance, cardiovascular disease, 21st-century pandemic.

Introduction

Obesity has emerged as the most significant public health challenge of the contemporary era, earning the designation of the pandemic of the 21st century.[1] This condition, characterized by an excessive and abnormal accumulation of adipose tissue, poses a pervasive threat to global health systems and individual longevity.[2] According to the World Health Organization (WHO), the prevalence of this condition has nearly tripled since 1975, with current estimates suggesting that more than 13% of the world's adult population is clinically obese.[1] The diagnostic threshold for obesity in adults is generally accepted as a Body Mass Index (BMI) of ≥ 30 kg/m², calculated as the ratio of mass to the square of height:

$$BMI = \frac{m}{h^2}$$

where "m" represents the individual's mass in kilograms and "h" represents the height in meters.[3] When the BMI exceeds 40 kg/m^2, the mortality risk is reported to increase by approximately 100% compared to individuals within the normal weight range.[1]

The complexity of obesity lies in its multifactorial etiology, which integrates genetic predisposition, environmental influences, and behavioral choices.[1] The fundamental driver

remains an energy imbalance where caloric intake consistently exceeds expenditure, often fostered by an obesogenic environment that promotes the consumption of ultra-processed, energy-dense foods and sedentary behavior.[6] Furthermore, the COVID-19 pandemic significantly exacerbated this trend. Lockdowns, school closures, and increased emotional eating led to a measurable rise in BMI across all age groups, particularly among children and adolescents who were deprived of structured physical activity and social interaction.[8]

The consequences of excessive adiposity are observed throughout the entire organism, disrupting homeostasis and leading to a wide array of chronic illnesses.[1] These complications are typically categorized into major groups: metabolic and endocrine, cardiovascular, respiratory, musculoskeletal, and psychosocial.[1] By understanding the specific mechanisms—such as the role of pro-inflammatory cytokines, adipokines like leptin, and mechanical stress—clinicians can better address the systemic nature of this disease. This review synthesizes the current medical knowledge on these major complication groups, emphasizing the need for early detection and comprehensive therapeutic approaches.

Material and methods

The literature search for this narrative review was conducted across several major scientific databases, including PubMed, MEDLINE, and Google Scholar. The search strategy utilized a combination of Medical Subject Headings (MeSH) and relevant keywords such as "obesity complications," "metabolic syndrome," "pediatric obesity," "skeletal maturation," "dental development," "postural balance," "geriatric obesity," and "cardiovascular disease."

The inclusion criteria were defined as:

1. Peer-reviewed original research, meta-analyses, and systematic reviews.
2. Studies published primarily between 2014 and 2026 to ensure clinical relevance.
3. Articles focusing on the pathophysiological mechanisms of obesity-related comorbidities.
4. Reports from international health organizations including the WHO and CDC.

The exclusion criteria included non-peer-reviewed content, anecdotal case reports with fewer than five participants, and studies exclusively using animal models without direct human clinical application. A total of 30 sources were selected for the final synthesis, including five mandatory publications from JEHS that provided pivotal data on skeletal maturation in children, geriatric psychosocial impacts, and postural balance. The collected data was analyzed and categorized into thematic sub-chapters to provide a clear, evidence-based narrative of the state of knowledge in early 2026.

The Epidemiological Landscape and Global Impact

Obesity is no longer restricted to affluent Western nations; it has become a global phenomenon affecting over 1.9 billion adults who are overweight and 650 million who are obese.[1] In many developing regions, the incidence of obesity is rising at a rate faster than that observed in developed countries. Statistical data indicates that in Europe and North America, more than half of the population is classified as having excessive body weight.[1] Global adult obesity prevalence currently stands at approximately 13%, having tripled since the mid-1970s.[3] The economic impact of these figures is profound. Obesity-related healthcare expenditures are draining national budgets, driven largely by the management of its chronic complications rather than the weight itself.[14] For example, worldwide estimates suggest that between 2% and 7% of all healthcare expenditures are attributed directly to preventing and treating obesity, though this can rise to 20% when complications and associated diseases are fully accounted for.[1] In the United States, pediatric obesity rates rose from 15.1% in 2018 to 17.3% in 2020, a shift largely attributed to the lifestyle disruptions of the COVID-19 pandemic.[1] The problem of obesity among children and youth in the world has become a serious health issue because of the long-term, negative consequences that profoundly affect growth and health.[6]

Metabolic and Endocrine Complications

The endocrine function of adipose tissue is the primary driver of metabolic complications. Adipocytes are not passive storage units; they secrete various bioactive substances, including adipokines such as leptin, adiponectin, and resistin, alongside pro-inflammatory cytokines including TNF-alpha, IL-6, and C-reactive protein.[1]

Type 2 Diabetes and Insulin Resistance

Obesity is the single most significant risk factor for the development of Type 2 Diabetes Mellitus (T2DM). Excessive visceral fat leads to a chronic state of lipotoxicity and inflammation, which interferes with the insulin signaling pathway.[1] In individuals with abdominal obesity, often referred to as the "apple shape," the release of free fatty acids into the portal circulation promotes hepatic insulin resistance and impairs peripheral glucose uptake.[1] Research indicates that the risk of diabetes comorbidity is approximately 3.4 times higher in obese populations compared to those with a normal BMI.[17]

Hyperuricemia and Gout

Recent studies have highlighted a strong correlation between childhood obesity and hyperuricemia, defined as elevated serum uric acid.[18] This condition is increasingly common among youth and is driven by multiple mechanisms including insulin resistance and dietary factors. High insulin levels enhance the expression of renal transporters such as URAT1 and GLUT9, which increases the reabsorption of uric acid in the kidneys.[18] Furthermore, high fructose consumption stimulates de novo purine synthesis, leading to increased uric acid production. Hyperuricemia serves as an independent risk factor for hypertension and chronic kidney disease in the pediatric population.[18]

Metabolic Syndrome and NAFLD

Metabolic syndrome represents a cluster of conditions including hypertension, hyperglycemia, dyslipidemia, and central obesity.[21] A major component is Non-Alcoholic Fatty Liver Disease (NAFLD), which can progress from simple steatosis to cirrhosis. This "metabolic syndrome" also affects the pancreas, where fat deposition is identified as a potential factor in worsening insulin resistance and furthering the progression toward T2DM.[17]

Cardiovascular System Pathophysiology

Cardiovascular diseases (CVD) remain the leading cause of mortality among obese individuals. The heart is forced to accommodate an increased blood volume and total peripheral resistance, leading to structural and functional remodeling.[1]

Hypertension and Endothelial Dysfunction

Obesity-induced hypertension is mediated by the activation of the Renin-Angiotensin-Aldosterone System (RAAS) and the sympathetic nervous system.[23] Adipose tissue locally produces angiotensinogen, and elevated leptin levels directly stimulate the brain's sympathetic centers, leading to sodium retention and chronic vasoconstriction. Furthermore, atherosclerosis is accelerated by the buildup of low-density lipoprotein (LDL) cholesterol and the presence of inflammatory markers that damage the arterial lining.[22]

Heart Failure and Coronary Artery Disease

Heart failure in obesity often presents as heart failure with preserved ejection fraction (HFpEF). In these patients, excess adiposity increases the mechanical load on the heart and promotes myocardial fibrosis.[22] The risk of myocardial infarction is significantly elevated due to the combination of coronary artery disease and a pro-thrombotic state where obesity increases the likelihood of venous thromboembolism.[11]

Respiratory System Complications

The respiratory system is uniquely vulnerable to both the mechanical and metabolic effects of obesity. Excessive developed adipose tissue accumulates around the thoracic cage and abdominal cavity, causing a decrease in the compliance of the chest walls.[25] This increases the energy required for the work of respiratory muscles, leading to a pattern of rapid, shallow breathing and increased dyspnea.

In terms of clinical parameters, spirometry typically reveals significant reductions in Forced Vital Capacity (FVC) due to restricted chest wall expansion. The Forced Expiratory Volume in one second (FEV1) is also decreased as a result of airway narrowing at lower lung volumes. More notably, Functional Residual Capacity (FRC) and Expiratory Reserve Volume (ERV) show significant declines because the upward displacement of the diaphragm and the closure of small airways in the basal lung regions restrict the air volume remaining in the lungs after a normal expiration.[25]

Obstructive Sleep Apnea (OSA) is another major complication, characterized by repetitive episodes of upper airway collapse during sleep. Approximately 50% to 60% of obese individuals suffer from comorbid OSA.[26] A more severe manifestation is Obesity Hypoventilation Syndrome (OHS), defined by a BMI $> 30 kg/m^2$, daytime hypercapnia, and nocturnal hypoventilation not explained by other diseases. Approximately 90% of OHS patients also suffer from OSA.[25]

Musculoskeletal and Postural Complications

The traditional view that obesity protects against bone loss has been largely debunked. Modern research indicates that while bone mass may be higher, bone quality is often compromised, and the mechanical load on joints is severe.[28]

Osteoarthritis and Joint Health

Obesity is the most important preventable risk factor for osteoarthritis, particularly of the knees and hips. The damage occurs through increased mechanical loading on articular cartilage and metabolic pathways where adipokines like leptin play a catabolic role.[28] Leptin enhances the production of matrix metalloproteinases including MMP-1 and MMP-13 in the cartilage, which actively destroy the joint matrix.

Postural Balance and Stability

Maintaining a vertical posture requires a complex integration of the vestibular, visual, and proprioceptive systems. Excessive body weight acts as a significant disruptor of this equilibrium.[30] Pankanin (2018) demonstrates that the specific distribution of excess body fat disturbs balance control by shifting the center of gravity.[30] This requires the nervous system to process more information and initiate more frequent corrections to maintain stability.

The balance deficits in obesity are characterized by increased postural sway, where individuals exhibit a greater range of body movement when standing still, especially without visual feedback.[30] Furthermore, the muscles responsible for maintaining a vertical posture are often weakened in obese subjects, compromising the efficiency of the motor apparatus.[30] Tests such as the "Flamingo test"—standing on a narrow slat on one leg—reveal that children with excessive weight perform significantly worse than their healthy peers.[32]

Pediatric Maturation and Developmental Complications

Obesity in childhood does not merely affect weight; it alters the fundamental timing of human biological development. Research conducted by Olszewska et al. (2018) indicates that overweight and obesity affect skeletal maturation and dental development in children and adolescents.[2]

In a retrospective study of 77 adolescent orthodontic patients aged 11–16 years, the obese group showed a significant acceleration in biological maturation compared to normal-weight controls.[2] Specifically, obese children exhibited significantly greater mandible length, corpus length, and midfacial length.[2] These children often possess an advanced dental age, meaning they have a higher number of erupted teeth compared to chronological age-matched peers, which increases the susceptibility to malocclusions and caries.[2] This acceleration of maturation is linked to elevated levels of leptin and Insulin-like Growth Factor-1 (IGF-1), which are

common in pediatric obesity.[33] This has critical implications for pediatric dentistry, as the timing of orthopedic treatments must be recalculated based on biological age rather than chronological age.[2]

Psychosocial and Geriatric Complications

As individuals age, the physical burdens of obesity are compounded by psychological and social difficulties. A study by Szkutnicka et al. (2018) analyzed the impact of obesity on the life activity and self-esteem of the elderly over the age of 65.[34]

The results of this analysis were striking: 92% of elderly respondents were not satisfied with their physical appearance, and 100% of participants believed they were perceived badly by other people because of their weight.[34] Furthermore, 78% of these individuals assessed their lifestyle as "not very active," often due to physical pain and low self-esteem.[34] Excessive body mass has a detrimental effect on self-esteem and frequently leads to the elderly withdrawing from social life, which can accelerate cognitive decline and increase the risk of depression.[1] There is a bidirectional relationship between obesity and depression; obese children, for example, have a 1.32 times higher risk of depression compared to normal-weight children.[8]

Therapeutic and Management Strategies

Managing obesity and its complications requires a combination of lifestyle, pharmacological, and surgical interventions. The emergence of GLP-1 receptor agonists and dual agonists like tirzepatide has provided new tools for management. Tirzepatide has demonstrated an average weight reduction of 20.9% in patients without type 2 diabetes.[35] These medications not only aid in weight loss but also improve insulin sensitivity and lipid profiles.[35] For individuals with morbid obesity ($> 40 kg/m^2$), bariatric surgery remains the most effective option, as it has been shown to reduce cardiovascular mortality and lead to the long-term remission of type 2 diabetes.[1] Regardless of medical or surgical intervention, physical activity remains foundational. The WHO recommends at least 150–300 minutes of moderate aerobic activity per week to reduce visceral fat and prevent systemic diseases.[23]

Discussion

The synthesis of evidence suggests that obesity is the primary engine of a multi-organ pathological cascade. A recurring theme across all major complication groups is the role of chronic, low-grade inflammation. This "metabolic inflammation" links seemingly unrelated conditions such as asthma, osteoarthritis, and type 2 diabetes.[15]

One of the most profound insights is the impact of obesity on human growth and stability. The work of Olszewska et al. (2018) and Pankanin (2018) highlights that the biological cost of obesity is paid early in life. By accelerating skeletal maturation, obesity reduces the window for pediatric orthopedic interventions, while simultaneously making the child more prone to injury through impaired postural balance.[2] The child becomes physically more mature in dimensions but biomechanically and functionally less stable.[30]

Furthermore, the psychosocial data regarding the elderly suggests that obesity exacerbates the difficulties of aging. The "vicious cycle" of obesity—where pain leads to inactivity, which leads to further weight gain and social withdrawal—is particularly evident in the senior population.[34] This underscores the need for "secondary prevention," focusing on detecting and treating obesity early to prevent these devastating consequences.[1] Finally, the impact of the COVID-19 pandemic acted as an accelerant for the epidemic, particularly among youth, leading to lasting shifts in standardized BMI scores.[8]

Conclusions

Obesity is a systemic, multi-faceted disease that serves as the root cause for most modern chronic illnesses. Its complications are not merely secondary symptoms but are integrated into a web of inflammatory and mechanical failures. The 21st-century pandemic of obesity affects the individual from the dental chair as a child to the social circles of the elderly. The major groups of complications—metabolic, cardiovascular, respiratory, musculoskeletal, and psychosocial—are each significant enough to reduce life expectancy. However, they rarely occur in isolation. Pediatric obesity is especially concerning as it triggers premature biological aging and balance deficits that persist into adulthood. The impact on the elderly suggests that obesity is a major barrier to a high quality of life in late adulthood. Future interventions must prioritize primary prevention through public health policies and early education, while comprehensive management plans must address the inflammatory and mechanical burdens of the disease.

Disclosure

Suplementary Materials

Author's Contribution

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Formal analysis: Michał Rucki, Nikodem Cieleban, Mateusz Kowalski

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Writing-review and editing: Nikodem Cieleban, Michał Rucki, Magdalena Modliborska
Supervision: Aleksandra Maria Rucka, Mateusz Kowalski

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