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Complications of Adult Obesity: Difficulties and Clinical Implications

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ABSTRACT:

The complications associated with adult obesity are overwhelming nationalhealthcare systems. No country has yet implemented a successful population-level strategyto reverse the rising trends of obesity. This article presents epidemiological data on thecomplications of adult obesity and discusses some of the challenges associated withmanaging this disease at a population and individual level.

Keywords: complications, epidemiology, morbidity, obesity.

INTRODUCTION:

In 2014, 10.8% of men (266 million) and 14.9% of women (375 million) were predicted to be adults who had adult obesity (body mass index (BMI) >30kg/m2) in the world. When compared to global statistics from 1975, when 3.2% of men and 6.4% of women were obese, this has increased by more than twice as much. 18% of men and 21% of women will be obese by 2025 if this trend continues. [1] Except for morbid obesity (BMI > 40 kg/m2), which has continued to climb since 2006, the rate of adult obesity has remained steady in many affluent nations since that time. [2] In developing nations, the prevalence of obesity is increasing to levels comparable to those in the West. [3] To achieve the overarching goal of preventing premature death from the four most common non-communicable diseases — cardiovascular disease (CVD), diabetes, cancer, and chronic respiratory disease — the World Health Organization (WHO) has set governments around the world the challenge of preventing further increases in obesity by 2025. [4]

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Epidemiological information about the complications of adult obesity is presented in the current review, along with some of the difficulties in treating this condition on both a population and an individual level.

Obesity, mortality and BMI

The most frequent causes of death are cardiovascular disease (CVD) and cancer, and obesity, as measured by BMI, is linked to an increased risk of all-cause mortality. 5–8 All-cause mortality was found to be lowest between a BMI of 20–25 kg/m2, but significantly increased just below this range and throughout the overweight/obese categories, according to a meta-analysis of 239 prospective studies involving 10.6 million people from Asia, Australia, New Zealand, Europe, and North America. [8] This finding suggests a J-shaped relationship between BMI and mortality. There are ethnic disparities in the BMI ranges that define overweight and obesity, particularly between Caucasian and Asian populations, which is a reflection of the higher risk of cardiometabolic problems in the latter population at a lower BMI. 9 Although waist circumference (WC) or waist-to-hip ratio (WHR) are superior markers of abdominal obesity, BMI is still the simplest and most widely used anthropometric tool for diagnosing obesity. [10,11]

Combining BMI with WC or WHR will more accurately reflect total body fat distribution than BMI alone and could aid in the earlier detection of people with metabolic syndrome. Given that most people are aware of their waist circumference, this measure may be easier to self-report than height and weight, which are frequently reported incorrectly. [12]

Mechanisms by which obesity causescomplications

Obesity's characteristic excess adiposity can have consequences due to its effects on the anatomy and metabolism.

Anatomical effects: Obstructive sleep apnea (OSA), obesity hypoventilation syndrome (OHS), and osteoarthritis, particularly in weight-bearing joints, can all be caused by an increase in fat tissue. [16–18] Additionally, oesophageal illnesses such gastro-oesophageal reflux disease (GORD) and Barrett's oesophagus are linked to elevated intra-abdominal pressure. [19] By storing extra calories as triglycerides through adipocyte hyperplasia and hypertrophy, subcutaneous adipose tissue acts as a "metabolic sink," preserving the health of lean visceral organs like the heart, kidney, liver, and pancreas. Obesity is linked to diastolic heart failure, chronic kidney disease (CKD), nonalcoholic fatty liver disease (NAFLD), and type 2 diabetes mellitus. However, if subcutaneous adipose tissue capacity is exceeded, hypertrophied adipocytes rupture, inducing inflammation, and triglycerides are deposited within visceral adipose tissu0 (T2DM). [20-21]

Metabolic effects: Tumor necrosis factor alpha (TNF-), interleukin (IL)-1, and IL-6 are proinflammatory cytokines that are abundant in visceral adipose tissue and are linked to infectious, malignant, and cardiometabolic disorders in obese people. [20] Increased levels of

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free fatty acids and lipid intermediates like ceramides have been linked to lipid-induced cellular insults (lipotoxicity), which are also implicated in cardiometabolic illnesses like insulin resistance, NAFLD, and cardiovascular disease (CVD). [22] Two other important mechanisms that link obesity to CVD are endothelial dysfunction and chronic inflammation. [23]

Type 2 diabetes mellitus: In 2013, 56.3 million adults in Europe, or 8.5% of the total population, had diabetes mellitus. [24] According to the most recent statistics, 4.7 million people in the United Kingdom (UK), or 6% of the population, have diabetes, with 90% having T2DM. By 2025, it is predicted that 5 million people in the UK would have diabetes. [25]

Because the risk of T2DM rises with BMI, the obesity and T2DM epidemic during the past few decades is referred to as "diabesity." According to a recent population study involving 2.8 million UK people between 2000 and 2018, individuals with a BMI of 30-35 kg/m2 had a five times higher risk of developing T2DM, while those with a BMI of 40-45 kg/m2 had a 12 times higher risk. [26]

Extra hepatic triglycerides are carried to all organs by extremely low-density lipoproteins, including the pancreatic beta-cells. Over the course of several years, this causes progressive pancreatic beta-cell dedifferentiation, which is followed by a relatively sudden onset of clinical diabetes. [27] Data from the Counterpoint, Counterbalance, and DIRECT trials have shown that a very low calorie diet (600-853kcal/day) for 8 weeks to achieve weight loss of 15 kg was effective in remission of T2DM and improvements in liver and pancreatic fat using magnetic resonance imaging. [28–30]

Cardiovascular disease: Every year, 17.9 million individuals worldwide die from CVD, which accounts for 31% of all fatalities. Globally, ischemic heart disease and stroke are the two leading causes of death. [31]

Coronary heart disease: Independent of age, gender, ethnicity, smoking status, or CVD risk factors, a case-control research involving 27,000 participants from 52 countries showed that WHR was the best predictor of myocardial infarction (MI) (hypertension, diabetes, dyslipidaemia). Across ethnic groups, there was a lower and less consistent correlation between BMI and MI. [32] After controlling for BMI, smoking, hypertension, and hypercholesterolemia, the EPIC-Norfolk prospective cohort study, which included 24,508 UK men and women tracked over 9.1 years, discovered that WHR was more consistently and robustly predictive of coronary heart disease (CHD). [33] Obesity is undoubtedly significantly linked to CHD, however abdominal obesity measures are more accurate predictors than BMI. [34] Due to ectopic visceral fat that promotes chronic inflammation and takes part in all stages of atherosclerosis, including acute thrombosis, the distribution of fat independently mediates the connection between obesity and CHD. [35]

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Stroke: Obesity raises the risk of stroke, although the link between obesity and ischemic stroke is stronger and more reliable. A meta-analysis of 25 studies with 2,247,961 participants from Western and Eastern countries revealed that those with a BMI greater than 30 kg/m2 had a 64% higher risk of ischaemic stroke (relative risk (RR) 1.64, 95% confidence interval (CI) 1.36-1.99) and a 24% higher risk of hemorrhagic stroke (RR 1.24, 95% CI 0.99-1.54), but not a statistically significant, increase in risk. [36] Obesity and ischaemic stroke are linked by traditional, modifiable CVD risk factors as well as independent mechanisms involving proinflammatory cytokines, low levels of adiponectin, and a prothrombotic state (hyperfibrinogenaemia, hyperviscosity), which promote atherosclerosis and endothelial cell dysfunction. [37,38] Obesity and hemorrhagic stroke have a less reliable correlation. [39]

Gastrointestinal complications: Obesity has a number of gastrointestinal and hepatobiliary consequences, many of which are prevalent and manifest earlier than cardiometabolic problems. [19] Therefore, routine weight reduction intervention should include checking for obesity in patients with gastrointestinal and hepatobiliary illness.

Non-alcoholic fatty liver disease: The estimated prevalence of NAFLD is 25.2% globally and 23.7% in Europe40, although it is difficult to determine the true incidence because different research use different diagnostic standards. Over the past forty years, obesity has increased concurrently with the frequency of NAFLD. 40 A meta-analysis of 20 studies, 17 from Asia and 3 from the West, with 12,065 cases and 33,693 controls each, revealed that the risks of developing NAFLD rose by 3–10% for every inch of waist circumference and by 13–38% for every unit of BMI. 41 Even while BMI and WC were both independently linked to NAFLD, measures of abdominal obesity were better predictors and remained so even after BMI was taken into account. This may help to explain why some people with normal BMIs might acquire NAFLD, a condition that is more prevalent in rural parts of some Asian nations (25–30%) than it is in the US and Europe (10–20%). 41 Therefore, to determine the risk of NAFLD, both BMI and WC or WHR should be included. While longitudinal studies indicate that NAFLD precedes the metabolic syndrome and T2DM, NAFLD is thought to be the hepatic manifestation of the metabolic syndrome42. 43 Due to the increased risk of T2DM, hypertension, dyslipidaemia, and CKD caused by NAFLD, it should come as no surprise that CVD is the main cause of death in this patient population. 10,11 The risk of non-alcoholic steatohepatitis (NASH), which can lead to liver cirrhosis, hepatocellular carcinoma (HCC), decompensated liver cirrhosis, and mortality, is present in up to one-third of NAFLD patients.

Biliary complications: The likelihood of gallbladder disease rises with obesity. A systematic evaluation of 17 prospective studies with 1,921,103 individuals produced a risk ratio (RR) for a 5-unit rise in BMI of 1.63 and an RR for a 10-unit increase in waist circumference of 1.46. 46 From the lower to the high limit of the normal BMI range (18.5–24.9kg/m2), there was an almost two-fold increased risk of gallbladder disease, which implies that even modest increases in adiposity increase risk. [46]

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Oesphageal complications: Oesophageal diseases and obesity are related to one another. Obesity raises the risk of GORD, and meta-analyses reveal a beneficial relationship between GORD and BMI. [47,48] The effects of GORD (oesophitis, Barrett's oesophagus, andadenocarcinoma) are independently predicted by central obesity. [49]

Respiratory

Obstructive sleep apnoea: The most frequent risk factor for the emergence of OSA is obesity.

Class I and III obesity were linked to a 5-times and a 22-times greater risk of OSA, respectively, according to observational data from 2.8 million UK adults26, suggesting that the risk of OSA rises significantly with increasing BMI.Untreated OSA can result in excessive daytime somnolence, have a detrimental impact on work performance, raise the risk of CVD, and jeopardise a driver's licence if it interferes with driving. [50,51]

Obesity hypoventilation syndrome: Obesity-related hypoventilation syndrome (OHS) is characterised by a combination of obesity (BMI>30 kg/m2), daytime hypercapnia (pCO2>6 kpa), and sleep disordered breathing that is not brought on by other disorders linked to alveolar hypoventilation. 17 8.5% of OSA patients and 19-31% of obese patients are thought to have OHS, respectively. [55,56] Leptin resistance, which results in central hypoventilation, a compromised compensatory response to hypercapnia, and compromised respiratory mechanics brought on by obesity may all play a role in the pathogenesis of OHS. [57]

The efficacy of pharmaceutical therapy in treating OSA and OHS has not been established. [16] Despite the fact that data on OHS is scarce due to the associated pulmonary and cardiac problems, weight loss in this population of patients with chronic cardiorespiratory disease might be difficult. Bariatric surgery is an effective treatment for OSA and indices of sleep quality. [17] There are currently no randomised control studies supporting bariatric surgery as a treatment for OHS. [59]

Cancer

Obesity is the second-leading avoidable cause of cancer in the UK, behind smoking, and maintaining a normal weight may stop 22,800 new cases from occurring there each year. [60] The International Agency for Research on Cancer came to the conclusion that 11% of colon cancers and 10% of post-menopausal breast cancers were caused by obesity in 2001. The risks related to BMI alone were 25%, 37%, and 39% for kidney, lower oesophageal adenocarcinoma, and endometrial cancer, respectively. [61]

Obesity and cognition: Obesity-related issues that raise the risk of dementia and Alzheimer's disease include cardiovascular risk factors such T2DM, dyslipidemia, and hypertension. [21]

Genitourinary: Because it is linked to two key CKD risk factors, diabetes mellitus and hypertension, obesity is a significant avoidable risk factor for the development of CKD.

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Musculoskeletal: In weight-bearing joints, especially the knee, obesity is a known risk factor for the onset and progression of osteoarthritis. 18 Every 2 units of BMI increase increases the likelihood of developing knee osteoarthritis by 36%, and individuals who are obese experience more severe joint deterioration.

Psychosocial: In settings related to education, health, and employment, obese people frequently face stigma. Obesity discrimination as a result of this has increased by 66% over the last ten years, with prevalence rates that are comparable to those of racial discrimination. [62] Discrimination can lead to low self-esteem and a negative body image, which can have an adverse effect on physical activity participation. [63] Obesity and psychiatric comorbidity are linked.

Challenges

Due to the complicated aetiology of obesity, both population-wide and individualised prevention and treatment strategies must be comprehensive. [64] The social ecology model can offer a framework to help identify the individual and social factors that contribute to obesity, which will aid in the creation of solutions. [65] In fact, both primary and secondary obesity prevention require input and cooperation from a variety of organisations, including the government, policymakers, legislative authorities, and healthcare system. A summary of a few interventions that have been used in several nations, placed on a modified social ecology model. To date, no nation has successfully devised a population-level approach to counter the rising trends of obesity. [1]

CONCLUSION:

The risk of the most prevalent non-communicable chronic diseases of the twenty-first century rises due to the multisystemic condition known as obesity. [21,57] People are becoming obese at younger ages, and it is likely that they will experience morbidity for longer. 2,118 Due to the risk of irreversible disease progression brought on by multi-organ damage, this will be a challenge for clinicians. Priority should be given to early detection of obese people by straightforward anthropometric measurements in order to launch effective interventions to stop morbidity and the ensuing medical and financial consequences. [66]

Taking on obesity calls for a holistic strategy.Instead of individuals, governments and policymakers have the power to alter the food environment through regulation, taxes, and limiting both adult and child access to high-calorie processed foods. Patients with obesity who experience weight-based prejudice demand laws and policies that work to end such discrimination. This will contribute to changing the prevalent social stigma that is fueled by the idea that obese people are to blame for their illness.It is possible to avoid discrimination and make referrals to specialised weight management facilities where a multidisciplinary team can provide individualised patient care by dispelling this illusion with scientific knowledge.

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