

Opinion

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Diabetes and Obesity: A Closely Interlinked Metabolic Challenge

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Abstract

Diabetes mellitus and obesity are two of the most important metabolic disorders worldwide, and their prevalence has increased considerably over the past few decades. Obesity, particularly excess visceral adiposity, is a major modifiable risk factor for the development of Type 2 Diabetes Mellitus (T2DM). The relationship between the two conditions is complex and involves insulin resistance, chronic low-grade inflammation, altered adipokine secretion and abnormalities in lipid metabolism. At the same time, diabetes and some of its treatments may contribute to further weight gain, creating a vicious cycle. Management therefore requires an integrated approach focusing not only on glycaemic control but also on weight reduction and reduction of cardiovascular and metabolic risk. Recent advances in pharmacotherapy, particularly glucagon-like peptide-1 receptor agonists and glucose-dependent insulinotropic polypeptide/glucagon-like peptide-1 receptor agonists, have provided effective options for addressing both hyperglycaemia and obesity. Early recognition and comprehensive management of obesity in individuals with diabetes may improve long-term outcomes.

Introduction

Obesity and T2DM frequently coexist and represent a major global health burden. The increase in obesity has paralleled the rising prevalence of T2DM, making excess body weight one of the most important preventable risk factors for diabetes. According to the International Diabetes Federation, hundreds of millions of adults are currently living with diabetes worldwide, with T2DM accounting for the vast majority of cases [1]. The association between obesity and diabetes is particularly strong when excess adipose tissue is concentrated in the visceral compartment. Although obesity does not inevitably lead to diabetes, increasing adiposity substantially raises the risk, especially in individuals with a genetic or metabolic predisposition. The coexistence of obesity and diabetes also increases the risk of cardiovascular disease, chronic kidney disease, non-alcoholic fatty liver disease and other complications.

Pathophysiological Link Between Obesity and Diabetes

Insulin resistance is one of the major mechanisms linking obesity with T2DM. In obesity, expansion of adipose tissue is accompanied by increased release of free fatty acids and alterations in adipokines and inflammatory mediators. These

changes interfere with insulin signalling in skeletal muscle and the liver, resulting in reduced glucose uptake and increased hepatic glucose production. Adipose tissue is not simply a storage site for excess energy but also functions as an endocrine organ. Obesity is associated with increased production of pro-inflammatory mediators such as tumour necrosis factor- α and interleukin-6, while beneficial adipokines such as adiponectin are reduced. This chronic, low-grade inflammatory state contributes to insulin resistance. Initially, pancreatic β -cells compensate for insulin resistance by increasing insulin secretion. With persistent metabolic stress, however, β -cell function progressively declines. The combination of insulin resistance and inadequate insulin secretion eventually results in persistent hyperglycaemia and clinical T2DM [2]. The relationship is bidirectional. Diabetes-related lifestyle limitations and certain glucose-lowering therapies, particularly insulin and sulfonylureas, can promote weight gain. Consequently, obesity and diabetes may reinforce each other over time.

Clinical Impact of Weight Loss

Weight reduction is an important component of diabetes management. Even modest weight loss can improve insulin

sensitivity and glycaemic control, while greater reductions in body weight may produce more substantial metabolic benefits. The Diabetes Remission Clinical Trial (DiRECT) demonstrated that substantial weight loss achieved through a structured low-calorie dietary intervention could lead to remission of T2DM in a proportion of individuals, particularly when weight loss was maintained [3]. Lifestyle modification remains the foundation of management. Dietary interventions should be individualized, with emphasis on reduction of energy-dense foods, refined carbohydrates and excessive saturated fat while encouraging vegetables, whole grains, legumes, lean protein and other nutrient-rich foods. Regular physical activity improves insulin sensitivity even when substantial weight loss does not occur. However, long-term maintenance of weight loss with lifestyle intervention alone can be difficult. This has led to increasing interest in pharmacological therapies that address both obesity and diabetes.

Emerging Pharmacological Approaches

The treatment landscape has changed considerably with the development of incretin-based therapies. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) improve glucose-dependent insulin secretion, suppress glucagon secretion and delay gastric emptying. They also reduce appetite and food intake, resulting in clinically meaningful weight loss. Semaglutide has demonstrated significant reductions in body weight and improvements in glycaemic parameters in individuals with obesity and/or T2DM. In the STEP trials, semaglutide 2.4 mg once weekly, when combined with lifestyle intervention, produced substantial weight loss in people with obesity [4]. More recently, tirzepatide, a dual glucose-dependent insulinotropic polypeptide (GIP) and GLP-1 receptor agonist, has demonstrated even greater effects on body weight and glycaemic control. In the SURMOUNT-1 trial, tirzepatide produced marked and sustained weight reduction among adults with obesity without diabetes [5]. Studies in people with T2DM have similarly demonstrated improvements in HbA1c along with significant weight loss. These therapies are particularly relevant because they address two major components of the metabolic problem simultaneously. Sodium-glucose cotransporter-2 inhibitors also have an important role in selected patients with T2DM, particularly because of their cardiovascular and renal benefits, although their weight-reducing effect is generally more modest.

Cardiovascular and Metabolic Considerations

Obesity in diabetes is associated with a high burden of cardiovascular risk factors, including hypertension, dyslipidaemia

and systemic inflammation [6]. Therefore, treatment should extend beyond glucose lowering. Contemporary diabetes management emphasizes a comprehensive approach incorporating weight management, blood pressure control, lipid lowering and reduction of cardiovascular and renal risk. Importantly, treatment should be individualized. Choice of antidiabetic therapy should consider body weight, cardiovascular disease, heart failure, chronic kidney disease, hypoglycaemia risk, cost and patient preference. Therapies associated with weight gain should generally be avoided or minimized when weight reduction is a major treatment goal and suitable alternatives are available.

Conclusion

Diabetes and obesity are closely interconnected disorders with overlapping metabolic and inflammatory mechanisms. Excess adiposity promotes insulin resistance and increases the risk of T2DM, while diabetes and some of its treatments may further promote weight gain. Consequently, weight management should be regarded as an integral component of diabetes care rather than a separate therapeutic goal. Lifestyle modification remains fundamental, but newer pharmacological therapies such as GLP-1 receptor agonists and dual GIP/GLP-1 receptor agonists have substantially expanded treatment options. A patient-centered strategy that combines sustained weight reduction with effective glycaemic, cardiovascular and renal risk management is likely to provide the greatest long-term benefit.

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