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The primary aim of *World Journal of Diabetes* (*WJD*, *World J Diabetes*) is to provide scholars and readers from various fields of diabetes with a platform to publish high-quality basic and clinical research articles and communicate their research findings online.

WJD mainly publishes articles reporting research results and findings obtained in the field of diabetes and covering a wide range of topics including risk factors for diabetes, diabetes complications, experimental diabetes mellitus, type 1 diabetes mellitus, type 2 diabetes mellitus, gestational diabetes, diabetic angiopathies, diabetic cardiomyopathies, diabetic coma, diabetic ketoacidosis, diabetic nephropathies, diabetic neuropathies, Donohue syndrome, fetal macrosomia, and prediabetic state.

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EDITORIAL

Diabetes remission and nonalcoholic fatty pancreas disease

Wen-Jun Wu

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Abstract

This editorial focuses on the relationship between nonalcoholic fatty pancreas disease (NAFPD) and the development and remission of type 2 diabetes (T2D). NAFPD is characterized by intrapancreatic fatty deposition associated with obesity and not associated with alcohol abuse, viral infections, and other factors. Ectopic fat deposition in the pancreas is associated with the development of T2D, and the underlying mechanism is lipotoxic β-cell dysfunction. However, the results on the relationship between intrapancreatic fat deposition (IPFD) and β-cell function are conflicting. Regardless of the therapeutic approach, weight loss improves IPFD, glycemia, and β-cell function. Pancreatic imaging is valuable for clinically monitoring and evaluating the management of T2D.

Key Words: Diabetes remission; Type 2 diabetes; Pancreatic fat content; β cell function; Weight loss

Core Tip: Excess fat in the pancreas impairs β-cell function. The remission of type 2 diabetes and the improvement of β-cell function are achieved by decreasing intrapancreatic fat deposition during weight loss.

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INTRODUCTION

The remission of type 2 diabetes (T2D) is achievable despite the need for drug therapy. In clinical practice, T2D remission is defined as glucose levels lower than the thresholds established by the World Health Organization and the American Diabetes Association for 3 months without glucose-lowering pharmacotherapy[1]. Several interventions can effectively achieve T2D remission, including nutrition and weight management, pharmacotherapy, and bariatric surgery[2-4]. The mechanism shared by these therapeutic approaches is the improvement of β-cell function, especially first-phase insulin secretion (FPIS)[5]. β cells, located in the pancreatic islets of Langerhans, secrete insulin in response to an increase in postprandial glucose levels. Glucose-stimulated insulin secretion occurs in an early phase and a late phase, characterized by fast and slow secretory activity, respectively. FPIS dysfunction is an early marker of β-cell dysfunction during T2D progression. Studies on weight loss-induced T2D remission demonstrated that responders had shorter diabetes duration, better β-cell function, and less pancreatic fat at baseline than non-responders; nonetheless, there was no between-group difference in hepatic fat[6-8]. Hepatic insulin sensitivity improved regardless of the duration of diabetes, whereas β-cell dysfunction increased with the duration of diabetes[7]. Weight loss was positively associated with diabetes remission[9, 10]. Weight loss can decrease excess fat deposition in the liver and pancreas, reduce insulin resistance, and restore β-cell function; the reverse is also true[8,11]. This evidence supports Taylor's twin-cycle hypothesis and suggests that the capacity to regain β-cell function may be related to intrapancreatic fat deposition (IPFD) and duration of diabetes.

IPFD AND β-CELL FUNCTION

Animal studies *in vitro* and *in vivo* demonstrated that IPFD impaired β-cell function[12]. Moreover, there is evidence that lipotoxic β-cell dysfunction occurs in humans. A meta-analysis of cross-sectional studies showed that nonalcoholic fatty pancreas disease (NAFPD) was associated with a significantly greater risk of T2D[13]. Of four longitudinal studies, three showed that NAFPD increased T2D incidence[14-17]. Magnetic resonance imaging (MRI) showed that T2D was associated with decreased pancreatic volume, IPFD, and pancreas with irregular borders[18]. Moreover, pancreatic morphology changed as T2D progressed. Other studies found that a 6-month remission of T2D was related to less irregularity in pancreatic borders and no increase in pancreatic volume[19], and a 2-year remission of T2D was associated with regular borders and restoration of the pancreatic volume[20].

The correlation between IPFD and β-cell function is unclear. Some studies demonstrated that IPFD affects β-cell function in patients with glucose tolerance[21-23]. Other studies found no link between IPFD and β-cell function[24-26]. One explanation for the conflicting results is cohort differences in age, ethnicity, glucose metabolic status, and the duration of diabetes. After controlling for age, T2D duration, and the effects of drug therapy, pancreatic fat quantified by MR Dixon imaging revealed that IPFD correlated negatively with β-cell function[27]. Furthermore, a longitudinal study showed that IPFD was linked to lower insulin secretory capacity in patients with T2D[28]. Weight loss also decreased IPFD in T2D and was associated with increased insulin secretory capacity[29,30]. Thus, IPFD influences β-cell function.

The mechanism by which IPFD affects β-cell function is lipotoxicity. Evidence from cellular and animal models demonstrated that IPFD inhibited insulin secretion and caused the atrophy of β-cells[31]. The detailed molecular mechanisms include oxidative and endoplasmic reticulum stress, mitochondrial dysfunction, hypertriglyceridemia, and an increase in proinflammatory factors.

CLINICAL IMPLICATIONS

Early diagnosis and treatment to preserve and restore β-cell function are clinically important for T2D remission. Pancreatic morphology and IPFD are closely related to β-cell function. Thus, imaging-based fat quantification is recommended during routine clinical examination for two reasons. First, T2D is often accompanied by decreased pancreatic volume, IPFD, and pancreas with irregular borders. Second, weight loss and the consequent intrapancreatic fat loss are essential for T2D remission.

The fat content of the pancreas can be determined by ultrasound (US), computed tomography (CT) and MRI[32]. Despite the low cost, convenience, and wide applicability, US has low sensitivity for pancreatic fat quantification in individuals with obesity. CT is precise, and measurements are reproducible; however, ionizing radiation limits the use of CT for follow-up monitoring. MRI is safe and non-radioactive and accurately quantifies fat deposition. MRI is ideal for pancreatic fat quantification despite the high cost and long processing time.

CONCLUSION

The epidemic of T2D and associated complications are a global health priority. T2D remission is crucial to improve the health of individuals with T2D and reduce economic and health burden. β-cell function is a key factor for the onset development and remission of T2D. Evidence suggests that IPFD impairs β-cell function and affects T2D onset more strongly than intrahepatic fat deposition. The quantification of pancreatic fat is useful for the early screening, remission prediction, and personalized management of T2D. In future, it is very essential to explore potential therapeutic approaches that target pancreatic fat reduction.

FOOTNOTES

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