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From Control to Conquest: Clinical Pathways and Strategies for the Remission of Non-Insulin Dependent Diabetes Mellitus

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Abstract
Based on several studies, it was observed that patients who are treated for non-insulin dependent diabetes mellitus (NIDDM) also called type 2 diabetes (T2D) through metabolic surgery, intensive therapeutic interventions, and significant lifestyle modifications specifically low energy diet plus supervised exercise showed a durable normoglycemic state. The study also indicated that it happened through the restoration of functional β-cell mass to its normal level. Therefore, the expert consensus was introduced for expressing the concept of remission. Diabetes remission was defined as an HbA1c below 7.5 % (48 mmol/mol) without glucose- lowering medication for at least 12 weeks. Throughout the present review article, recently updated consensus statement and perspective on the remission was shared. There is a need for more researches to provide a critical clinical perspective on achieving and maintaining remission world-wide and in India. The remission of diabetes mellitus of type 2 appears to be driven by proactive treatment of hyperglycemia through early and aggressive glycemic control along with marked weight loss must be achieved, before irreversible β-cell failure occurs. Rather than viewing as an inevitably progressive chronic syndrome, the contemporary management should reframe it as an acute state and so the diabetes mellitus of late requires rapid therapeutic interventions. In fact we must be prompt to grasp this paradigm shift in our understanding of present disorder as well as for the benefit of our patients as endocrine experts.

Keywords: *Hyperglycemia, β-cell mass, Recovery of function, Metabolic surgery, Life style, Therapeutic.*

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I. INTRODUCTION

Diabetes is traditionally categorized into two forms, Insulin dependent diabetes mellitus (IDDM) or type 1 diabetes and Non-insulin dependent diabetes mellitus (NIDDM) or type 2 diabetes. Unlike type 1 diabetes, where autoimmune destruction of pancreatic cells causes insulin deficiency, in type 2 diabetes, insulin is generally present but the ability of the cells to respond to insulin and take glucose from the blood is impaired. Abnormal insulin response to glucose and insulin resistance of target peripheral tissues is the major features observed in type 2 diabetes.

This situation comes about because of the wear and tear and abuse of the insulin / sugar regulating process [1]. Predominantly manifesting after middle age, type 2 diabetes onsets is closely linked to weight gain, diet, and lifestyle [2]. Most patients experience a continuous decline in β -cell function [3] requiring intensified pharmacotherapy over time [4] to mitigate the development of irreversible end-organ complications [5].

A hallmark of NIDDM disease progression is the continuous decay of functional β-cell mass, frequently requiring treatment intensification over time to prevent irreversible macrovascular and microvascular complications. Sustained normoglycemia without ongoing medical intervention is attainable in select patients with type 2 diabetes mellitus, particularly following metabolic surgery, intensive medical regimens, and substantial lifestyle modifications [6-11]. These approaches have been shown to recover functional β-cell mass [12]. The leading expert panels to formally define this outcome as *remission* in 2009 [13]. Here, we review the guidelines on NIDDM remission and discuss key clinical perspectives [14].

II. DEVELOPMENT OF DIABETES MELLITUS

Glucose is the exclusive fuel of the human nervous system and primary fuel for most of the body systems. The brain utilizes approximately 20% of all the glucose metabolized by the entire body at rest mainly via the two major alternative pathways of carbohydrate metabolism that is, glycolysis coupled to the

tricarboxylic acid cycle and the pentose phosphate pathway. A healthy level of glucose in the blood is, therefore essential to maintain and sustain life and health. Hypoglycemia or reactive hypoglycemia is a state of low blood sugar that results from a reaction to rapid absorption of sugar. Sugar in and of itself is not a bad thing; it occurs naturally in many food stuffs. The body evolved over many generations to process the sugar consumed from natural sources. Sugar from natural sources is absorbed slowly and is metabolized as it is being released. Sugar from refined sources is concentrated, causing higher quantities to be consumed in a short time and without the associated fiber and other nutrients to slow down the absorption and release process. When excess sugar goes into the blood, the pancreas reacts instantly by releasing a large quantity of insulin into the blood. Since this is an "*emergency*" release, the insulin is generally in excess of what is required to maintain a steady state level and too much sugar is removed from the blood. When the blood sugar swings below the threshold level, a different panic response is triggered because a low level poses a serious crisis in the body. This response called hypoglycemia causes the adrenal glands to release glucocorticoid hormones which facilitate conversions of glycogen in the liver and muscle tissue into glucose. This results in the glucose level to rise again and, since this was a panic response, the glucose level will likely exceed its base level triggering yet another insulin release. The swing from high glucose levels to low glucose levels and back again may continue as a dampened oscillation for several hours. This is exactly what is observed in a glucose tolerance test (GTT). The patient fasts for 12 hours, has a blood sample taken to determine his base line, and then swallows defined amount of glucose. The blood sugar level is taken at half an hour to one hour intervals for the next several hours. In an individual demonstrating the reactive hypoglycemia response, the glucose level will rise sharply within half an hour. This rise is followed by a sharp drop and in the one to two hour interval; the blood sugar will be sharply lower. At this point, women become irrational and hysterical, men become aggressive and abusive and sweet old ladies curse like sailors. When the blood sugar level rises, the mood and behavior return to normal. The same response occurs when large amounts of refined sugar are consumed, although it is usually not as extreme. The amount of sugar consumed may not be as large as the 100 gram hit of glucose, and other food which releases glucose slower, is usually consumed with the sugar. When the insulin level is high, a lot of glucose is removed from the blood quickly, more quickly than it can be metabolized. This glucose is converted into fat in the cells. In addition, the individual craves for food, especially sweets, when the blood sugar is low, causing excess food consumption. This pattern of blood sugar swings contributes to the development of obesity and cumulative stress on the pancreas, adrenal glands and liver. The prolonged stress of compensating for the blood sugar swings contributes to the development of diabetes [15].

III. CLINICAL PATHWAYS FOR NIDDM REMISSION

Defining NIDDM remission is difficult. Unlike diseases that sometimes resolve completely and are classified as disease status versus healthy, diabetes is defined by hyperglycemia based on constantly changing blood glucose levels in the body that may be affected in the short term by temporary events such as drug effects, pregnancy, and acute illnesses [16-17]. To standardize terminology for sustained, drug-free normoglycemia [18-20] the updated framework replaced the original multi-tiered categories (partial, complete, prolonged) with a single diagnostic threshold < 6.5 % sustained for at least 3 months after discontinuing antidiabetic therapy [21]. When glycosylated hemoglobin (HbA1c) fails to accurately reflect glycemic status fasting plasma glucose or 2-hour postprandial plasma glucose during an oral glucose tolerance test (OGTT) may serve as diagnostic alternatives, however OGTT is generally discouraged owing to test complexity and intra-individual variability. With the expanding clinical adoption of continuous glucose monitoring (CGM), metrics such as the estimated HbA1c level (eA1c) or glucose management indicator (GMI) are now recommended. Crucially, because existing criteria are expert-driven and do not signify permanent disease resolution, the durability and long-term clinical outcomes of remission remain vital subjects of ongoing investigation [22].

3.1 Evidence Based Strategies for Achieving NIDDM Remission

Spontaneous or conventional therapy-induced remission occurs at an exceptionally low rate during the natural history of type 2 diabetes mellitus. In a large cohort study adults receiving standard medical care, only 1.5% achieved glycemic normalization permitting treatment cessation, with a mere 0.007% maintaining prolonged normalization over a 5 year period. Conversely, contemporary therapeutic approaches leveraging intensive glycemic control and substantial weight reduction consistently yield remission rates exceeding 50%. This target is further supported by the advent of novel pharmacotherapeutic agents capable of inducing significant weight loss and are expected to help patients achieve remission of type 2 diabetes mellitus [23- 24].

3.2 Bariatric and Metabolic Surgical Interventions in Severe Obesity

Surgical procedures promote satiety and limit nutrient absorption by structurally restricting the gastrointestinal tract. Beyond driving weight reduction, these procedures positively influence glycemic

regulation, partly through structural alterations that enhance incretin hormone secretion [25-26]. Options for metabolic surgery include adjustable gastric banding, vertical sleeve gastrectomy, and Roux-en-Y gastric bypass (RYGB) with outcomes varying by technique [27-29]. The clinical justification for remission of type 2 diabetes mellitus was initially established through metabolic (bariatric) surgery [30] and subsequently corroborated by multiple randomized controlled [31-38]. A comprehensive meta-analysis incorporating 19 observational studies and 4,070 patients demonstrated an overall post-surgical NIDDM remission rate of 78% [39]. Further meta-analyses of clinical trials confirm the superiority of bariatric surgical interventions over non-surgical approaches, providing rapid and robust glycemic recovery [40]. Nonetheless, surgical alteration of gastrointestinal anatomy does not ensure permanent remission; secondary weight regain can lead to glycemic deterioration over time [41]. Furthermore, as an invasive modality requiring general anesthesia, metabolic surgery carries acute risks including perioperative mortality [42] alongside chronic complications such as micronutrient deficiencies and psychological disorders [43-45]. Consequently, current clinical consensus reserves metabolic surgery for select populations, specifically individuals with severe or morbid obesity (BMI ≥ 30-35 kg/m²) who remain refractory to standard pharmacological and lifestyle management [46-47]. In these patients, metabolic surgery serves as a reliable mechanism to achieve the substantial weight loss required for visceral adiposity reduction and subsequent NIDDM remission [48].

3.3 Early Glycemic Correction via Comprehensive Insulin Therapy in Newly Diagnosed Patients

Remission has been documented in newly diagnosed patients presenting with uncontrolled hyperglycemia following a two to three-week course of intensive insulin therapy [49]. Subsequent randomized clinical trials compared conventional regimens with various insulin-based strategies to assess remission endpoints [50-54]. Evidence indicates that intensive insulin therapy can elicit remissions lasting over two years, with a shorter diagnostic-to-intervention interval correlating with an increased likelihood of sustained remission. Furthermore, early intensive intervention preserves β-cell function and structural integrity [55-56] while early combination therapies confer long-term glycemic durability [57]. Because chronic hyperglycemia exerts detrimental effects on β-cell secretory capacity and peripheral insulin sensitivity [59] and prolonged glycemic exposure escalates microvascular and macrovascular complication risks [60-62] proactive treatment intensification is vital to mitigate cumulative glucotoxicity. Given the multifaceted pathogenesis of type 2 diabetes mellitus, combining pharmacological agents with complementary mechanisms can generate synergistic benefits. Early combination approaches also circumvent delays in achieving optimal glycemic control that often arise from clinical inertia during sequential up-titration [63-64] highlighting the growing significance of combination therapies in restoring β-cell function [65]. Conversely, indiscriminate initial intensive management may lead to overtreatment in patients with borderline glycemic parameters who could otherwise maintain glycemic targets through lifestyle modifications or monotherapy alone. Furthermore, combination regimens incur higher costs [66] and elevate the risk of polypharmacy-related adverse events [67]. In elderly populations, a conservative therapeutic approach is often warranted given the inverse relationship between hypoglycemia risk and life expectancy. Consequently, a personalized strategy is essential, as a stepwise escalation originating from minimal intervention remains clinically effective for select patient subgroups [68-69].

3.4 The Role of Caloric Restriction-Induced Weight Loss as an Intensive Strategy for NIDDM Remission

Among lifestyle interventions caloric restriction emerges as a primary strategy for optimizing both weight management and glycemic control [71-73]. Very-low calorie diets (VLCDs) defined as caloric intakes of 800 kcal or less per day providing essential micronutrients and a high protein-to-calorie ratio, typically administered as meal replacements for 3 to 4 months [74] have gained recognition as a therapeutic intervention for obesity-related type 2 diabetes [75]. Recent clinical evaluations demonstrate significant potential for diabetes remission using VLCD protocols [76-79]. For instance a recent study demonstrated that integrating VLCD with routine primary care achieved type 2 diabetes remission in 46% of participants, highlighting a positive correlation between the magnitude of weight loss and remission success [78]. A direct positive correlation exists between the magnitude of weight loss and the diabetes remission rate. Although VLCDs are generally considered safe, clinical implementation mandates strict medical supervision [80]. Early-stage adverse effects frequently include transient fatigue and gastrointestinal disturbances [81], while rare instances of fatal arrhythmias have been documented [82]. Consequently, VLCD protocols are contraindicated for individuals of normal weight [83]. Furthermore, preceding investigations indicate that preoperative VLCD administration in patients with severe obesity scheduled for metabolic surgery effectively mitigates perioperative complications and optimizes surgical efficacy [84].

3.5 Low Energy Diet Plus Supervised Exercise Leads Remission of Early- Onset NIDDM

In a recent study published in *The Lancet* a low energy diet paired with exercise successfully reversed early-onset NIDDM in 54% of young adult participant after 24 weeks without medication compared to just a

4% remission rate in the standard care group. Current study also finds that the intervention was associated with substantial fat loss while preserving fat-free mass and muscle volume. In-addition the participants also demonstrated greater hand grip strength and improved cardio-respiratory fitness relative to body mass. The study found that the intervention improved remission while promoting fat loss, preserving muscle mass, and enhancing strength, fitness and patient reported outcomes. No serious adverse events were reported during study. The present study had several limitations including the absence of a low energy diet only group, making it difficult to determine the independent effect of supervised exercise. Overall, combining a low energy diet with structured aerobic and resistance exercise achieved substantial remission in early-onset NIDDM while improving body composition and fitness. Longer term studies are needed to access sustained remission and the added benefit of supervised exercise [85].

3.6 Novel Drug Development: From Molecular Optimization to Clinical Efficacy and Practice

Recent advancements in pharmacotherapy have introduced novel medications with unique dual metabolic benefits the most notably significant weight reduction along with glycemic control. Sodium-glucose cotransporter 2 (SGLT2) inhibitors exemplify this class operating independently of insulin secretion or sensitivity by blocking proximal tubular glucose resorption. This induction of glycosuria results in substantial caloric and visceral fat loss establishing SGLT2 inhibition as a cornerstone in intensive combination regimens for metabolic disease management resulting in a remission rate of 66% to 81% depending on the drug dose over 52 weeks of use in a clinical study [86]. Furthermore, these agents produce a profound weight loss effect while maintaining a low risk of hypoglycemia [87]. Consequently, they offer a favorable alternative to more aggressive interventions associated with substantial medical risk such as bariatric surgery, early intensive insulin therapy and very-low-calorie diets. However, as these are relatively recent therapeutic modalities, long-term evidence specifically targeting diabetes remission remains limited highlighting the need for further investigation. In parallel, recently developed novel antidiabetic agents feature unique mechanisms centered on significant weight reduction. For instance SGLT2 inhibitors act on the proximal convoluted kidney tubule to decrease glucose resorption, thereby, inducing glycosuria. This provides a glucose-lowering effect entirely independent of insulin secretion or sensitivity [88]. Beyond glycemic control this class facilitates reductions in total body weight and visceral fat due to the chronic caloric loss associated with urinary glucose excretion [89-92]. Supporting this, a clinical trial evaluating intensive intervention with SGLT2 inhibitors added to basal insulin and metformin demonstrated a higher remission rate compared to conventional therapy (24.7% vs. 16.9%) along with a 43% reduction in the risk of diabetes recurrence. Despite their metabolic advantages the clinical utility of these novel agents is constrained by specific contraindications and adverse effect profiles. SGLT2 inhibitors are largely precluded in patients with advanced chronic kidney disease and carry an elevated risk of normoglycemic ketoacidosis compared to conventional therapies [93]. Meanwhile the injectable nature of glucagon-like peptide1 (GLP1) receptor agonists and tirzepatide can introduce psychological barriers, local injection-site reactions, and frequent gastrointestinal side effects that frequently lead to treatment discontinuation [94]. Furthermore high acquisition costs present a substantial barrier to accessibility. Consequently a precision medicine framework focused on careful patient selection is essential to maximize therapeutic benefits and mitigate these risks.

IV. PATHOPHYSIOLOGY OF NIDDM REMISSION: BETA-CELL RECOVERY AND INSULIN SENSITIVITY RESTORATION

4.1 Cellular Plasticity: Dedifferentiation Pathways in Insulin Secreting Beta-Cell Dysfunction

Susceptibility to diabetes mellitus is shaped by a combination of genetic predispositions [95] prenatal nutritional status [96] and early-life environmental exposures [97]. Initially pancreatic β-cells undergo compensatory proliferation and hypersecretion to meet the body's escalating insulin demands driven by weight gain [98]. However β-cell failure ultimately manifests as a progressive functional decline wherein therapeutic demands steadily escalate due to chronic hyperglycemia and weight-related metabolic overload [99]. Previously the worsening course of diabetes was attributed to permanent β-cell apoptosis and cell death. Nevertheless, with the introduction and substantiation of the cellular dedifferentiation paradigm, a new foundation has been established for therapeutic strategies aimed at β-cell revitalization and functional restoration [100-101].

4.2 The Gastro-Intestinal Backtrack: Metabolic Insights and Surgical Perspectives

Diabetes mellitus is conceptualized as *an operable intestinal disease*, drawing focus to the rapid normalization of glucose homeostasis observed following bariatric surgery [102]. Because glycemic improvements occur too quickly to be explained by weight loss alone [103], structural alterations within the gastro-intestinal tract have been proposed as a primary mechanism. Changes resulting from nutrient diversion around the proximal small intestine are designated as the "*foregut hypothesis"* whereas those triggered by the accelerated delivery of nutrients to the distal small intestine constitute the "*hindgut hypothesis"* [104]. Intestinal

hormones specifically incretins play a central role in these adaptive responses linking diabetes to gut hormone regulation via the enteroinsular axis [105]. Investigations into food-stimulated gut hormones and their influence on insulin secretion [106] have highlighted glucagon-like peptide1 (GLP 1) and glucose-dependent insulinotropic polypeptide (GIP) as critical mediators. Consequently, receptor agonists targeting these incretin pathways have been successfully developed as potent antidiabetic agents, underscoring the need for ongoing clinical research regarding their capacity to induce sustained remission.

4.3 Hepato-Pancreatic Feedback Loops: The Twin Cycle Hypothesis and Individual Adipose Thresholds

Achieving NIDDM remission fundamentally relies on significant weight loss accompanied by targeted reductions in visceral adiposity [107]. Consequently emerging research has increasingly emphasized the pathological contribution of non-alcoholic fatty liver disease (NAFLD) to systemic energy imbalance and metabolic disruption [108-109]. Because hepatic lipid accumulation stems from excess energy storage, it is among the earliest metabolic anomalies to improve following the onset of weight reduction [110]. Furthermore, the magnitude of adipose tissue reduction is region-specific. Clinical studies have demonstrated that an overall weight loss of 16% yields disproportionate improvements corresponding to a 30% decrease in intra-abdominal fat and a profound 65% clearance of intrahepatic triglycerides [111-112]. Twin cycle hypothesis proposed that pathogenesis of type 2 diabetes mellitus is driven by a feedback loop of ectopic lipid deposition within the liver and pancreas [113]. Chronic caloric surplus induces hepatic steatosis and systemic hyperlipidemia when hepatic storage capacity is exceeded that concurrently impair insulin sensitivity and secretion. Subsequent pancreatic lipid accumulation further compromises β-cell functionality [114]. To account for the onset of diabetes mellitus in non-obese individuals, the personal fat threshold model assumes that susceptibility is dictated by genetically predetermined limits in subcutaneous fat storage capacity [115]. Consequently, targeted weight reduction remains a clinically efficacious strategy for diabetes prevention even among normoweight or mildly overweight prediabetic groups [116].

V. ROLE OF TIME AND BASELINE PREDICTORS OF NIDDM REMISSION

Because inducing diabetes remission triggers substantial physiological shifts, careful patient selection and optimal timing of interventions remain critical considerations. Following metabolic surgery several predictive scoring models have been established to estimate preoperative remission outcomes. These instruments primarily rely on baseline parameters such as residual β-cell function (assessed via C-peptide levels), glycemic control (HbA1c) diabetes severity (insulin dependence and the number of potential antidiabetic agents) and disease duration [117-122]. To enhance prognostic accuracy expanded models incorporating demographic characteristics, surgical modality and comorbidities have been proposed along with recent efforts to streamline predictions using novel biochemical biomarkers. [123-124]. Evidence regarding patient age and remission outcomes presents a dichotomy while surgical and dietary weight-loss interventions yield higher remission frequencies in younger groups [125]. Conventional medical therapies report higher relative remission prevalence among older adults [126]. Nonetheless, extended disease duration and pharmacological complexity particularly baseline insulin dependence consistently predicts poor remission outcomes due to irreversible β-cell loss [127-129]. Ultimately, prognostic success is dictated less by baseline demographics (age, sex, BMI) and more by clinical indices of disease severity and glycemic control. Emphasizing early therapeutic intervention in newly diagnosed patients remains vital to halting disease progression and improving long-term prognosis.

VI. DOES NIDDM REMISSION MAINTAINED? THE STORY AFTER REMISSION

Individuals who have previously been diagnosed with diabetes are more susceptible to diabetes than other individuals and have a higher risk of recurrence. Researchers believe that weight loss and maintenance are key to maintaining remission of non-insulin dependent diabetes mellitus [130-131]. Maintaining these behavioral changes over the long term requires both individual commitment and coordinated psychosocial support systems [132]. In addition, hyperglycemia appears to continue to affect the body even after it has improved, as the long-term consequences for hyperglycemic conditions, are referred to as the "*legacy effect*" or "*metabolic memory*" [133]. This effect is mainly associated with microvascular complications rather than effects on macrovascular complications or survival [134]. Therefore it is thought that individuals liberated from hyperglycemia will require continuous clinical monitoring, managing recurrence risks and complications in patients even after remission is confirmed

VII. WHAT NEEDS REMAIN UNMET?

7.1 Evaluation of Remission Criteria

Current remission definitions rely primarily on expert consensus derived from standard diabetes diagnostic criteria indicating a need to evaluate novel benchmarks such as maintenance duration or stricter glycemic

thresholds. Lowering diagnostic cutoffs may also prove necessary to mitigate relapse risk. Although the consensus acknowledges continuous glucose monitoring (CGM) utility, it currently restricts recommendations to estimated HbA1c (eA1c) values, highlighting the potential for novel metrics like time in range (TIR) to be incorporated into future remission evaluations.

7.2 Monitoring Following Diabetes Remission

Current consensus guidelines recommend annual assessments of glycemic status and diabetes related complications for individuals with a history of post-remission. This approach is predicated on the phenomenon of "*metabolic memory*," wherein prior hyperglycemia exerts lingering pathophysiological effects despite subsequent improvements in glycemic control [135-136]. Given these long-term medical outcomes following remission the optimal frequency of monitoring intervals and the specific diagnostic procedures required for complication, screening remain important areas for future investigation and debate.

7.3 Interventions through Public Health Policy

As obesity rates continue to parallel the escalating incidence of type 2 diabetes mellitus obesity has transcended individual clinical management to become a major public health and socioeconomic challenge [137-138]. Mitigating the long term clinical and economic impact of diabetes requires shifting the current disease management paradigm toward broad societal interventions, including population-wide health initiatives and beverage taxation policies [139-140].

7.4 Perspectives on Asian Ethnicity

Targeted studies assessing treatment-specific remission outcomes in Asian populations *specifically in India* still remain a critical gap in contemporary diabetes research [141]. Adding agony to the scenario is the fact the Asians in general and Indians in particular are genetically more predisposed to diabetes mellitus of type 2. Severity of the glucose abnormalities trigger a vast group of diabetic complications.The genetic predisposition combined with adoption of western life style, which makes Indians obese, inactive and stressed-out, is playing havoc not only with elderly but even with the younger generations. The information technology revolution, urban migration and consumerism have made Indians in cities and even in small towns adopt the Coca-Cola culture where fast food is preferred to desi cuisines. So while improvement in nutrition and hygiene has led to a decrease in communicable diseases there has been a sharp rise in non-communicable ailments like diabetes [142-148].

VIII. CONCLUSIONS

Current evidences suggest that diabetes remission is primarily mediated by intensive glycemic control and significant lifestyle modification prior to the onset of irreversible β-cell deterioration. By implementing short-term intensive insulin therapy in patients with uncontrolled hyperglycemia at an early stage of the disease β-cell function improves and remission of NIDDM can be secured for a considerable period. Significant weight loss induced through metabolic surgery or very low calorie diets protocol successfully restores normoglycemia in more than fifty percent of overweight, newly diagnosed patients. Overall, combining a low- energy diet with structured aerobic and resistance exercise achieved substantial remission in early-onset NIDDM while improving body composition and fitness. Emerging therapeutic classes (new drugs) such as sodium-glucose cotransporter 2 (SGLT2) inhibitors and glucagon-like peptide1 receptor agonists (GLP1-RAs) have a favorable weight loss effect so they can help to achieve this remission goal in a safer way. Intensive glycemic control not only increases the likelihood of remission at the earlier stage of the disease but also curtails microvascular and macrovascular late complications and risk profiles of NIDDM even if remission is not achieved. Nonetheless, from the perspective of long-term survival, it is essential to pay attention to the risk of hypoglycemia in elderly patients and hence an additional research is needed on the long-term medical outcomes of diabetes remission highlighting critical gaps for future prognostic studies. Patients who have previously been diagnosed with diabetes are exposed to the risks of diabetes related complications, even in patients who have improved and confirmed remission. Support and continuous medical supervision from medical staff, family members and friends are essential so that they can maintain a healthy lifestyle and screen for comorbidities associated with diabetes. NIDDM is not a fate for individuals to helplessly accept. The present review article discussed remission of NIDDM that can be achieved through a personalized approach and tailored remission strategies. It may be essential to change the concept of diabetes mellitus towards that of an urgent condition that requires rapid intervention rather than a persistent pervasive disease. We must understand this paradigm shift in our understanding of NIDDM for the benefit of our patients as endocrine experts.

Conflicts of interest statement

The author declares that the present review article was written in the absence of any commercial or financial relationship with anybody in any form. No potential conflict of interest relevant to this article was reported.

Ethical considerations

The review article is original and it has not been published elsewhere. No AI tools was used during the study and writing the manuscript.

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