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Overview on type 5 diabetes mellitus: Causes, symptoms and dietary management

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Abstract
Type 5 Diabetes Mellitus is an emerging subtype of diabetes, classified under "secondary diabetes," primarily associated with pancreatic diseases such as pancreatitis, pancreatic cancer, and cystic fibrosis. Unlike the classical types Type 1 and Type 2, Type 5 diabetes arises from direct damage to the exocrine and endocrine functions of the pancreas, affecting insulin production and glucose metabolism. This review presents an in-depth overview of the pathophysiology, etiological factors, clinical manifestations, and dietary interventions for managing Type 5 Diabetes Mellitus. Emphasis is placed on the unique challenges this subtype poses and the importance of individualized nutrition plans in conjunction with medical treatment.

Keywords: Type 5 Diabetes mellitus, human health, foods, glucose metabolism, dietary interventions

1. Introduction
Diabetes mellitus (DM) is a group of metabolic disorders characterized by chronic hyperglycemia due to defects in insulin secretion, insulin action, or both (Garai *et al*., 2023) [31]. Traditionally classified into type 1 and type 2, the spectrum of diabetes has broadened significantly in recent years, with increasing recognition of less common but clinically significant forms. One such emerging category is Type 5 Diabetes Mellitus, a subset of secondary diabetes induced by specific pharmacological or medical interventions, particularly certain medications such as glucocorticoids, immunosuppressants, and antipsychotics (American Diabetes Association [ADA], 2024) [4].
Type 5 diabetes mellitus, although not universally adopted in global classification systems like the World Health Organization (WHO), is gaining traction in endocrinological and clinical research communities (Jana *et al*., 2019) [44]. It encompasses diabetes caused by external influences that interfere with glucose metabolism through mechanisms distinct from autoimmunity or lifestyle-induced insulin resistance. These influences include drug-induced insulin resistance, pancreatic damage due to infections or surgery, hormonal disturbances, and endocrine disorders (Gupta *et al*., 2023) [34]. Type 5 diabetes is thus not a standalone disease entity but a classification to distinguish diabetes arising secondary to identifiable, often preventable or modifiable, causes (Gang and Ma, 2008) [30].
This form of diabetes is crucial to identify due to its unique pathophysiology, management strategies, and prognosis. Unlike primary forms of diabetes, where lifestyle interventions or insulin therapy are the mainstay, management of Type 5 diabetes necessitates a thorough understanding of the underlying cause. For instance, drug-induced hyperglycemia often resolves or improves upon dose adjustment or discontinuation of the causative agent, provided there is no irreversible damage to pancreatic β-cells or insulin receptors (Chakraborty & Singh, 2022) [8].

1.1 Etiology and Causative Agents
The primary hallmark of Type 5 diabetes is its association with identifiable external factors. One of the most common causes is glucocorticoid-induced diabetes, which arises from the use of corticosteroids in treating conditions such as asthma, rheumatoid arthritis, and organ transplantation.

Glucocorticoids exert anti-inflammatory effects but simultaneously promote insulin resistance in liver and muscle tissues, leading to hyperglycemia (van Raalte *et al*., 2009) [60]. Prolonged use can impair β-cell function, making glycemic control progressively difficult.

Another significant contributor is the use of immunosuppressive drugs, such as tacrolimus and cyclosporine, commonly administered post-transplantation. These agents interfere with insulin secretion at the pancreatic level and also promote insulin resistance, significantly elevating the risk of new-onset diabetes after transplantation (NODAT), (Hjelmesaeth *et al*., 2004) [39]. Similarly, atypical antipsychotic medications like olanzapine and clozapine are known to induce weight gain and impair glucose metabolism, precipitating diabetes even in individuals without previous risk factors (Newcomer, 2005) [57].

Other endocrine disorders, such as Cushing's syndrome, acromegaly, and hyperthyroidism, also fall under the broad umbrella of Type 5 diabetes due to their potential to disrupt glucose homeostasis through hormonal dysregulation. Furthermore, conditions such as chronic pancreatitis, pancreatectomy, and hemochromatosis can lead to destruction of pancreatic tissue, thereby reducing insulin production (Holt *et al*., 2021) [40].

1.2 Symptoms and Clinical Presentation

The clinical manifestations of Type 5 diabetes often mirror those of classic diabetes mellitus, including polyuria (frequent urination), polydipsia (excessive thirst), polyphagia (increased hunger), and unexplained weight loss. However, because it frequently arises in patients already under medical treatment or suffering from a chronic disease, these symptoms may be subtle or misattributed to the underlying illness (Chatterjee *et al*., 2021) [9].

In cases such as glucocorticoid-induced diabetes, patients may exhibit postprandial hyperglycemia with relatively normal fasting blood glucose levels in the early stages. Over time, both fasting and postprandial levels may elevate, leading to classical diabetic symptoms and complications. In transplant recipients or cancer patients on immunosuppressants, the development of hyperglycemia may be sudden and severe, necessitating prompt intervention to prevent complications such as diabetic ketoacidosis or hyperosmolar hyperglycemic state (Dungan *et al*., 2009) [23].

Importantly, the risk of microvascular (retinopathy, nephropathy, neuropathy) and macrovascular complications (cardiovascular disease, stroke) remains comparable to other forms of diabetes, especially if glycemic control is not achieved swiftly. Therefore, early identification and targeted treatment of the underlying cause are crucial in minimizing long-term sequelae.

1.3 Dietary Management and Nutritional Strategies:

Nutritional management plays a pivotal role in the overall treatment approach to Type 5 diabetes, although the dietary strategy may differ slightly based on the underlying cause and concurrent medical therapy. The primary objectives are to maintain normoglycemia, prevent complications, support the patient's overall health, and improve treatment outcomes for the underlying disease.

The American Diabetes Association (ADA) recommends individualized dietary planning, with emphasis on complex carbohydrates, high dietary fibre, lean proteins, and low glycemic index (GI) foods. Reducing the intake of refined sugars and saturated fats is essential for preventing spikes in blood glucose levels and supporting cardiovascular health (ADA, 2024) [4].

For patients on glucocorticoid therapy, insulin resistance predominantly affects postprandial glucose levels. Therefore, distributing carbohydrate intake evenly across meals and including protein-rich snacks can help maintain stable glycemic levels. Foods rich in magnesium, such as whole grains and leafy greens, may also improve insulin sensitivity (Hruby & Meigs, 2015) [41].

In cases related to immunosuppressant or antipsychotic therapy, monitoring weight gain is crucial. These drugs may increase appetite and fat deposition, especially around the abdomen, leading to metabolic syndrome. A calorie-controlled diet with regular physical activity is advised to mitigate these effects. In such patients, plant-based diets, low in processed foods and high in phytonutrients, have shown promise in improving insulin sensitivity (Toumpanakis *et al*., 2018) [69].

Moreover, certain nutraceuticals, such as berberine, cinnamon extract, and fenugreek, have been studied for their insulin-sensitizing properties and may serve as adjuncts under professional supervision (Gulati *et al*., 2019) [33]. However, caution is necessary to avoid interactions with medications.

Hydration, portion control, and routine monitoring of blood glucose levels should be integral to dietary management. Registered dietitians or certified diabetes educators should be involved in creating tailored nutritional plans, especially for patients with multiple comorbidities.

2. Defining type 5 diabetes mellitus

Type 5 Diabetes Mellitus is categorized under secondary diabetes, which refers to diabetes resulting from an underlying medical condition or external factor that disrupts normal insulin function. Unlike Type 1 and Type 2 diabetes, which arise primarily from autoimmune destruction of beta cells or insulin resistance respectively, Type 5 diabetes is a consequence of pancreatic dysfunction due to various causes such as chronic pancreatitis, pancreatic cancer, cystic fibrosis, hemochromatosis, surgical removal of the pancreas, or certain medications.

2.1 Classification

Table 1: Classification of type 5 diabetes mellitus based on underlying Etiology

Category	Specific Cause	Mechanism of Diabetes Development	Reference
Pancreatic Disorders	Chronic Pancreatitis	Destruction of islet cells and impaired insulin secretion	Ewald & Bretzel, 2013 [26]
	Pancreatic Cancer	Infiltration of pancreatic tissue affecting endocrine function	Hart *et al.*, 2016 [37]
	Cystic Fibrosis	Fibrosis and cyst formation impair both exocrine and endocrine roles	Moran *et al*., 2009 [55]
	Hemochromatosis	Iron overload leads to oxidative damage of beta cells	Niederau *et al*., 1996 [58]
	Pancreatectomy	Loss of insulin-producing tissue	Rickels *et al*., 2013 [64]
Systemic Endocrine Disorders	Cushing's Syndrome	Excess cortisol induces insulin resistance	Anagnostis *et al.*, 2009 [5]
	Acromegaly	Growth hormone excess reduces insulin sensitivity	Colao *et al*., 2004 [14]
	Hyperthyroidism	Increases hepatic glucose output and reduces insulin action	Dimitriadis *et al*., 2000 [22]
Drug-Induced Diabetes	Glucocorticoids	Increase gluconeogenesis and insulin resistance	Clore & Thurby-Hay, 2009 [13]
	Thiazide Diuretics	Hypokalemia impairs insulin secretion	Zillich *et al*., 2006 [74]
	Antipsychotics	Weight gain and insulin resistance	Newcomer, 2005 [57]
	Immunosuppressants (e.g., Tacrolimus)	Beta-cell toxicity and reduced insulin gene expression	Hecking *et al*., 2004 [38]

2.2 Pathophysiology

The pathophysiology of Type 5 Diabetes Mellitus is primarily rooted in structural and functional damage to the pancreas, which disrupts its dual roles as an endocrine and exocrine gland. Unlike Type 1 or Type 2 diabetes, where autoimmune destruction or insulin resistance predominates (Koufakis *et al*., 2021) [48], Type 5 diabetes emerges as a secondary condition resulting from external or internal insults that compromise pancreatic tissue. This damage can be caused by chronic inflammation (e.g., pancreatitis), surgical resection, tumors, cystic fibrosis, or iron overload conditions like hemochromatosis (Hsu *et al*., 2022) [42].

The endocrine component of the pancreas consists of the islets of Langerhans, which contain various hormone-secreting cells most notably the beta cells (which produce insulin) and alpha cells (which produce glucagon) (Mann *et al*., 2016) [54]. In Type 5 diabetes, injury to pancreatic tissue leads to significant beta-cell dysfunction or destruction, resulting in decreased insulin secretion (Fu *et al*., 2013) [28]. Insulin is essential for the regulation of glucose uptake by cells, and its deficiency leads to persistent hyperglycemia, the hallmark of all forms of diabetes mellitus (Rahman *et al*., 2021) [61].

In addition to insulin, the production of glucagon by alpha cells may also be impaired (Dunning *et al*., 2005). Glucagon plays a vital role in counteracting hypoglycemia by promoting gluconeogenesis and glycogenolysis in the liver (Ramnanan *et al*., 2011) [62]. When glucagon secretion is disrupted, the body's ability to correct falling blood glucose levels is compromised, increasing the risk of hypoglycemic episodes, especially in patients who are on exogenous insulin therapy or oral hypoglycemics (D'alessio, 2011) [16].

The exocrine function of the pancreas is also significantly affected in Type 5 diabetes. The pancreas produces digestive enzymes like amylase, lipase, and proteases that are essential for the digestion and absorption of nutrients in the small intestine. When exocrine function is impaired often termed pancreatic exocrine insufficiency (PEI) nutrient malabsorption occurs. This malabsorption is especially problematic for fat and fat-soluble vitamins (A, D, E, and K), leading to steatorrhea (fatty stools), weight loss, and deficiencies that can impact immunity, vision, bone health, and coagulation (Larger *et al*., 2012) [50].

This combination of endocrine and exocrine dysfunction creates a complex metabolic disorder that is often difficult to manage (Ahsan *et al*., 2021) [1]. The concurrent presence of hyperglycemia, unpredictable hypoglycemia, and malnutrition presents a unique clinical challenge. Moreover, due to the compromised digestive process, there is a risk of protein-energy malnutrition, anemia, and delayed wound healing (Srivastava *et al*., 2024) [67].

Additionally, pancreatic inflammation or fibrosis may affect neighboring structures such as the duodenum and bile ducts, exacerbating gastrointestinal symptoms and further hindering nutritional status. Chronic inflammation also contributes to a pro-inflammatory state and oxidative stress, which can worsen insulin resistance and accelerate the progression of diabetes-related complications (Wronka *et al*., 2022) [72].

3. Causes of type 5 diabetes mellitus

Type 5 diabetes can stem from various medical conditions and external factors:

3.1 Pancreatic Disorders

Pancreatic disorders are key contributors to the development of Type 5 Diabetes Mellitus due to their impact on insulin-producing beta cells and exocrine functions of the pancreas (Ciochina *et al*., 2022) [12]:

- Chronic Pancreatitis involves long-term inflammation that progressively damages pancreatic tissue, particularly the islets of Langerhans, impairing insulin secretion (Kleeff *et al*., 2017) [46].
- Pancreatic Cancer, especially in the head or body of the pancreas, may infiltrate and destroy endocrine cells, disrupting glucose metabolism (Zhao and Liu, 2020) [73].
- Cystic Fibrosis causes thick mucus accumulation, leading to scarring and cysts in the pancreas, which can damage both exocrine and endocrine components (Sheppard and Nicholson, 2002) [66].
- Hemochromatosis results in iron accumulation in the pancreas, leading to oxidative damage and beta-cell dysfunction (Backe *et al*., 2016) [6].
- Surgical Removal (Pancreatectomy) of the pancreas, whether partial or complete, significantly reduces or abolishes insulin production, causing secondary diabetes (Maeda and Hanazaki, 2011) [53].

3.2 Endocrinopathies and Systemic Diseases

Endocrinopathies and systemic diseases represent important non-pancreatic causes of Type 5 Diabetes Mellitus. These conditions disrupt normal endocrine function, which can

impair insulin secretion or reduce insulin sensitivity, leading to persistent hyperglycemia. The hormonal imbalances associated with such diseases often create a state of insulin resistance or β-cell dysfunction, thereby contributing to the pathogenesis of secondary diabetes. Among the most prominent endocrinopathies involved are Cushing's syndrome, acromegaly, and hyperthyroidism (Ciochina *et al*., 2022) [12].

3.3 Cushing's Syndrome

Cushing's syndrome is characterized by prolonged exposure to high levels of cortisol, either due to endogenous overproduction (e.g., adrenal tumors or pituitary adenomas) or exogenous corticosteroid use (Reincke and Fleseriu, 2023) [63]. Cortisol, a glucocorticoid hormone, plays a significant role in glucose metabolism by promoting gluconeogenesis in the liver and inhibiting glucose uptake in peripheral tissues (Munck, 1971) [56]. Excess cortisol leads to pronounced insulin resistance, primarily in muscle and adipose tissue. As a result, pancreatic β-cells are forced to compensate by increasing insulin secretion. Over time, this compensatory mechanism fails, and hyperglycemia develops, culminating in secondary diabetes (Del Prato *et al*., 2017) [20].

Furthermore, chronic hypercortisolemia can impair pancreatic β-cell function directly and increase visceral fat deposition, further exacerbating insulin resistance (Janssen, 2022) [45]. Clinically, patients with Cushing's syndrome often exhibit classic features of diabetes such as polyuria, polydipsia, and unexplained weight gain, along with specific signs like truncal obesity, purple striae, muscle wasting, and hypertension (Icecreamwala and Shwayder, 2019) [43].

3.4 Acromegaly

Acromegaly is caused by excessive secretion of growth hormone (GH), usually due to a pituitary adenoma. GH antagonizes the actions of insulin by inhibiting glucose uptake in muscles and adipose tissue and stimulating lipolysis and hepatic glucose production (Olarescu and Bollerslev, 2016) [59]. This creates a state of severe insulin resistance, necessitating increased insulin secretion from β-cells to maintain glucose homeostasis (LeRoith, 2002) [52].

In early stages, β-cell compensation may prevent overt hyperglycemia. However, persistent GH excess eventually leads to β-cell exhaustion, resulting in impaired glucose tolerance or overt diabetes (Wajchenberg, 2007) [70]. Studies show that nearly 40-50% of patients with acromegaly develop glucose intolerance or diabetes mellitus. Acromegalic patients with diabetes may not show classical diabetic symptoms initially, as features such as enlarged hands and feet, coarse facial features, and jaw protrusion often dominate the clinical picture (Hannon *et al*., 2017) [36].

Treating acromegaly through surgery, medications (e.g., somatostatin analogs), or radiation often results in improvement or resolution of diabetes, emphasizing the reversible nature of hormonal-induced insulin resistance (Feelders *et al*., 2009) [27].

3.5 Hyperthyroidism

Hyperthyroidism, particularly in conditions such as Graves' disease, leads to an increased basal metabolic rate and enhanced glucose turnover (Chng *et al*., 2016) [11]. Thyroid hormones stimulate hepatic gluconeogenesis and glycogenolysis, increase intestinal glucose absorption, and elevate the rate of insulin degradation. Collectively, these effects can cause mild to moderate insulin resistance and, in some cases, glucose intolerance or secondary diabetes **(**Bayati and Al-Khateeb, 2021**)** [3].

Additionally, thyroid hormone excess may increase β-cell workload and oxidative stress, contributing to β-cell dysfunction (Lenzen, 2008) [51]. Though hyperthyroidism-associated diabetes is generally less severe than that seen in Cushing's syndrome or acromegaly, it can aggravate preexisting glucose intolerance, especially in elderly or genetically predisposed individuals (Chauhan and Patel, 2024) [10].

Management of hyperthyroidism often leads to improved glycemic control. However, failure to recognize and treat thyroid dysfunction in diabetic patients may worsen glucose instability and complicate disease management (Biondi *et al*., 2019) [7].

3.6 Drug-Induced Diabetes

Drug-induced diabetes is a significant and often underrecognized contributor to secondary diabetes, including Type 5 Diabetes Mellitus. This condition arises when prolonged use of certain medications interferes with glucose metabolism, insulin secretion, or insulin sensitivity. Individuals who are on long-term pharmacological treatments for chronic conditions, particularly those involving immunosuppressive therapy, psychiatric disorders, or inflammatory diseases, are at elevated risk (Chakraborty & Singh, 2022; Holt *et al*., 2021) [8, 40]. While the underlying mechanisms vary by drug class, the resultant effect is either impaired insulin secretion or increased insulin resistance, leading to persistent hyperglycemia (Dungan *et al*., 2009) [23].

3.7 Glucocorticoids

Glucocorticoids, such as prednisone, dexamethasone, and hydrocortisone, are among the most common drugs associated with secondary diabetes. These medications are frequently prescribed for autoimmune diseases, inflammatory disorders, and post-transplant immunosuppression. They increase blood glucose levels by enhancing hepatic gluconeogenesis, inhibiting peripheral glucose uptake, and impairing beta-cell function (van Raalte *et al*., 2009) [60]. Chronic glucocorticoid therapy can lead to significant insulin resistance and beta-cell exhaustion, ultimately resulting in a diabetes-like state. This form of drug-induced diabetes is dose-dependent and may be reversible upon drug discontinuation, though prolonged use may cause permanent damage (Chatterjee *et al*., 2021) [9].

3.8 Thiazide Diuretics

Thiazide diuretics, including hydrochlorothiazide and chlorthalidone, are widely used antihypertensive agents. While effective in managing blood pressure, their prolonged use has been linked to impaired glucose tolerance and the development of diabetes (Gupta *et al*., 2023) [34]. Thiazides may reduce insulin secretion by causing hypokalemia (low potassium levels), which adversely affects pancreatic beta-cell function. They also contribute to insulin resistance through mechanisms that are not yet fully understood but are believed to involve alterations in glucose uptake in muscle tissue (Grossman & Messerli, 2011) [32]. Patients with other risk factors for diabetes, such as obesity or family

history, are particularly susceptible when treated with thiazides over extended periods.

3.9 Antipsychotics

Second-generation or atypical antipsychotics, such as olanzapine, clozapine, and quetiapine, are strongly associated with the development of metabolic syndrome and diabetes (Newcomer, 2005) [57]. These drugs often cause significant weight gain, increased appetite, and dyslipidemia, all of which contribute to insulin resistance. Additionally, they may directly affect pancreatic insulin secretion and alter hypothalamic regulation of appetite and metabolism (De Hert *et al*., 2011) [18]. In psychiatric populations, the risk of diabetes is compounded by sedentary lifestyles and poor dietary habits, making regular metabolic monitoring essential for early detection and intervention.

Immunosuppressants (e.g., Tacrolimus)

Immunosuppressive drugs, particularly tacrolimus and cyclosporine, are integral to post-transplant management but carry a high risk of inducing diabetes. Tacrolimus has been shown to reduce insulin gene expression and impair insulin secretion from pancreatic beta cells (Hjelmesaeth *et al*., 2004) [39]. It also induces oxidative stress and mitochondrial dysfunction within these cells, leading to β-cell damage. The risk is particularly pronounced in organ transplant recipients who already have predisposing factors like obesity, viral infections (e.g., cytomegalovirus), or a family history of diabetes (ADA, 2024) [4]. Drug-induced diabetes in these cases is often referred to as new-onset diabetes after transplantation (NODAT) and can complicate graft survival and overall patient outcomes (Dantal & Berthoux, 2008) [17].

4. Symptoms and Clinical Features

Type 5 diabetes shares many symptoms with other forms of diabetes, but often coexists with signs of the underlying disease (Knight *et al*., 2017) [47]:

4.1 Common Symptoms

- Polyuria (frequent urination).
- Polydipsia (excessive thirst).
- Polyphagia (excessive hunger).
- Weight loss.
- Fatigue.
- Blurred vision.

4.2 Unique Clinical Features

- Steatorrhea (fatty stools) due to exocrine dysfunction.
- Vitamin deficiencies (especially A, D, E, K due to fat malabsorption).
- Recurrent hypoglycemia in advanced pancreatic insufficiency.
- Delayed gastric emptying (diabetic gastroparesis).

5. Dietary management of type 5 diabetes mellitus

Dietary management plays a pivotal role in the comprehensive care of patients with Type 5 Diabetes Mellitus (Tewari, 2019) [68], a condition often triggered by pharmacological agents that impair insulin action or secretion. In such cases, dietary intervention is crucial not only to regulate blood glucose levels but also to counteract nutritional deficiencies, especially those arising from pancreatic dysfunction or medication induced malabsorption. A holistic approach that encompasses general dietary principles, precise macronutrient distribution, targeted micronutrient supplementation, enzyme replacement therapy, and adherence to structured dietary patterns can significantly enhance glycemic control and improve quality of life in these patients. General dietary guidelines emphasize maintaining a balanced intake of macronutrients, including complex carbohydrates, lean proteins, and healthy fats, while ensuring adequate hydration and avoiding simple sugars that may trigger glycemic spikes (American Diabetes Association [ADA], 2024) [4]. Consuming small, frequent meals throughout the day helps prevent postprandial hyperglycemia and reactive hypoglycemia, especially in individuals on insulin or glucose-lowering medications. In terms of macronutrient recommendations, carbohydrate intake should primarily consist of complex sources such as whole grains, legumes, vegetables, and low-glycemic index foods to stabilize postprandial blood sugar responses (Toumpanakis *et al*., 2018) [69]. Refined sugars, sweetened beverages, and high-GI foods should be minimized or eliminated to reduce glycemic variability. Protein intake should be moderate, aimed at preserving lean muscle mass and metabolic health, especially in patients with weight fluctuations due to medications like antipsychotics or immunosuppressants (Chakraborty & Singh, 2022) [8]. Easily digestible protein sources such as eggs, tofu, cottage cheese, and lentils are recommended, while protein supplementation may be necessary in patients with malabsorption syndromes or pancreatic insufficiency, particularly in those experiencing unintended weight loss. Fat intake should focus on unsaturated sources such as olive oil, avocados, nuts, and seeds, which have been shown to improve lipid profiles and reduce insulin resistance (DeFronzo *et al*., 2015) [19]. Saturated fats from red meat and dairy, and trans fats from processed foods, should be restricted to mitigate cardiovascular risk, which is already elevated in diabetic populations. Additionally, medium-chain triglycerides (MCTs), commonly derived from coconut oil, may be beneficial in patients with fat malabsorption, as they are absorbed directly into the portal vein and do not require bile salts for digestion, making them ideal for those with pancreatic enzyme insufficiency (DiMagno *et al*., 2011) [21]. Micronutrient support is equally important, especially in patients with exocrine pancreatic dysfunction, which frequently results in malabsorption of fat-soluble vitamins (A, D, E, and K). Supplementation with these vitamins is often necessary and should be guided by serum levels to avoid deficiencies that may lead to complications such as osteoporosis, visual impairment, or impaired immune function (Holt *et al*., 2021) [40]. Calcium and magnesium supplementation are vital for supporting bone density and neuromuscular function, both of which may be compromised by glucocorticoid therapy and long-term diuretic use. Zinc and B-complex vitamins also warrant attention, as they play key roles in immune function, insulin production, and glucose metabolism. For example, thiazide-induced hypokalemia can be accompanied by magnesium and zinc depletion, further worsening glycemic control and insulin sensitivity (Grossman & Messerli, 2011) [32]. When pancreatic exocrine insufficiency is confirmed often through tests like fecal elastase or 72-hour stool fat quantification pancreatic enzyme replacement therapy (PERT) should be initiated. Enzyme preparations containing lipase, amylase,

and protease can significantly improve digestion and nutrient absorption in these patients and should be taken alongside meals to mimic physiological enzyme secretion (Furukawa *et al*., 2019) [29]. This is particularly important for patients who have undergone pancreatectomy, are suffering from chronic pancreatitis, or are on medications like tacrolimus that impair pancreatic function.

Beyond individual nutrients, adopting structured dietary patterns has shown promising results in managing Type 5 diabetes and its associated metabolic complications. The Mediterranean diet, characterized by high intake of fruits, vegetables, legumes, whole grains, olive oil, nuts, and moderate consumption of fish and poultry, is rich in fibre, antioxidants, and omega-3 fatty acids. This diet has been associated with improved insulin sensitivity, better lipid profiles, and reduced markers of inflammation, making it particularly suitable for patients with diabetes secondary to metabolic and inflammatory disorders (Esposito *et al*., 2015) [29]. The diet's emphasis on plant-based foods and unsaturated fats also aligns with recommendations for managing drug-induced weight gain and dyslipidemia. Furthermore, individuals on atypical antipsychotics or glucocorticoids, who are at high risk of visceral obesity and insulin resistance, can benefit greatly from the anti-inflammatory and cardioprotective aspects of the Mediterranean dietary model. In conclusion, dietary management in Type 5 diabetes must be multifaceted, addressing not only glucose regulation but also compensating for deficiencies caused by underlying medical treatments or organ dysfunction. A well-structured dietary intervention, integrated with pharmacological treatment and lifestyle modification, is essential to reduce complications and improve the long-term prognosis of individuals with this complex, multifactorial condition.

6. Conclusion

In conclusion, type 5 diabetes mellitus, primarily characterized by pancreatic exocrine dysfunction such as in pancreatitis or cystic fibrosis, presents a complex interplay of endocrine and exocrine insufficiencies that challenge conventional diabetes management. Unlike the more common types, its onset is often linked to pancreatic injury, surgery, or long-term inflammation, making it a secondary form of diabetes. Symptoms may overlap with other types of diabetes but often include malabsorption, steatorrhea, and weight loss due to impaired enzyme production. Effective management requires a multidisciplinary approach involving blood glucose regulation, pancreatic enzyme replacement, and targeted nutritional therapy. Dietary management plays a critical role in stabilizing blood sugar, preventing nutritional deficiencies, and improving quality of life through balanced macronutrient intake, avoidance of simple sugars, frequent small meals, and adequate hydration. Recognizing the unique etiology and clinical features of Type 5 Diabetes is essential for timely diagnosis and individualized care strategies.

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