World Journal of Advanced Research and Reviews
eISSN: 2581-9615 CODEN (USA): WJARAI
Cross Ref DOI: 10.30574/wjarr
Journal homepage: https://wjarr.com/

(RESEARCH ARTICLE)

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The effect of the ketogenic diet on HbA1c levels in type 2 diabetes mellitus patients and its correlation with dental caries

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World Journal of Advanced Research and Reviews, 2025, 25(01), 001-008

Publication history: Received on 21 November 2024; revised on 28 December 2024; accepted on 30 December 2024

Article DOI: https://doi.org/10.30574/wjarr.2025.25.1.4025

Abstract

Background: Diabetes mellitus (DM) is currently a significant global health concern and a leading cause of illness and death. The prevalence of diabetes in adults is estimated to have exceeded three hundred million and is projected to increase by 55% by 2035. The ketogenic diet has been confirmed to increase insulin sensitivity in type 2 diabetes mellitus (T2DM) sufferers.

Purpose: To explain the effect of a ketogenic diet on HbA1c levels in patients with T2DM and provide an overview of its correlation with dental caries.

Discussion: Carbohydrate restriction in the ketogenic diet triggers the mechanism of gluconeogenesis and ketogenesis in the liver and changes the pattern of energy supply to be based on ketone bodies. There is a significant relationship between the risk of dental caries and fasting blood glucose levels as well as HbA1c and salivary glucose. One study showed that T2DM sufferers who had HbA1c ≥ 8.0% had more dental caries than those who had HbA1c 7.0%–7.9%.

Conclusion: A reduction in HbA1c levels resulting from a ketogenic diet intervention may be linked to a lower risk of dental caries in patients with T2DM. However, additional studies are necessary to explore the direct impact of the ketogenic diet on the risk of dental caries in these patients.

Keywords: T2DM; ketogenic diet; HbA1c; Dental caries; Good health and well being

1. Introduction

Diabetes mellitus (DM) is currently a significant global health concern and a leading cause of illness and death. The prevalence of diabetes in adults is estimated to have exceeded three hundred million and is projected to increase by 55% by 2035. Elevated blood glucose levels are the main indicator of diabetes mellitus, which is a metabolic disorder resulting from impaired insulin function or production, or a combination of both. Hyperglycemia is one of the typical symptoms of diabetes mellitus. Chronic high blood sugar levels commonly seen in individuals with DM are linked to prolonged damage, impaired function, and failure of organs, particularly the nerves, blood vessel, kidney, eyes, and heart [1].

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Diabetes mellitus is divided into two main types: type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM). T1DM is characterized by total insulin deficiency due to pancreatic B-cell destruction, which is usually triggered by autoimmune diseases. Type 2 diabetes, on the other hand, is caused by insulin resistance accompanied by relative insulin deficiency or impaired secretion, with insulin resistance as the main factor [2]. One of the main risks of T2DM is obesity or being overweight, which increases the risk twofold [3]. Obesity is responsible for 80-85% of the total risk for developing type 2 diabetes mellitus. The risk of T2DM increases linearly with the increasing body mass index (BMI) above 24 kg/m^2 [4].

The easiest parameter for diagnosis and monitoring glycemic control of T2DM is blood glucose measurement. The use of the Oral Glucose Tolerance Test (OGTT) as an index of hyperglycemia is considered relatively cheap and sensitive for detecting disorders of glucose homeostasis. However, there are still several disadvantages of the OGTT, such as wide biological variability, and its susceptibility to other factors such as food, stress, exercise, and certain drugs [1]. Glycated hemoglobin (HbA1c) is used as a marker to measure the average blood glucose concentration and has an important role in the management of diabetes mellitus (Ding et al, 2018).

The HbA1c indicator is considered superior to the blood glucose indicator because it has pre-analytical and analytical stability and well-standardized testing, providing examination results with high accuracy [1]. In January 2010, the usage of HbA1c as a T2DM diagnostic test was formally approved by health associations [6]. As part of a collaborative protocol in the management of diabetes and cardiovascular disease, the European Society of Cardiology (ESC) and the Association for the Study of Diabetes (EASD) included HbA1c in the diagnostic algorithm for type 2 diabetes (T2DM) [6]. In 2010, the American Diabetes Association recommended HbA1c levels between 5.7% to 6.4% as an indication of prediabetes, and more than 6.5% for the diagnosis of diabetes (Ding et al, 2018).

Diet plays an important role as a risk factor for type 2 diabetes mellitus (T2DM). Various large studies have highlighted specific eating habits that could raise the risk of T2DM, including the intake of processed meats, fried foods, excessive white rice, and sugary beverages [4]. The ketogenic diet, which is known for its low carbohydrate intake (under 50 grams per day), high fat, moderate protein, and low calories, has been recognized as a dietary approach for managing epilepsy. The diet utilizes ketone bodies as the main source of energy, mimicking the body's metabolic processes under starvation conditions due to low carbohydrate intake [7]. There are four variations of ketogenic diets, namely standard, cyclic, targeted and high-protein ketogenic diets [8]. Based on this, this study purposes to analyze the effect of the ketogenic diet on HbA1c levels in patients with T2DM and examine its relationship with the risk of dental caries.

1.1. Diabetes Mellitus

Type 2 diabetes is more prevalent than type 1 diabetes mellitus (DM), a chronic metabolic disorder that is increasingly being diagnosed worldwide. Type 1 diabetes results from an autoimmune attack that harms pancreatic beta cells, hindering insulin production. The onset of this condition is largely influenced by genetic and environmental factors. In contrast, type 2 diabetes is linked to insulin resistance and compromised function of pancreatic beta cells, often triggered by obesity, poor lifestyle choices, and the intake of high-calorie foods [9].

Several risk factors for type 1 diabetes include a family history of the condition, specific viral infections, and genetic predispositions. Type 2 diabetes, on the other hand, is influenced by a family history of the disease, obesity, advancing age, and physical inactivity. Acute complications of diabetes mellitus (DM) involve severe hypoglycemia and diabetic ketoacidosis (DKA), while long-term complications encompass macrovascular conditions such as coronary artery disease and stroke, along with microvascular problems like diabetic retinopathy, nephropathy, and neuropathy [9].

The mechanism of DM varies depending on the type. In type 1 DM, damage to pancreatic beta cells due to an autoimmune reaction causes the body to lose its ability to produce insulin. Meanwhile, in type 2 DM, The primary cause is insulin resistance in target tissues like the liver, muscle, and adipose tissue, combined with the inability of pancreatic beta cells to compensate for insufficient insulin production. This condition disrupts blood glucose regulation and causes chronic hyperglycemia [9].

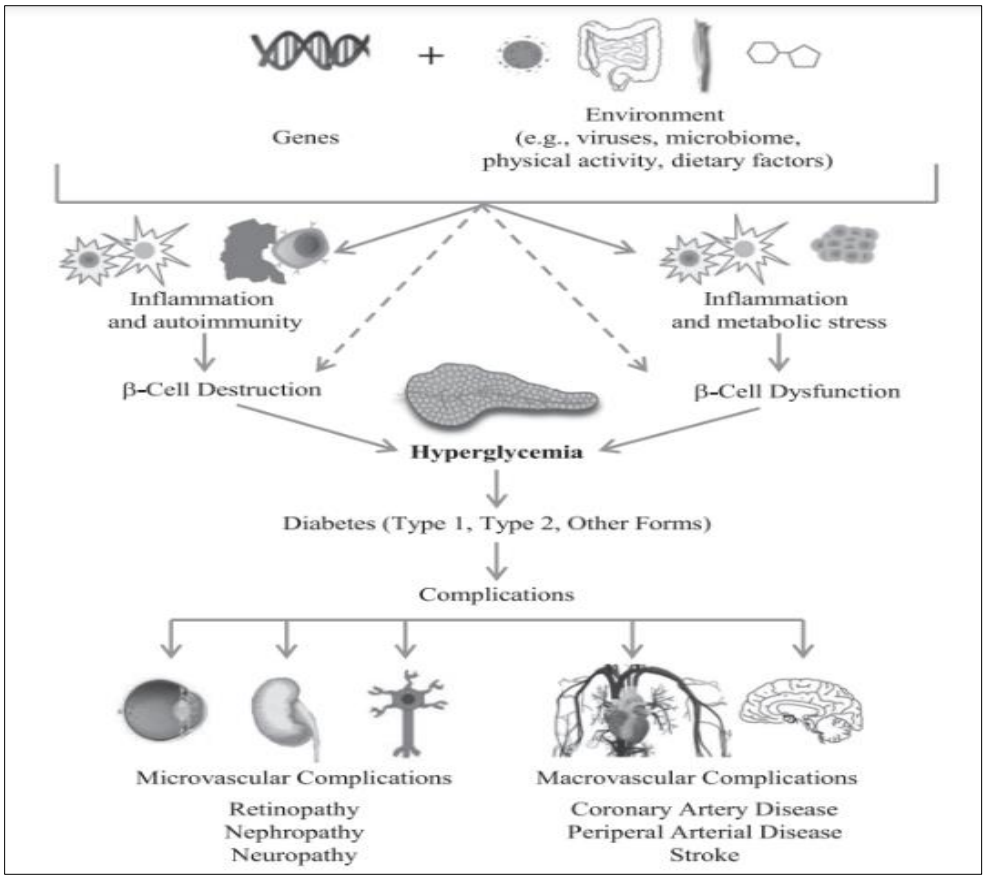

Figure 1 Mechanism of Diabetes Mellitus

1.2. HbA1c

HbA1c is a test used to measure a person's blood glucose levels, reflecting the average glucose levels in the body over a 90-day period [10]. In general, hemoglobin is divided into three types: HbA (α2β2), HbA2 (α2δ2), and HbF (α2γ2). About 6% of total HbA consists of HbA1, which is further separated into four subtypes (HbA1a1, HbA1a2, HbA1b, and HbA1c) according to their characteristics in electrophoresis and chromatography [11].

According to Ding et al. (2018), the biochemical explanation for HbA1c is that glucose is persistently attached to the hemoglobin β-chain's N-terminal valine. Hemoglobin becomes glycated through a non-enzymatic reaction involving glucose and the N-terminal of the β-chain, leading to the creation of a Schiff base [5]. This Schiff base then undergoes a reorganization, forming the Amadori product, or HbA1c. Hemoglobin and blood glucose combine in an adjustable reaction to generate aldimine during the first step of HbA1c production. Aldimine is then gradually transformed into a stable form of ketoamine during the subsequent, irreversible stage. The normal physiological cycle includes the production of HbA1c. Nonetheless, the rise in plasma glucose levels is directly related to the rise in HbA1c levels. A key characteristic of the HbA1c test is its capacity to estimate the average blood glucose level from the past two to three months [11].

HbA1c has been recommended as a diagnostic tool for diabetes mellitus for decades. However, limited access to HbA1c testing methods as well as the absence of clear standards make its use as a diagnostic tool not fully accepted until now. [5]. The advantage of the HbA1c test as a tool for measuring blood glucose levels and a means of diagnosing diabetes mellitus is that it can estimate blood glucose levels over a wide period of time, so the variability rate is low; no need to fast or use a specific time sample; is a standardized measurement; and illustrates the association with chronic complications better than fasting blood glucose (Ding et al 2018).

1.3. Dental caries

Dental caries is an infectious disease that causes significant health problems in both developed and developing countries. It is the primary cause of tooth loss in both adults and children. Over the past 20 years, dental caries has become increasingly prevalent in emerging nations such as Thailand and Indonesia. Despite advances in dental care,

dental caries continues to be a major problem, Even in industrialized nations like the United States and certain regions of Europe. According to the WHO Oral Health Database, the DMFT (decay, missing, filled teeth) index for 12-year-old children in 188 countries continues to show significant values. The epidemiology of dental caries in various developing countries, including those in Asia (Indonesia, Thailand, the Philippines, China, and Taiwan), Africa (Zambia, Sudan, and Nigeria), and South America (Brazil), has recorded an increase in prevalence over the last 10 years [12].

Dental caries results from the ongoing loss of minerals in tooth tissue, which is triggered by acid production from the fermentation of carbohydrates by bacteria that cause cavities. This acid lowers the pH of saliva. Diets high in carbohydrates, especially sucrose, can decrease the pH of the biofilm on the tooth surface, reaching levels that favor the growth of Streptococcus mutans (S. mutans). The fermentation of carbohydrates by these bacteria produces acid, which destroys hydroxyapatite crystals, leading to the demineralization of enamel and dentin in hard tooth tissue [12].

Saliva plays an vital role in controlling the risk of dental caries, particularly through Secretory Immunoglobulin A (sIgA), which acts as an antibacterial agent. Factors influencing the development of caries include the host's immune response, the presence of plaque bacteria as antigens, dietary patterns, and the duration of exposure. Furthermore, both genetic and environmental factors are thought to play a role in the risk of developing caries. Research indicates a link between genetic factors and the immune response to dental caries. Host genetics influence antigen recognition, immune responses, and dietary habits. Studies in both humans and animals show that genetic differences can lead to variations in the immune response to antigens that play a role in dental caries [12].

The process of dental caries involves the interaction between the hard tissue of the tooth and microbial biofilm. This condition affects calcified tooth tissue, including the crown and root, which becomes exposed following gingival recession. Caries is triggered by an imbalance in the cariogenic biofilm and occurs only when there is an active pathogenic biofilm along with frequent exposure to carbohydrates in the diet, particularly free sugars such as fructose, sucrose, maltose and glucose. As a result, dental caries is considered a disease influenced by both diet and microbial activity. Fluoride acts a critical role in inhibiting the development of caries by modifying the expression of the disease [13].

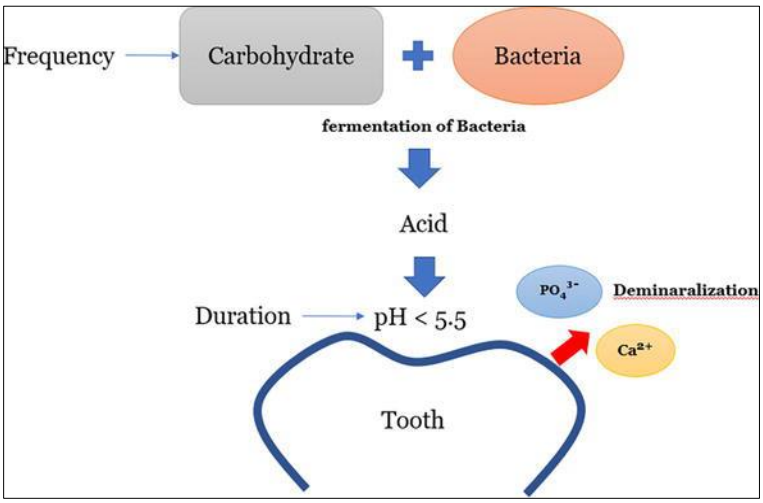

Figure 2 Mechanism of dental caries development

1.4. Diet type

Based on their function, there are many types of diets designed for health purposes. Some of these include the Atkins Diet, which limits carbohydrate consumption to less than 20 g per day for the first two weeks, then gradually increases to 50 g per day, with recommended exercise. The Vegetarian Diet focuses on plant-based foods and avoids protein and animal products, with no specific calorie or exercise recommendations. The Mediterranean diet focuses on eating healthy foods such as nuts, fish, lean meats, and whole grains, with a recommendation to exercise, but without specific calorie restrictions. The Paleo Diet focuses on natural foods such as meat, fruit, and vegetables, while avoiding processed sugar, dairy, and grains, with exercise recommended. The Ultra Low Fat Diet provides 75-80% of calories from plant-based foods and limits food heating to 115°F (46°C), with no specific calorie or exercise recommendations. The Zone Diet regulates the calorie composition with a proportion of 40% carbohydrates, 30% protein, and 30% fat, and

encourages exercise without specific calorie restrictions. Meanwhile, the ketogenic diet emphasizes a high-protein, low-carbohydrate, and high-healthy fat diet, without exercise recommendations or calorie specifications [14].

1.5. Ketogenic diet

Initially, the ketogenic diet was used as an alternative therapy for epilepsy patients. In recent years, however, it has gained attention for its potential benefits in managing obesity and type 2 diabetes mellitus (T2DM). This diet involves strictly limiting carbohydrate intake to only 5-10% of total daily energy requirements (approximately 20-50 grams per day), with moderate protein intake (20-25%) and very high fat intake (70-75%). Carbohydrate restriction is achieved by eliminating nutrients such as nuts, fruits, starchy vegetables, and grains from the daily diet. High fat and low carbohydrate consumption encourages the liver to break down fat, which is influenced by hormone-sensitive lipase (HSL), into free fatty acids (FFA). FFA then undergoes beta-oxidation in liver mitochondria, producing ketone bodies (ketogenesis). This process reduces insulin levels and inhibits lipogenesis, forcing the body to rely on fat as its main energy source [15].

The ketogenic diet offers several health benefits, including improved insulin sensitivity and glucose control (by reducing insulin resistance, insulin requirements, and HbA1c levels), aiding weight loss in obese individuals, improving lipid profiles, and reducing symptoms of metabolic syndrome. However, the ketogenic diet can also cause side effects in T2DM patients. A study by Goday et al. reported that during a 15-day very low carbohydrate ketogenic diet (VLCKD) intervention, 20% of patients experienced dizziness and nausea, 15% experienced weakness and vomiting, and 17% of patients experienced constipation in the fourth month. Additionally, research shows that reducing carbohydrate consumption, as observed with the ketogenic diet, can increase the risk of nutritional deficiencies, including vitamin D, vitamin B complex, calcium, and beta-carotene [15, 26].

In general, foods containing carbohydrates are absorbed as glucose, which causes blood glucose levels to rise rapidly in T2DM patients. The ketogenic diet, which emphasizes high fat and protein intake with low carbohydrates, can trigger the body to enter a state of starvation ketosisIn this condition, the body transitions from relying on glucose for energy to utilizing ketones. This ketone-derived energy requires fat to increase catabolism and decrease fat synthesis. Insoluble triglycerides will be converted into water-soluble ketone bodies, such as acetoacetate, β-hydroxybutyrate, as well as water-insoluble acetone, enabling the body to convert ketone bodies into energy [16].

Carbohydrate consumption limited to under 50 g per day stimulates the processes of gluconeogenesis and ketogenesis in the liver [17]. When glycogen stores in the body become depleted, the body activates the gluconeogenesis mechanism, causing endogenous glucose production in the liver to shift from glucose to glycerol, lactic acid, and the amino acids alanine and glutamine. Meanwhile, ketogenesis typically occurs when endogenous glucose reserves are exhausted. This leads to a reduction in blood insulin levels, which in turn restricts the body's ability to store fat and glucose [18].

2. Discussion

2.1. Effect of Ketogenic Diet on HbA1c Levels

The substantial decrease in insulin resistance assessment (HOMA-IR) reinforces the idea that a ketogenic diet may aid in lowering oxidative stress and enhancing insulin sensitivity in individuals with type 2 diabetes (T2DM) [19]. In this regard, HOMA-IR is regarded as an indicator for assessing insulin resistance in individuals with type 2 diabetes. Furthermore, it is believed that the increase in peripheral blood ketosis brought on by the ketogenic diet intervention will improve peripheral insulin sensitivity through the following mechanisms: lowering stress associated with hyperinsulinemia, decreasing the need for external insulin, and blocking its secretion, which will reduce blood glucose levels and insulin resistance [20]. According to Gershuni et al. (2018), ketone bodies, which are produced as a result of hepatic glucose metabolism, may have a direct impact on the drop in blood glucose levels and HBA1c [7]. Furthermore, ketones can raise the amount of glucose in cells and have metabolic effects which mimic the effects of insulin without triggering the insulin signaling. pathways. This means that moderate ketosis can be used therapeutically to treat insulin resistance [27].

Compared to other diets, a study conducted by Sumei Li et al. (2022) found that the group undergoing ketogenic diet intervention showed a more significant reduction in HbA1c levels. As ketone bodies play an important role in providing energy to the body, the rates of blood glucose reduction, blood fat reduction, and weight loss were much higher than those in the control group [21].

A study by Myette Cote et al. (2018) conducted over 6 to 12 months found that the group following the diet experienced a significant decrease in HbA1c and fasting glucose levels compared to the control group. Furthermore, the research confirmed that the ketogenic diet can rapidly and effectively improve blood glucose regulation by decreasing fasting insulin levels, reducing intermittent glucose levels, and minimizing fluctuations in blood glucose among patients with T2DM [22].

Although many studies suggest that the ketogenic diet is beneficial for controlling blood glucose, some studies report the opposite. In their research, Saslow et al. (2017) demonstrated that the ketogenic diet with low carbohydrates did not result notable differences in the rate of blood glucose reduction compared to the group following the control diet. Their study further indicated that a 12-month ketogenic diet intervention did not produce significant changes in HOMA-IR, implying no improvement in insulin resistance in individuals with T2DM. Additionally, Wong et al. (2021) noted that carbohydrate restriction on a ketogenic diet, when combined with medication or insulin, could increase the risk of hypoglycemia [23, 24, 25, 28].

However, the ketogenic diet can still be applied to T2DM patients who do not experience complications, provided they are regularly monitored for blood glucose and ketone levels, as well as the dosage of anti-diabetic medications, to prevent hypoglycemia.

2.2. Risk of Caries in T2DM Sufferers

Dental caries is a long-term microbiological condition that leads to the destruction of tooth enamel and dentin, caused by lactic acid generated by bacteria fermenting carbohydrates from food. The development of dental caries involves two primary groups of bacteria: Streptococcus mutans (S. mutans), as the main cause of caries, as well as Lactobacilli that play a more active role in the development of this disease, dental caries and diabetes mellitus are intertwined in a complex relationship. Factors such as age, blood glucose levels, saliva secretion and oral hygiene influence this relationship [29].

Controlled and uncontrolled chronic T2DM conditions influence the incidence of caries. In uncontrolled diabetes mellitus sufferers, glucose levels in the gingival crevicular fluid (GCF) are higher than in controlled T2DM sufferers or non-sufferers. This will cause more bacterial growth in the mouth, thereby accelerating the formation of biofilm and plaque on the surface of the teeth which will facilitate the occurrence of dental caries [30]. Furthermore, the correlation between diabetes and dental caries is correlated to carbohydrate consumption and increased insulin limitation can causehyposalivation and increased salivary glucose levels, which causes dental caries [31].

2.3. Correlation of HbA1c Levels with the Occurrence of Caries

Individuals with T2DM frequently experience a reduction in saliva production, despite the fact that pH and saliva flow rate are key factors in protecting against dental caries in diabetes. Saliva plays a crucial role in oral health through both general physicochemical properties and more targeted effects. Proteins such as proline, statherin, and histatin influence calcium phosphate dynamics, the early stages of plaque development, and candida infections. In addition, saliva containing fluoride, calcium, and phosphate helps in the process of plaque mineralization, which in turn contributes to preventing caries [29].

A notable association exists between the risk of dental caries and fasting blood glucose levels, HbA1c, and glucose present in saliva. Almusawi et al. (2018) found that the glucose concentration in the saliva of T2DM patients was considerably elevated, which contributed to a higher risk of dental caries [29]. This aligns with the findings of Goodson et al. (2017), which also indicated hyperglycemia from T2DM results in increased glucose levels in saliva. Such an environment can make the mouth more acidic and alter the oral microbiome. This mechanism underlines the connection between hyperglycemia and an elevated risk of tooth gingivitis, erosion, and caries [32].

In an observation conducted by Mohan et al, (2022) it was found that T2DM patients with low glycemic control (HbA1c > 8%) had a very high risk of dental caries. This is proven by the results of research which shows that T2DM sufferers who have HbA1c ≥ 8.0% highly susceptible to dental caries than those who have HbA1c 7.0% –7,9% [33].

3. Conclusion

Implementing ketogenic diet intervention in T2DM sufferers can reduce HbA1c levels and increase insulin sensitivity. The risk of dental caries is closely related to salivary HbA1c and glucose levels. The reduction in HbA1c levels due to ketogenic diet intervention can be correlated with a reduced risk of dental caries in T2DM sufferers. However, further research is needed to understand the direct effect of ketogenic diet on dental caries risk in individuals with T2DM.

Compliance with ethical standards

Disclosure of conflict of interest

No conflict of interest to be disclosed.

Statement of informed consent

Informed consent was obtained from all individual participants included in the study.

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