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Exploring the Nexus Between Vitamin D and Diabetes: Are Men and Women Affected Differently?

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Abstract **Original Research Article**

Vitamin D is increasingly recognized for its role in metabolic regulation, including its influence on insulin secretion, glucose metabolism, and overall endocrine function. Emerging evidence suggests that inadequate vitamin D levels may contribute to disturbances in metabolic homeostasis. Additionally, physiological and hormonal differences between men and women may influence vitamin D metabolism and its effects on metabolic parameters. However, gender-specific variations in the association between vitamin D status and metabolic indicators remain insufficiently explored. The present study aims to investigate the relationship between serum vitamin D levels and selected metabolic parameters and to evaluate potential gender-based differences in this association. The study included a total of 200 participants, comprising both male and female subjects. Serum vitamin D concentrations and relevant biochemical parameters were measured and analysed using appropriate statistical methods to determine patterns of association and gender-related variations.

Keywords: Vitamin D, Type 2 Diabetes, Glycaemic control, Gender Distribution, Glucose Metabolism.

INTRODUCTION

Diabetes mellitus is one of the most common endocrine diseases, characterized by an increase in plasma glucose. Different forms of diabetes with very distinct pathogenesis exist. Over time, diabetes can lead to blindness, kidney failure, and nerve damage. Diabetes is also an important factor in accelerating atherosclerosis, leading to stroke, coronary heart disease, and other large blood vessel diseases, all ultimately associated with increased mortality risks. The most prevalent form of diabetes is type 2 diabetes, currently affecting more than 300 million people worldwide. It is characterized by the combination of insulin resistance and failing β-cell function. The discovery of receptors for 1α,25-dihydroxyvitamin D3 (1,25(OH)2D3), the activated form of vitamin D, in tissues with no direct role in calcium and bone metabolism (e.g. pancreatic beta cells and cells of the immune system) has broadened our view of the physiological role of this molecule [1]. An increased prevalence of type 2 diabetes has been described in vitamin D-deficient individuals, and insulin synthesis and secretion have been shown to be impaired in beta cells from vitamin D-deficient population. Glucose tolerance is restored when vitamin D levels return to normal. There is accumulating evidence that suggests that altered vitamin D and calcium homeostasis may play a role in the development of type 2 diabetes. Cross-sectional studies showed that low vitamin D status was associated with prevalence of glucose intolerance or diabetes, and observational studies showed that low vitamin D status was associated with incidence of type 2 diabetes [3].

MATERIALS AND METHODS

The study was conducted in a tertiary care centre. The data were obtained from patients who attended the out-patient department of our chief general physician.

Inclusion criteria:

Individuals diagnosed to have type II diabetes who were >21 years of age Individuals with no previous history of intake of vitamin D supplements

Exclusion criteria:

- Individuals diagnosed with type I diabetes
- Individuals with previous history of diagnosed vitamin D insufficiency
- Individuals with previous history of treatment with vitamin D supplements and calcium

Individuals who were diagnosed to have type II diabetes attended the out-patient department were randomly selected. Detailed history was obtained among the screened patients and people not fitting into the criteria were excluded. A total of 200 individuals were selected, 70 males and 130 females involving various age groups. Their serum 25- hydroxy cholecalciferol levels were assessed. The results were analysed in gender distribution and studied.

RESULTS

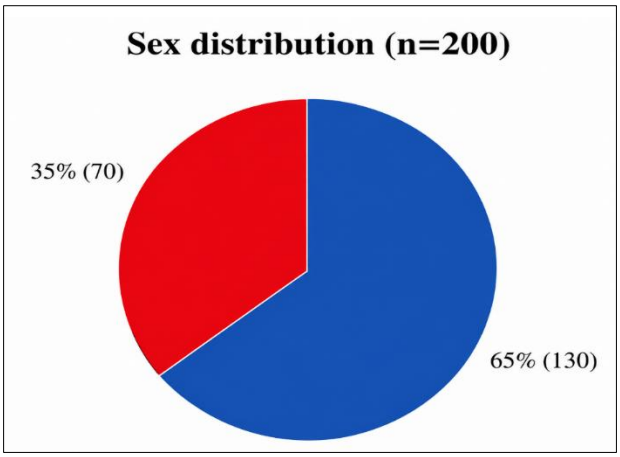
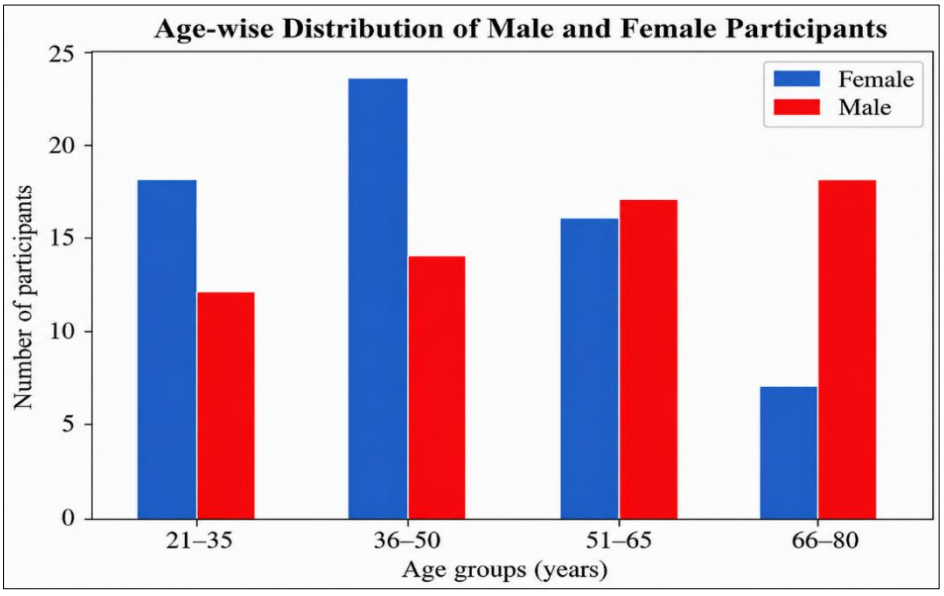


Figure 1: Among 200 participants, 130 (65%) were females and 70 (35%) were males

Among gender distribution, there were 70 males and 130 females in the study group. More number of individuals were found in the age between 36 to 50 years of age – mid of fourth and fifth decade of life. That is the time period they were first diagnosed with type II diabetes. The patients with well controlled sugar levels presenting with vague muscle fatigue and generalised malaise all over body whose vitamin D levels checked were found to be insufficient. When they were supplemented with oral vitamin D supplements, they symptomatically recovered better. Vitamin D insufficiency also presents with such vague malaise and fatigue symptoms.

Serum 25-OH, cholecalciferol levels	
Deficiency	< 10 ng/ml
Insufficiency	10 – 30 ng/ml
Sufficiency	30 – 70 ng/ml

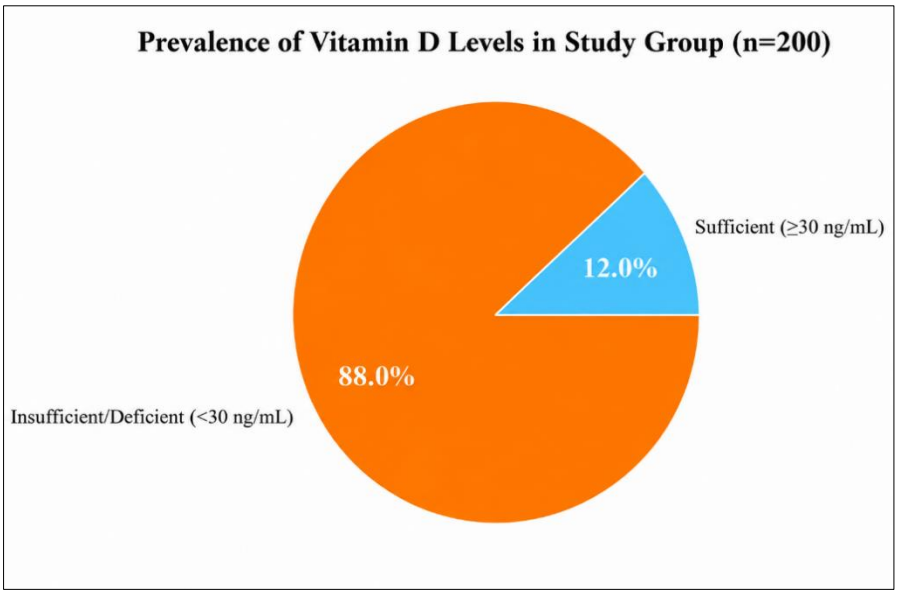
Age distribution:



Mean distribution of serum S.25-OH cholecalciferol among diabetics

Sex	Mean S.25-OH cholecalciferol (ng/ml)	Vitamin D status
Male diabetics	21.9 ng/ml	Insufficient
Female diabetics	12.9 ng/ml	Insufficient

Vitamin D status assessment revealed a high prevalence of insufficiency and deficiency in the study population. Of the 200 participants evaluated, only 24 individuals (12%) had sufficient serum 25-hydroxycholecalciferol levels (≥30 ng/mL), while the remaining 176 participants (88%) were classified as vitamin D insufficient or deficient. Gender wise analysis demonstrated notable differences in vitamin D status. Among male participants (n=70), 14 individuals (20%) exhibited sufficient vitamin D levels, whereas 56 participants (80%) were insufficient or deficient. In contrast, vitamin D insufficiency or deficiency was more pronounced among female participants (n=130), with 120 individuals (92.3%) showing inadequate vitamin D levels and only 10 participants (7.7%) achieving sufficiency. These findings highlight a substantial burden of hypovitaminosis D in the study population, particularly among females.



REVIEW OF LITERATURE

Vitamin D is a secosteroid that is generated from 7-dehydrocholesterol in skin under the influence of UV light. Therefore, by definition, vitamin D cannot be considered a true vitamin but rather a prohormone, as the natural source of vitamin D in evolution of vertebrates and primates is photosynthesis in the skin. Indeed, the normal human diet is usually poor in vitamin D except for fatty fish. Vitamin D deficiency is prevalent in infants who are solely breastfed and who do not receive vitamin D supplementation and in adults of all ages who have increased skin pigmentation or who always wear sun protection or limit their outdoor activities. Vitamin D deficiency is often misdiagnosed as fibromyalgia [1].

Vitamin D metabolism:

Most vertebrates synthesise vitamin D in their skin under the influence of UV light. An 'efficient' sun exposure - exposure of the face and hands to the sun for 2 hr/ weeks is probably sufficient to maintain normal levels. The findings of the present study indicate that vitamin D insufficiency and deficiency were significantly more prevalent among female patients. This higher prevalence may be related to social and cultural practices, including religious customs that require women to cover most parts of the body and wear a hijab when outdoors. Such clothing substantially reduces exposure of the skin to ultraviolet B radiation, which is essential for cutaneous synthesis of 25-hydroxyvitamin D. Exposure limited to the face and hands, as observed in hijab-wearing women, appears to be inadequate for maintaining sufficient vitamin D levels, even in regions with ample sunlight [2]. Food supplementation is required during pregnancy and lactation and for newborns and young children (especially in dark-skinned children living in northern countries). Vitamin D can be obtained from dietary sources of vegetable (vitamin D2, also known as ergocalciferol) or animal origin (vitamin D3, also known as cholecalciferol). The best food sources are fatty fish or their liver oils; however, small amounts are also found in butter, cream and egg yolk. Human and cow's milk are poor sources of vitamin D. In many parts of the world, especially North America, fluid and dried milk, as well as some margarines, butter and

cereals are supplemented with vitamin D. However, the real vitamin D content is frequently quite different from the labelling standard and often insufficient to reach the daily requirements for vitamin D (400–600 IU/day). Skimmed milk, in particular, frequently has no detectable vitamin D. Vitamin D3 itself is biologically inert and requires two successive hydroxylation's, one in the liver (on C25) and one in the kidney (on the α position of C1), to form its hormonally active metabolite, 1,25(OH)2D3 (Fig 1) Liver25-hydroxylases and kidney 1α-hydroxylase belong to the large family of cytochrome P450-dependent steroid hydroxylases [5].

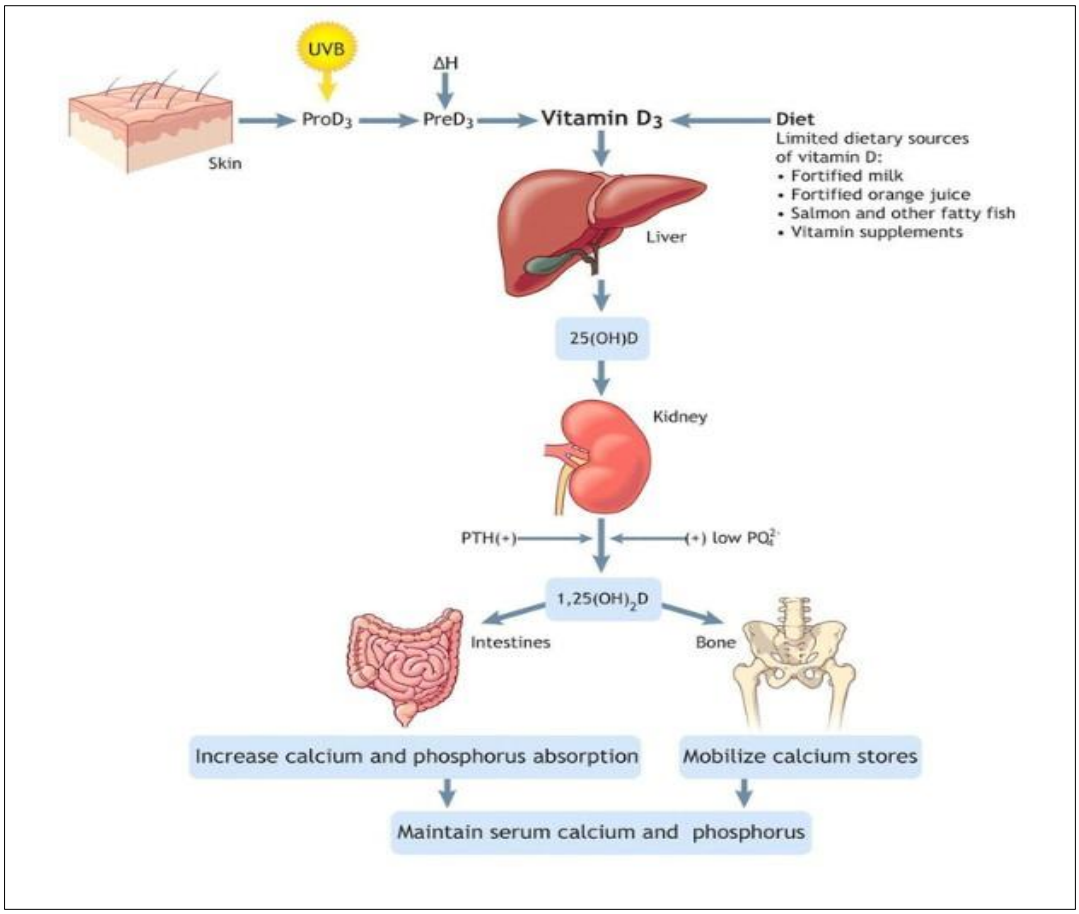


Fig 2

Type 2 diabetes:

Type 2 diabetes is a disorder that results from defects in both insulin secretion and insulin sensitivity, and accounts for 90% of all diabetes cases. The growing rate of type 2 diabetes is worrisome, with in the United States alone an estimated 1 million new cases every year. Initially, patients counteract their increased insulin resistance and stabilize circulating glucose levels through increased insulin production by pancreatic beta cells. As the disease progresses and functional alterations are accentuated, patients show decreased insulin secretion, and eventually they can also present loss of β cell mass [6]. The exact mechanisms involving the development are still unknown, but lifestyle (e.g., obesity, sedentary lifestyle, and unhealthy eating habits) and genetic components (e.g., PPARG and CAPN10 genes, and a whole set of gene polymorphisms each with small contributing effects) seem to be involved. This disease is most prevalent in obese, sedentary individuals with a concomitant elevation in free fatty acids and proinflammatory cytokines, and relatives of T2D patients also have an increased probability of developing this disease [7].

Vitamin D in glucose metabolism:

There is growing evidence that vitamin D deficiency could be a contributing factor in the development of both type 1 and type 2 diabetes. First, the β-cell in the pancreas that secretes insulin has been shown to contain VDRs as well as the 1 alpha hydroxylase enzyme. (Fig 2) Evidence indicates that vitamin D treatment improves glucose tolerance and insulin resistance. Vitamin D deficiency leads to reduced insulin secretion. Supplementation with vitamin D has been shown to restore insulin secretion in animals. Researchers have also found an indirect effect on insulin secretion, potentially by a calcium effect on insulin secretion. Vitamin D contributes to normalization of extracellular calcium, ensuring normal calcium flux through cell membranes; therefore, low vitamin D may diminish calcium's ability to affect insulin secretion.

Other potential mechanisms associated with vitamin D and diabetes include improving insulin action by stimulating expression of the insulin receptor, enhancing insulin responsiveness for glucose transport, having an indirect effect on insulin action potentially via a calcium effect on insulin secretion, and improving systemic inflammation by a direct effect on cytokines [8]. Vitamin D may also influence insulin resistance indirectly through modulation of the renin–angiotensin–aldosterone system (RAAS). Additionally, vitamin D insufficiency has been associated with increased fat infiltration in skeletal muscle, independent of body mass, which may contribute to reduced insulin sensitivity [4].

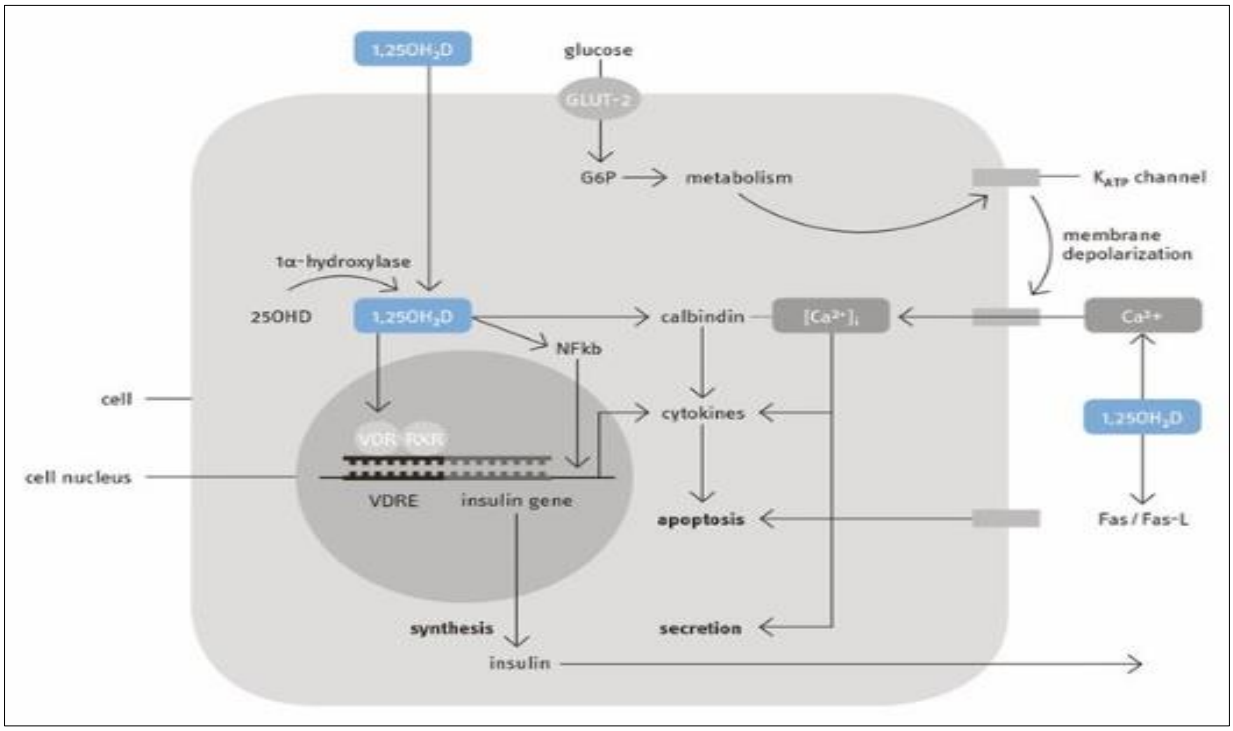


Fig 3

Vitamin D and type II diabetes:

The National Health and Nutrition Examination Survey (NHANES) III study between 1988 and 1994 demonstrated that there is a strong inverse association between low levels of 25(OH)D and diabetes prevalence. Low vitamin D levels have also been shown to be predictive of the future development of type 2 diabetes [9]. One study showed that increasing vitamin D serum levels to normal led to a 55% relative reduction in the risk of developing type 2 diabetes [10]. As with most disease states and vitamin D, prospective studies related to vitamin D supplementation and diabetes are rare and limited. Prospective trials of vitamin D and diabetes to date were either too small or used inadequate amounts of vitamin D [11].

Kaamini *et al* performed a linear regression analysis of 712 subjects after evaluating serum 25(OH)D levels and assessing insulin sensitivity by means of the homeostasis model of insulin resistance. Their results indicated that vitamin D was significantly correlated to insulin resistance and β-cell function in their multiethnic sample. The researchers concluded that low vitamin D levels may play a significant role in the pathogenesis of type 2 diabetes [12]. Impaired glucose tolerance (IGT) and impaired fasting glucose (IFG) are prediabetic states characterized by insulin resistance and β-cell dysfunction. IGT primarily involves peripheral (muscle) insulin resistance with defects in both early- and late-phase insulin secretion, whereas IFG mainly reflects hepatic insulin resistance with impaired early- phase insulin secretion. These distinct mechanisms may require different preventive approaches [13]. Recent large observational studies indicate an association between vitamin D deficiency and the development of type 2 diabetes. Vitamin D influences insulin action and may affect multiple pathways involved in diabetes pathogenesis. Further research is needed to determine whether vitamin D supplementation can help prevent type 2 diabetes [14].

Matilla *et al* studied the population study considering women of South Asian origin living in Auckland, New Zealand there was a threefold higher prevalence of self-reported diabetes in South Asians living in New Zealand compared with the general population that have been previously reported a prevalence of hypovitaminosis D of 84 % in South Asian women [15]. Therefore, vitamin D can affect tissues that are not only involved in calcium homeostasis and bone metabolism. It has been suggested that altered vitamin D status may play a role in the development of diabetes

mellitus [16]. Kunadian *et al* analysed 1811 patients undergoing coronary angiography (after excluding 47 patients on vitamin D supplementation), 530 were females and 1281 were males. An independent association was observed between female gender and vitamin D deficiency, with women showing lower 25(OH)D levels compared to men. Hypovitaminosis D was significantly associated with greater severity and more aggressive coronary artery disease (CAD) in females but not in males. These findings suggest that vitamin D deficiency plays a more critical role in influencing CAD severity in women, highlighting the need for future studies to evaluate the potential benefits of vitamin D supplementation, particularly among females [17].

Muscoguiri *et al.,* studied 500 participants (250 males and 250 females), where BMI was comparable between sexes, males demonstrated significantly higher Vitamin D concentrations than females. Vitamin D insufficiency was more prevalent among females, whereas vitamin D sufficiency was higher among males. While the prevalence of vitamin D deficiency did not differ significantly between sexes, a higher trend was observed in females. Across all BMI categories, females exhibited significantly lower Vitamin D levels, which may be largely attributed to their higher fat mass percentage (FM%). Females with vitamin D deficiency had significantly higher FM% than males ($p < 0.001$), with similar trends observed in the insufficiency and sufficiency groups. These findings indicate that sex and body composition are important determinants of vitamin D status and should be considered in future supplementation guidelines. Overall, the results suggest that females are more prone to lower vitamin D levels and related deficiency compared to males [18].

Sharma *et al.,* study was conducted to compare the serum vitamin D levels between two groups: hypothyroidism patients (n = 30) and healthy control subjects (n = 30) among females aged 18–65 years. The results of the study revealed that among the hypothyroid population, 73.3% (n = 22) had low levels of vitamin D, while 26.6% (n = 8) had normal vitamin D levels. Similarly, Kumari *et al.,* demonstrated that vitamin D deficiency was present in both hypothyroid and euthyroid populations. Their findings suggested that decreased vitamin D levels in hypothyroid patients may indicate a possible association between vitamin D deficiency and autoimmune hypothyroidism. Furthermore, Turashvili *et al.,* conducted a gender-based analysis and reported that hypothyroidism was more prevalent among women than men. The study also found that vitamin D deficiency was significantly higher in women, suggesting a potential association between vitamin D deficiency and hypothyroidism, particularly among females [19].

CONCLUSION

Accumulating evidence indicates that vitamin D deficiency adversely affects pancreatic βcell function, impairs glucose tolerance, and increases the risk of type 2 diabetes mellitus (T2DM). Furthermore, vitamin D deficiency during early life has been associated with a greater likelihood of developing autoimmune diabetes in later years. These findings clearly demonstrate that vitamin D deficiency is undesirable not only for skeletal health and calcium homeostasis but also for maintaining optimal glucose metabolism.

Current evidence supports a significant role of vitamin D status in both the development and management of diabetes. Optimal serum vitamin D concentrations may vary among individuals at risk for diabetes, patients with established diabetes, and healthy populations. Adequate vitamin D supplementation may potentially reduce the incidence of T2DM and improve metabolic outcomes in affected individuals.

Similarly in the context of the present study, females demonstrated lower vitamin D levels compared to males, suggesting an increased susceptibility to vitamin D deficiency among diabetics. Considering the established association between vitamin D deficiency and impaired glucose metabolism, this study highlights that females may be at a comparatively greater risk of developing T2DM in the presence of vitamin D deficiency.

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