Original Article

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Prevalence and Clinico-Epidemiology of Vitamin D deficiency in Patients with Type-2 Diabetes Mellitus and Hypertension: A Cross-sectional, Observational, Pan-India Study

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Abstract

Background: The objective of this clinico-epidemiological, Pan-India study was to evaluate the prevalence of vitamin D (vitD) deficiency in Indian patients with type-2 diabetes mellitus (T2DM), hypertension (HT) and both T2DM and HT and to understand their management practices. *Methods:* Adults with a diagnosis of T2DM, HT or both were enrolled across 29 sites in India. All the patient-related data were extracted from medical records. VitD insufficiency and deficiency was defined as serum 25(OH)D levels 21-29 ng/ml and ≤20 ng/ml, respectively. *Results:* A total of 1501 (99.5%) patients completed the study (T2DM:99.2%; HT:99.6%; T2DM+HT: 99.8%). Mean age at diagnosis of vitD deficiency was 52.5±10.77 years. Prevalence of patients with low vitD levels (vitD deficiency [60.9%] and insufficiency [22.9%]) was 1257 (83.7%); 1231 (82%) were newly diagnosed cases. Prevalence of low vitD levels amongst patients with T2DM, HT and T2DM+HT was 84.2%, 82.6% and 84.5%, respectively. Out of 1257 patients, 84.8% received vitD supplementation. Preferred dose and route of administration was 60,000 IU (70.2%) and oral (79.6%). Preferred frequency of dose was once in a week (76.7%). VitD deficiency (26.9%), vitD insufficiency (34.5%), symptoms of vitD deficiency (10.4%) and co-morbid condition (1.8%) were the factors considered while prescribing vitD supplements. *Conclusion:* Prevalence of vitD deficiency was higher in newly diagnosed cases compared to already diagnosed cases in all three cohorts. This study indicates the magnitude of overlap between vitD deficiency and T2DM or HT, and the need for routine screening for early diagnosis and effective management.

Keywords: Deficiency; hypertension; type-2 diabetes mellitus; insufficiency.

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Introduction

Non-communicable disease continues to be an imperative public health problem in India, leading to substantial increase in mortality and morbidity. Among these, Type-2 diabetes mellitus (T2DM) and hypertension (HT) are increasing at an alarming rate [1].

Defects in pancreatic β-cell function, insulin sensitivity, and systemic inflammation are few of the contributing factors towards the development of T2DM. On the other hand, HT results due to the imbalance between vasoconstriction and vasodilation, favoring vasoconstriction. Genetic and epigenetic factors, including nutritional risk factors, plays a major role in the pathogenesis of these diseases.

Recently, an intersecting underlying pathology between T2DM and HT with vitamin D (vitD) insufficiency/deficiency has been noted [2-4]. The presence of vitD receptors on the pancreatic beta cells, adipose tissues and skeletal muscle cells indicates the function of vitD in the glucose metabolism [5-7]. Further, it has been propounded that altered vitD and calcium homeostasis may play a role in the development of T2DM [8]. VitD enhances the synthesis of insulin hormone and its release from the pancreatic beta cells. VitD may play

a functional role on glucose tolerance through its effects on insulin secretion and insulin sensitivity [9]. Furthermore, 25-hydroxyvitD (25[OH] D) inhibits the release of the pro-inflammatory cytokine TNFα, regulates the activity of NF-κB, and suppresses the expressions of TLR2 and TLR4 proteins and mRNA in human monocytes, reducing the release of cytokines. Thus, vitD may also function to reduce insulin resistance and the risk of diabetes by decreasing inflammatory responses [10,11]. Thus, patients with low vitD levels may be at higher risk of developing T2DM, worsening glycemic control, increasing serum lipid levels, thereby leading to a higher prevalence of cardiovascular disease. Chiu C and Audrey in a study in 126 healthy participants showed a direct relation between insulin sensitivity and 25(OH)D level and that vitD deficiency has a negative effect on pancreatic β-cell function [12].

Low vitD levels are also found to be associated with an increased risk of HT and cardiovascular events in population-based studies [13-15]. Studies suggests that vitD supplementation may decrease blood pressure in patients with T2DM, although conflicting results have been reported [16]. Vit D had an inhibitory effect on the synthesis of renin, by converting angiotensin I to II, or on the regulation of inflammation in laboratory animals [17,18]. VitD improves endothelial function by reducing vascular inflammation, regulating blood pressure, inhibiting proliferation of vascular smooth muscle cells, and antagonizing the formation of foam cells [19]. Further, antioxidant property of vitD complements the protective mechanisms on heart and vasculature. Sugden et al in their study showed that a single large dose of vitD2 (100,000 IU orally) improved blood pressure and endothelial function, a key surrogate marker of cardiovascular risk, at 8 weeks post dose, in patients with T2DM and baseline 25OHD levels below 50 nmol/l [20].

Owing to these multifarious implications on health, the epidemic of vitD deficiency in India is likely to contribute significantly to the enormous burden on the healthcare system of India. Further, the increasing prevalence of T2DM and HT, either alone or in co-existence, in a populated country like India, increases the peril of devastating complications and eventual mortality. Hence the objective of this study was to evaluate the prevalence of vitD deficiency in patients with T2DM, HT and both T2DM and HT, and to understand the current management practices in Indian real-world setting. Association between vitD levels and hypothyroidism, if any, was also ascertained in this study. Early detection and treatment of vitD deficiency in these disorders is imperative to achieve the treatment goals. To the best of our knowledge, this is the first of its kind study to evaluate the trend of co-occurrence of vitD deficiency and the management practices in Indian patients.

Materials and methods

Study design and Patient population

This cross-sectional, clinico-epidemiological, multicentric, PAN – India study was conducted across 29 centers, spanning different geographical sites in India, between June to September 2017. Adults (≥18 years) with a diagnosis of T2DM and/or HT (established [who were already receiving antidiabetic/antihypertensive therapies] or newly diagnosed cases [fasting plasma glucose (FPG) ≥ 126 mg/dL or glycated hemoglobin (HbA1c) ≥ 6.5% for T2DM and blood pressure (BP) ≥ 140/90 mmHg for HT), visiting physician for routine check-up were enrolled in this study. Patients on a fixed dose combination of calcium and vitD supplement or with a history of using drugs that may interfere with vitD metabolisms (eg: carbamazepine, phenobarbital, sodium valporate, gabapentin, isoniazid, corticosteroids, mineral oil and calcitonin) were excluded from the study.

Three cohorts were formed in this study based on the indication, viz., T2DM, HT, and T2DM+HT. Each cohort contained approximately equal number of patients recruited in a ratio of 1:1:1.

The study protocol was approved by local independent ethics committees. The study was conducted in accordance with the principles of Declaration of Helsinki, International Conference on Harmonization Good Clinical Practice (ICH-GCP) guidelines, and Indian regulatory guidelines (Indian Council of Medical Research and Indian GCP guidelines). All patients provided written consent in the patient authorization form to participate in the study.

Data Collection

After obtaining necessary permissions from the hospital authorities, the patient data were collected from their hospital medical records. The patient data included demographics and anthropometric characteristics, medical/surgical/family history, lifestyle parameters, concomitant medications, physical examination, vital measurements, details of T2DM and HT, including duration of disease, medications (dose, regimen), latest glycemic indicator values (FPG, postprandial glucose [PPG], HbA1c). In addition to patient hospital records, the

blood sample was collected from each patient for serum 25(OH)D analysis.

Study Definitions:

VitD:

- VitD deficiency

  25 (OH) D: < 20 ng/mL
- VitD insufficiency

  25 (OH) D: 20–29 ng/mL
- VitD sufficiency

  25 (OH) D: ≥30 ng/mL

Hypothyroidism:

- Overt hypothyroidism

  TSH > 4.50 µIU/mL, fT4 < 0.8 ng/dL, and fT3 < 1.4 pg/mL.
- Subclinical hypothyroidism

  TSH > 4.50 µIU/mL, fT4 0.8-1.8 ng/dL, and fT3 1.4-4.4 pg/mL

Obesity and Overweight

- Overweight

  BMI: ≥ 23 and < 25 kg/m^2
- Obese

  BMI: ≥ 25 kg/m^2

Study Outcome

The primary outcome was the percentage of patients with vitD deficiency in those with T2DM, HT or T2DM and HT. Association between different categories of vitD levels (deficient, insufficient and sufficient) and hypothyroidism and the prevailing management practices, i.e: percentage of patients receiving vitD supplementation along with regimen (doses in IUs, frequency and recommended duration) were also assessed in this study.

Statistical Analysis

Considering the lack of data on the prevalence of vitD deficiency in patients with T2DM or HT or both T2DM and HT, approximately 1500 patients, including 500 patients of T2DM, HT and T2DM+HT each were enrolled in the study, assuming 10% drop-out rate. Continuous variables were summarized descriptively. Categorical data was presented as frequencies and percentages. The association between different categories of vitD levels and hypothyroidism was derived by chi-square test at 5% level of significance. All statistical analyses were performed using SAS® version 9.4 (SAS Institute Inc, USA).

Results

Initial data was collected from 1508 patients (T2DM: 504; HT: 501; T2DM+HT: 503). Of these, 1501 (99.5%) completed the study (T2DM:500 [99.2%]; HT:499 [99.6%]; both T2DM and HT: 502 [99.8%]). Seven patients (T2DM: 4; HT: 2; T2DM+HT: 1) discontinued the study by withdrawing their consent.

Baseline and Demographic Characteristics

The mean±SD age of the overall population was 52.9±12.49 years. The mean ± SD age of the patients with T2DM, HT and both T2DM and HT was 50.7±12.18, 51.6±13.58, and 56.4±10.82 years, respectively. In the overall population, 792 (52.5%) were women and 716 (47.5%) were men. Baseline and demographics characteristics of patients from all the 3 cohorts are shown in Table 1.

From the total cohort, 1007 patients were reported to have T2DM. The mean duration of T2DM was 8.3±6.90 years; mean age at the time of diagnosis was 45.3±10.31 years. The HT was reported in 1004 patients. The mean duration of HT was 6.8± 5.87 years; mean age at the time of diagnosis was 47.3±11.20 years. About 8.3% patients with T2DM and 5.9% patients with HT had complications due to their indications. The most common complication due to T2DM and HT were diabetic neuropathy (52.4%) and renal disease (52.5%), respectively.

Forty-two (2.8%) patients had known history of vitD deficiency (T2DM: 16 [3.2%]; HT: 12 [2.4%]; T2DM and HT: 14 [2.8%]). The mean duration of vitD deficiency was 1.9±4.46 years. The mean age at the diagnosis of vitD deficiency was 52.5±10.77 years. In total, the mean vitD level at the time of diagnosis was 16.9±12.78 ng/ml.

The serum 25 (OH)D levels reported from 24 patients at the time of diagnosis was 22.4±16.31 ng/ml in patients with T2DM (n=7), 18.8 ±13.10 ng/ml in patients with HT (n=8), and 10.9 ±7.03 ng/ml in patients with both T2DM and HT (n=9). Majority of the patients with known history of vitD deficiency were treated with cholecalciferol (T2DM: 16; HT: 12; T2DM+HT: 13); one patient with T2DM +HT was treated with calcitriol.

Prevalence of vitamin D deficiency

Overall prevalence of low vitD was found to be 83.7% (1257 out of 1501 patients; T2DM: 421 [84.2%]; HT: 412 [82.6%]; T2DM+HT: 424 [84.5%]). The remaining 244 patients (T2DM: 79; HT: 87; T2DM+HT: 78) had vitD sufficiency.

Already established versus newly diagnosed cases with low level of vitD in patients with T2DM, HT and combined condition of T2DM and HT are summarized in Table 2. The prevalence of patients with low level of vitD amongst newly diagnosed cases was 1231 (82.0%) compared to 26 (1.7%) in already diagnosed cases. Number of already and newly diagnosed cases with low level of vitD were comparable in each cohort (Table 2).

Vitamin D levels in patients with T2DM, HT and combined condition of T2DM and HT

The new and already diagnosed cases with different categories of vitD levels in patients with T2DM, HT, and T2DM+HT are summarized in Table 3. More than half (60.9%) of the patients were reported with vitD deficiency. The highest number of vitD deficient cases were from T2DM only category (64.0%), followed by combined T2DM and HT (61.6%) and HT only (57.1%). VitD insufficiency was highest in HT only category (25.5%), followed by combined condition of T2DM and HT (22.9%).

Amongst new cases, more than half of the patients (59.8%) reported vitD deficiency. In addition, 333 (22.2%) patients had insufficient level of vitD. VitD sufficiency was reported in only 15.2% patients. The highest number of patients with newly diagnosed vitD deficiency was reported in patient with T2DM (63.0%), followed by those with combined condition of T2DM and HT (60.2%) and HT only (56.3%).

Amongst old cases, only 16 (1.1%) patients reported vitD deficiency. Moreover, 10 (0.7%) and 16 (1.1%) patients had insufficient and sufficient level of vitD, respectively. Amongst cohorts, vitD deficiency was highest in those who had combined condition of T2DM and HT (1.4%), followed by T2DM only (1.0%) patients.

Table 1: Baseline and Demographics Characteristics

Parameter	T2DM (N=504)	HT (N=501)	T2DM and HT (N=503)	Overall (N=1508)
Age (Years), Mean (SD)	50.7 (12.18)	51.6 (13.58)	56.4 (10.82)	52.9 (12.49)
Gender, n (%)				
Women	240(47.62)	283(56.49)	269(53.48)	792(52.52)
Men	264(52.38)	218(43.51)	234(46.52)	716(47.48)
Education, n (%)				
Graduate or Post Graduate	233(46.23)	232(46.31)	220(43.74)	685(45.42)
High School Certificate	79(15.67)	87(17.37)	75(14.91)	241(15.98)
Intermediate or post high school diploma	119(23.61)	121(24.15)	117(23.26)	357(23.67)
Middle School Certificate	43(8.53)	31(6.19)	51(10.14)	125(8.29)
Primary School Certificate	30(5.95)	30(5.99)	40(7.95)	100(6.63)
BMI (Kg/m2), Mean (SD)	26.3 (4.24)	27.1 (4.60)	27.4 (4.54)	26.9 (4.49)
BMI Categorization, n (%)				
Normal	97(19.25)	73(14.57)	70(13.92)	240(15.92)
Obese	303(60.12)	333(66.47)	349(69.38)	985(65.32)
Overweight	88(17.46)	86(17.17)	78(15.51)	252(16.71)
Underweight	16(3.17)	9(1.80)	6(1.19)	31(2.06)

T2DM: Type-2 diabetes mellitus; HT: hypertension

Table 2: Low level of vitamin D in Patients with T2DM, HT and Both T2DM and HT (N=1501)

Vitamin D, n (%)[95% CI]*	T2DM (N=500)	HT (N=499)	Combined condition of T2DM and HT (N=502)	Overall (N=1501)
Total (New + Old)	421 (84.2%) [80.70:87.29]	412 (82.6%) [78.95:85.79]	424 (84.5%) [80.99:87.52]	1257 (83.7%) [81.78:85.58]
New Cases	411 (82.2%) [78.56:85.45]	406 (81.4%) [77.66:84.69]	414 (82.5%) [78.86:85.70]	1231 (82.0%) [82.41:86.20]
Old Cases	10 (2.0%) [0.96:3.65]	6 (1.2%) [0.44:2.60]	10 (2.0%) [0.96:3.63]	26 (1.7%) [45.64:76.43]

T2DM: Type-2 diabetes mellitus; HT: hypertension * Patients with low level of vitamin D (Serum 25(OH) D =<30 ng/ml) were considered.

Vitamin D and hypothyroidism

In this study, 76.9% of hypothyroid patients had low level of vitD (<30 ng/ml). Significant association was noted between different levels of vitD and hypothyroidism (p< 0.0001) (Table 4).

Vitamin D and glycemic indices

No significant association between different levels of vitD and FPG, PPG, or HbA1c in patients with T2DM and T2DM+HT was noted.

Out of 126 patients with low vitD levels and T2DM, 84.37% patients had abnormal HbA1c levels. In patients with T2DM, with vitD insufficiency or deficiency, higher number of patients had abnormal HbA1c value as compared to normal HbA1c values

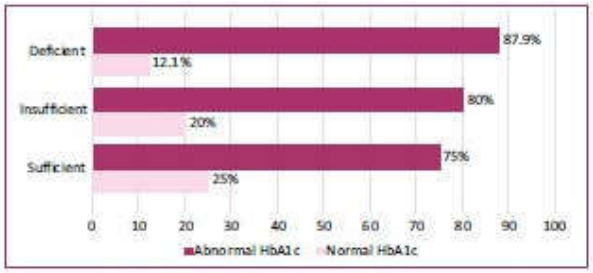

Fig. 1: Levels of Vitamin D and HbA1c in T2DM patients

Pharmacological Management for vitamin D deficiency

A total of 1066 out of 1257 (84.8%) patients with low level of vitD were prescribed with vitD supplements. Majority of patients (883 [70.2%]) received 60,000 IU dose of vitD. The preferred route of administration was oral (79.6%), once in a week (76.7%;). The mean duration of treatment was 7.6±3.49 weeks.

VitD deficiency (406 [26.9%]) and insufficiency (521 [34.5%]) levels, followed by symptoms of vitD deficiency (157 [10.4%]) were the major factors considered by physicians in prescribing vitD supplements to the patients.

Discussion

Research indicate that vitD deficiency is associated with an increased risk of developing T2DM and HT, thus leading to cardiovascular, and cerebrovascular diseases [21,22]. Majumdar et al recently reported that there is 3.13-fold increased risk of ischemic stroke associated with low vitD levels in patients with HT [21]. This rationale calls for routine evaluation of vitD deficiency in patients

Table 3: Vitamin D levels (deficient, insufficient and sufficient) in newly and previously diagnosed cases of T2DM, HT and combined condition of T2DM and HT

Vitamin D Levels, n(%) [95% CI] a,b,c	T2DM (N=500)	HT (N=499)	Combined condition of T2DM and HT (N=502)	Overall (N=1501)
Deficiency	320 (64.0%) [59.62:68.21]	285 (57.1%) [52.64:61.50]	309 (61.6%) [57.14:65.83]	914 (60.9%) [58.37:63.37]
Insufficiency	101 (20.2%) [16.77:23.99]	127 (25.5%) [21.68:29.51]	115 (22.9%) [19.30:26.84]	343 (22.9%) [20.75:25.06]
Sufficiency	79 (15.8%) [12.71:19.30]	87 (17.4%) [14.21:21.05]	78 (15.5%) [12.48:19.01]	244 (16.3%) [14.42:18.22]
New Cases				
Deficiency	315 (63.0%) [58.60:67.24]	281 (56.3%) [51.83:60.72]	302 (60.2%) [55.73:64.47]	898 (59.8%) [57.30:62.32]
Insufficiency	96 (19.2%) [15.84:22.93]	125 (25.1%) [21.31:29.09]	112 (22.3%) [18.74:26.21]	333 (22.2%) [20.11:24.37]
Sufficiency	73 (14.6%) [11.62:18.00]	81 (16.2%) [13.10:19.77]	74 (14.7%) [11.76:18.15]	228 (15.2%) [13.41:17.11]
Old Cases				
Deficiency	5 (1.0%) [0.33:2.32]	4 (0.8%) [0.22:2.04]	7 (1.4%) [0.56:2.85]	16 (1.1%) [0.61:1.73]
Insufficiency	5 (1.0%) [0.33:2.32]	2 (0.4%) [0.05:1.44]	3 (0.6%) [0.12:1.74]	10 (0.7%) [0.32:1.22]
Sufficiency	6 (1.2%) [0.44:2.59]	6 (1.2%) [0.44:2.60]	4 (0.8%) [0.22:2.03]	16 (1.1%) [0.61:1.73]

T2DM: Type-2 diabetes mellitus; HT: hypertension; aSufficiency: Serum 25(OH)D ≥ 30 ng/ml; bInsufficiency: Serum 25(OH)D between 21-29 ng/ml; cDeficiency: Serum 25(OH)D ≤ 20 ng/ml.

Table 4: Association between Different Categories of VitD Levels and Hypothyroidism

Vitamin D levels	Number of patients (%)	Hypothyroidism, number of patients (%)		P-value*
		Presence (n=436)	Absence (n=1065)	
Deficiency	913(60.8%)	224 (51.4%)	689 (64.7%)	< 0.0001
Insufficiency	343 (22.9%)	111 (25.5%)	232 (21.8%)	
Sufficiency	244(16.3%)	101 (23.2%)	143 (13.4%)	

T2DM: Type-2 diabetes mellitus; HT: hypertension; * calculated using chi square test

with T2DM and HT to tailor prevention strategies and early treatment. Further, the growing burden of T2DM and HT and the increasing prevalence of low level of vitD has apprehended the attention of health care practitioners, particularly in countries with co-occurrence of T2DM and HT.

Even though vitD deficiency prevails in epidemic proportions all over the Indian subcontinent with a prevalence range of 70% to 100% in general population, [23] very little information exists about its prevalence and their clinical practice management among patients with T2DM, HT, and those with co-occurrence of T2DM and HT in India. Our study is the first of its kind attempt to provide critical insights into the disease burden of vitD deficiency, its association with hypothyroidism and their management strategy in the cohort of T2DM, HT, and both T2DM+HT in India.

In this cross-sectional study, the overall prevalence of patients with low vitD levels (deficiency and insufficiency) was 1257 (83.7%). Out of this, 1231 (82%) were newly diagnosed cases. This high prevalence of vitD deficiency in newly diagnosed cases indicates that vitD deficiency may have been missed in a large proportion of patients with T2DM, HT, or both T2DM and HT. The proportion of patients with low vitD in patients with T2DM, HT and both T2DM + HT was found to be 84.2%, 82.6% and 84.5%, respectively. Our results were in agreement to the prevalence reported in other studies from India. A recent study among T2DM patients reported 71.4% patients with vitD deficiency and 15% with insufficiency in South India region [22]. Among patients with low levels of vitD, deficiency was observed in nearly two-third (60.9%) of patients, while insufficiency was noted in remaining (22.9%) patients. Sheth et al observed vitD deficiency in 91.4% of cases of T2DM and 93% in the control group. However, they could not establish any association between vitD deficiency and glycated haemoglobin [24]. Kumar et al in a study from Pondicherry observed vitD deficiency in 32% of cases with T2DM and 25% of controls. Further, low VitD levels were observed in 66.5% of the study population (including cases and controls) [25]. In another study by Kumar H et al, the prevalence vitD deficiency among patients with T2DM from Rajasthan was found to be 91.1% [26]. On the other hand, the prevalence of vitD deficiency in hypertensive patients from North India was found to be 80.4% [27].

This study reported a significant association between levels of vitD and hypothyroidism ($p<0.0001$). In agreement with this observation, Talaei et al. (2017) also reported that the lack of vitD contributed to the possibility of low thyroid hormones [28]. Additionally, a recent cross-sectional study correlated the deficiency of vitD with increased levels of TSH in hypothyroidism patients [29]. This could be attributed to the variation in the vitD receptor gene, which are thought to mediate susceptibility to various endocrinal autoimmune diseases.[30,31] Further, it has also been proposed that the decrease in the level of vitD in hyperthyroid patients could result in increased level of calcium, resulting in a negative impact on the secretion of parathyroid hormone and vitD synthesis [32].

In this study, a non-significant inverse correlation was observed between different levels of vitD and HbA1c in patients with T2DM and T2DM+HT (T2DM: $r=-0.10$, $p=0.21$; T2DM+HT: $r=-0.10$, $p=0.23$). Similar results were noted by Calvo-Romero and Ramiro-Lozano (2015), where inverse correlation between serum levels of 25(OH) D and glycosylated hemoglobin ($r = -0.74$, $P = 0.01$) were reported [33]. Another study demonstrated similar negative correlation association between vitD level and HbA1c ($P = 0.035$) in patients with diabetes [34]. Brijesh et al. observed 25 (OH)D levels of 23.63±3.71 ng/ml in patients with T2DM with HbA1c levels less than 7 g% and 25OH vitD levels of 19.41±4.76 ng/ml in patients with T2DM with HBA1c levels more than 7 g% [35]. This implies that supplementation of vitD could be effective in improving glycemic control in patients with T2DM with deficient or insufficient levels of vitD.

A total of 84.8% patients with low level of vitD were prescribed with vitD supplements. Most of the patients in our study were prescribed a dose of 60000 IU (70.2%) via oral route (79.6%) once in a week (76.7%). Physicians often prescribe 1500mg (60 000 IU) cholecalciferol per week for 8 weeks for vitD deficiency in India [36]. Nevertheless, data on maintenance therapy of vitD supplements to prevent recurrence of vitD deficiency, after treatment of acute deficiency, is lacking. Pietras SM et al showed that 50,000 IU of ergocalciferol weekly for 8 weeks is effective in treating vitD deficiency, and continued treatment with 50,000 IU of ergocalciferol every alternate week for up to 6 years prevents recurrent vitD deficiency in most patients [37]. However, commercially, ergocalciferol (50,000IU) preparation is not available in India. Hence, 60,000 IU cholecalciferol every alternate week for up to 6 years is an effective maintenance therapy, after treatment of acute deficiency, to prevent vitD deficiency in Indian patients.

Our study has strengths and limitations. The strengths being that this is the first study highlighting the burden of vitD deficiency in patients with co-occurrence of T2DM and HT. The results can be generalized as it is a Pan-India study covering a large number of patients across the different geographical region and provided critical insights into the disease burden of vitD deficiency in patients with T2DM and/or HT in India. One of the limitation of this study is that no control group was included to see the viability of results in studied population. Nevertheless, this is the first of its kind nationwide data on the prevalence of vitD deficiency and its association with hypothyroidism in patients with T2DM and/or HT. However, a longitudinal study is warranted to ascertain the long-term association between vitD deficiency and T2DM and/or HT and to assess its impact on disease progression. Additionally, randomized controlled trials are required to provide an insight into the efficacy and safety of vitD as a therapeutic tool for vitD deficiency in such patients.

Conclusion

A high prevalence of vitD deficiency was found in patients with T2DM, HT and T2DM+HT in India. Prevalence of vitD deficiency was higher amongst newly diagnosed cases compared to already diagnosed cases (in all three cohorts [T2DM/HT/T2DM and HT). This could indicate that vitD deficiency may have been missed in a large proportion of patients with T2DM, HT, or both T2DM and HT. The low level of vitD and its association with hypothyroidism was also evident from our findings. This study thus underscores the magnitude of overlap between vitD deficiency and T2DM or HT, and the need for routine vitD screening for early diagnosis and effective management.

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