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Clinical Observation of Lichen Based Vitamin D_3 (60000IU) Pill in Normal Healthy Peoples, Formulated by Ambrosiya Neo-Medicine Pvt. Ltd

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ABSTRACT

In the early 20th century in the United States, vitamin D fortification of food products was essential to the eradication of rickets. Since then, there has been a nearly 100-year period of building evidence connecting vitamin D insufficiency to a number of outcomes, which has coincided with growing public interest in and knowledge of the health advantages of vitamin D. Vitamin D supplements are now widely available in both developed and developing nations, and many of them are supplied to the general public in the form of unregulated formulations with minimal information about safe administration. Together, these factors have caused a shift in which incidences of vitamin D intoxication have dramatically increased across the globe. Vitamin D toxicity is characterised by serum 25-hydroxyvitamin D [25(OH)2D] values greater than 150 ng/ml (375 nmol/l) due to vitamin D overdose. Clinicians now have to manage an illness that might manifest as anything from asymptomatic problems to severe, life-threatening ones. A study of lichen-based vitamin D3 pills has been performed and analysed by different tests.

Keywords: *Vitamin D_3; Unregulated formulation; Intoxication; Lichen-based pill; Ambrosiya Neo-Medicine Pvt. Ltd*

Abbreviations

VDT: Vitamin D toxicity; PTH: Parathyroid hormone, IIH: Idiopathic Infantile Hypercalcemia; VDBP: Vitamin D binding protein; GS: Glucocorticoid therapy; CT: Calcitonin; BS: Bisphosphonates; VDR: Vitamin D receptor; PCV: Packed Cell Volume; MCV: Mean Corpuscular Volume; MCH: Mean Corpuscular Hemoglobin; MCH.C: Mean Corpuscular Hemoglobin Concentration.

Introduction

There is growing evidence that the steroid hormone vitamin D has positive effects that go much beyond bone health. As a result, over the past ten years, this molecule has attracted a lot of study attention, especially when contrasted with the generally stable research output related to other vitamins. The high frequency of vitamin D insufficiency is well known in Europe, but it is actually a global issue, with Middle Eastern female adolescents being particularly at risk [1, 2]. The considerable growth in the usage of vitamin D therapy may have been brought on by this as well as the positive effects of treatment on tissues other than bone. Similar to this, population-based recommendations and counsel from chief medical officers have further backed the widespread use of vitamin D supplements, with an intake of 400 IU per day suggested in the UK for anyone 4 years of age and older. Vitamin D toxicity is a potentially dangerous side effect of treatment, so vitamin D therapy is not without risk. Because obesity is a major risk factor for vitamin D insufficiency, it is possible that vitamin D deficiency will increase as obesity rates grow [3,4].

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This has led to a rise in the use of vitamin D formulations for therapy, as well as greater awareness of vitamin D deficiency in the general public on a global scale. But there has also been a rise in vitamin D therapy use; reports of vitamin D toxicity have significantly increased, with the bulk (75%) of these reports appearing since 2010. Many of these cases are the result of improper prescribing, the use of unlicensed or high-dose over-the-counter medications, and inappropriate prescribing. The most often observed clinical signs of vitamin D toxicity (VDT), also known as vitamin D intoxication or hypervitaminosis D, are confusion, apathy, repeated vomiting, stomach pain, polyuria, polydipsia, and dehydration. The causes of VDT and its clinical symptom, severe hypercalcemia, include excessive long-term vitamin D use, problems with the vitamin D metabolic pathway, or the presence of a concurrent illness that locally develops the active vitamin D metabolite [5].

Although VDT is uncommon, if it is not properly diagnosed, major health consequences may result. Exogenous (iatrogenic) and endogenous VDT come in a variety of forms. Exogenous VDT, which is linked to hypercalcemia, is typically brought on by the unintentional or incorrect consumption of excessively high dosages of pharmaceutical formulations of vitamin D. VDT is characterised by serum 25-hydroxyvitamin D [$25(OH)_2D$] values greater than 150 ng/ml (375 nmol/l) due to vitamin D overdose. Endogenous VDT can arise from either diminished or excessive synthesis of the active vitamin D metabolite 1,25(OH)2D in idiopathic infantile hypercalcemia, granulomatous diseases, and some lymphomas. Exogenous VDT in healthy people is typically brought on by extended usage of vitamin D megadoses (months), not by excessively high skin exposure to the sun or by eating a varied diet. The amount of pre-vitamin D (tachysterol and lumisterol) produced in the skin by ultraviolet-B radiation can be controlled by the human body. Vitamin D is not normally found in high concentrations in a varied diet, and little vitamin D is added to food products. Exogenous VDT caused by excessive vitamin D is identified by considerably increased 1, 25(OH)2D concentrations (>150 ng/ml), severe hypercalcemia, and very low or undetectable parathyroid hormone (PTH) activity [6-8].

The first measurable symptom of VDT is hypercalciuria. When an elevated quantity of calcium in serum inhibits PTH action, the 1, 25 $(OH)_2D$ concentrations in individuals with VDT may be within the reference range, slightly elevated, or lowered (less commonly). Through the suppression of 1-hydroxylase activity and the stimulation of 24-hydroxylase activity, 1,25(OH)2D is downregulated. Patients with lymphomas, granuloma-forming diseases, and idiopathic infantile hypercalcemia (IIH) are all at higher clinical risk for endogenous VDT. Patients with those illnesses are hypersensitive to vitamin D, and elevated 1,25 $(OH)_2D$ concentrations with hypercalcemia may appear after taking vitamin D supplements, consuming foods with higher vitamin D contents, or even after excessive sun exposure [9,10].

Despite the fact that both 25(OH)D and 1,25$(OH)_2D$ concentration readings in that disease may be either normal or raised, and the pathophysiological rationale is sometimes ambiguous, patients with Williams-Beuren syndrome still need to be monitored for vitamin D hypersensitivity. Endogenous VDT is linked to aberrant extra renal synthesis of 1,25$(OH)_2D$ by activated macrophages in granulomatous disorders like sarcoidosis, TB, leprosy, fungal illnesses, infantile subcutaneous fat necrosis, giant cell polymyositis, and berylliosis. [11].

Diagnostic cut-off points for 25-hydroxy-cholecalciferol (Vitamin D_3) concentrations

Table 1 indicates diagnostic cut-off points for 25-hydroxy-cholecalciferol (Vitamin D_3) concentrations [1].

Table 1: Diagnostic cutoff.

Category	nmol/L	µg/L
Deficiency	<50	<20
Insufficiency	51-74	21-29
Sufficient	>75	>30
Excess	>250	>100
Intoxication	>375	>150

Symptoms of Vitamin D toxicity

Symptoms of Vitamin D toxicity includes confusion, apathy, repeated vomiting, stomach pain, polyuria, polydipsia and dehydration.

VDT explains by three hypotheses.

1. Increased levels of the active hormonal form, 1, 25(OH)2D, in the serum, which result in an increase in its intracellular concentration, mediate toxicity. That claim lacks substantial evidence. Selby et al. (12), the only study to do so, found higher 1, 25$(OH)_2$D concentrations at VDT. Numerous other investigations showed that 1,25(OH)2D levels were either normal or barely raised [12].

2. 1,25 $(OH)_2$D due to its high affinity for VDRs and low affinity for the vitamin D binding protein (VDBP), is a crucial ligand with access to the transcriptional signal transduction apparatus. Because the binding capacity of VDBP is saturated in hypervitaminosis D, other vitamin D metabolites can reach the cell nucleus as a result of the considerably elevated amounts of other vitamin D metabolites, including 25(OH)D. With the greatest affinity for VDRs of all the vitamin D metabolites (a dose-dependent impact), 25(OH)D has the ability to induce transcription on its own at high blood concentrations [13].

3. Consuming vitamin D boosts levels of the vitamin itself as well as numerous other vitamin D metabolites, particularly 25(OH)D. Vitamin D metabolites such as 25(OH)D, 24,25$(OH)_2$D, 25(OH)2D, 25, 26(OH)2D, and 25(OH)D-26,23-lactone have significantly higher amounts in hypervitaminosis D. When vitamin D metabolite concentrations are abnormally high, they can no longer be bound by the VDBP, which results in the release of free 1,25$(OH)_2$D. This active metabolite then diffuses into the target cells and acts via the VDR [14].

Treatment of VDT consists of first- and the second-line treatment strategies.

First-line treatment

Discontinuing vitamin D supplements and lowering calcium consumption from food. It is advised that people with granulomatous illnesses, lymphomas, and IIH stay out of the sun as well as other ultraviolet-B rays [9].

To treat dehydration and restore kidney function, an isotonic sodium chloride solution should be administered. Once the volume has been regained and maintained, loop diuretics can be introduced. Replacing lost sodium, potassium, and chloride is crucial in situations of prolonged sodium chloride and loop diuretic therapy [15].

By limiting intestinal calcium absorption, slowing down transcellular active transport mechanisms, and boosting urine calcium excretion, glucocorticoid therapy (GS) lowers plasma calcium levels. Additionally, GS treatment modifies the hepatic metabolism of vitamin D to favour the synthesis of inactive metabolites.

In serious circumstances when hypercalcemia is brought on by a rise in the breakdown of osteoclastic bones as a result of 1,25(OH)2D's direct impact on bone tissue, antiresorptive therapy using calcitonin (CT), bisphosphonates (BS), or both may be beneficial. Responses to CT and BS are significantly dissimilar. CT acts quickly, but tachyphylaxis takes a few days to manifest. BS takes action after a few days, but the impact lasts for a while [16].

Second-line treatment

By inducing the hepatic microsomal enzyme, phenobarbital can reduce 25(OH)D concentrations and be an effective treatment for VDT [17].

Ketoconazole inhibits cytochrome P450, CYP27B1, which reduces the generation of 1,25(OH)2D by activated mononuclear cells, but long-term use is not advised because it blocks numerous other significant CYPs [18].

In granulomatous disorders, aminoquinolines (chloroquine, hydrochloroquine) reduce 1,25(OH)2D generation by activated mononuclear cells via an unidentified mechanism [19].

It has been discovered that particular inhibitors of CYP27B1 (1-hydroxylase) could be useful for selectively preventing the formation of 1, 25(OH)2 D without interfering with other cytochrome P450-containing enzymes [20].

Drugs like rifampin induce non-specific liver cytochrome P450 enzymes, particularly CYP3A4, which leads to an alternate catabolic destination for vitamin D metabolites from the 24-hydroxylation pathway and permits the non-specific degradation of abundant 1,25(OH)2D in individuals with IIH [21].

The most effective, economical, and environmentally friendly type of vitamin D3 is lichen-based vitamin D3. The simplest to use and vegan, vegetarian and all-natural vitamin D3 supplement is this one. A vegan source of vitamin D3, lichen-based vitamin D3 is made from lichens, an algal and fungal mixture. Patients with

vitamin D insufficiency received 60,000 IU of chemical-based vitamin D3 extract, which is equivalent to 2.6 grams of lichen-based vitamin D3 extract and contains 60,000 IU of vitamin D3. This makes it the most reasonably priced, potent, and absorbable form of vitamin D3.

Pathological report

Dr. Jain's Precision Diagnostic Centre conducted a study. Ten healthy people (five males and five females) of the age group 25–45 years were considered for the study. Out of ten patients, five were male (P1-P5) and five were female (P6-P10). Blood Sugar (F), Haematology Routine, Liver Function Test, Lipid Profile Test, Urine Examination, and Immunoassay have all been performed. The reports of the subject were generated before and after the lichen-based vitamin D_3 (60000 IU). Lichen-based vitamin D_3 was given to all people, and then after 48 hours, samples were analysed. All test reports are presented in the XLS sheet. The general symptoms of excess lichen-based vitamin D_3 in all people have been observed (Tabe 1 to 6).

Discussion

Lichen-based vitamin D_3 enhances the metabolism of glucose. In the study, it was observed that there was a marginal reduction in glucose level. Several researchers have reported the effects of vitamin D3 on glucose metabolism and hemostasis [22, 23]. After taking the lichen-based vitamin D3, there are no significant effects on the haematological report, but a little bit enhances the level of haemoglobin and DLC [24, 25]. The vitamin D receptor (VDR) in the liver is influenced by lichen-based vitamin D_3. Due to VDR's greater expression in chronic liver illnesses, which is naturally present in the liver cells (Benetti et al., 2018), inflammation can be reduced. Additionally, vitamin D has proliferative, anti-inflammatory, and anti-fibrotic actions on the liver. According to some studies on the effects of vitamin D on anthropometric and biochemical indices, vitamin D was linked to body weight, fasting blood sugar, insulin resistance, glucose homeostasis, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, triglycerides, total cholesterol, liver enzymes, and adiponectin. [26-28]. Supplementing with lichen-based vitamin D_3 seemed to lower serum levels of total cholesterol, LDL cholesterol, and triglycerides, but not HDL cholesterol. Patients with high cholesterol levels who are at high risk for cardiovascular illnesses and have vitamin D insufficiency may benefit from vitamin D supplements [29, 30]. The findings highlighted the necessity for testing and lichen-based vitamin D_3 treatment in people with subclinical hypothyroidism by showing that taking vitamin D supplements significantly reduced TSH mean levels [31-40] (Figure 1).

Table 2: Biochemistry Test.

Blood Sugar (F) Fluoride Plasma, GOD-POD method		
Without Vitamin D3	P1	92 mg/dl
	P2	89 mg/dl
	P3	85 mg/dl
	P4	83 mg/dl
	P5	91 mg/dl
	P6	80 mg/dl
	P7	85 mg/dl
	P8	79 mg/dl
	P9	89 mg/dl
	P10	93 mg/dl
Normal Range (70.00-110.00)		
With Lichen Based Vitamin D3, test after 48 hrs.	P1	89 mg/dl
	P2	87 mg/dl
	P3	82 mg/dl
	P4	81 mg/dl
	P5	89 mg/dl
	P6	77 mg/dl
	P7	81 mg/dl
	P8	75 mg/dl
	P9	85 mg/dl
	P10	89 mg/dl

Table 3: Hematology Routine.

		Complete Blood Count		Differential Leucocyte Count (DLC)											
		Haemoglobin (Gm%) (13.50-18.50)	Total Leucocyte Count (Cell/Cumm) (4,000-11,000)	Polymorph (40.00-75.00%)	Lymphocytes (20.00-45.00%)	Lymphocytes (01.00-06.00%)	Monocytes (02.00-10.00%)	Basophils (0.00-1.00%)	P.C.V. (Pcv/Hct) (40-54%)	M.C.V. (80.00-96.00 Fl)	M.C.H (27.00-32.00 Pg)	M.C.H.C. (30.00-35.00%)	Red Blood Cells (4.60-6.00 Million)	Platelet Count (1.50-4.50l/Cumm)	E.S.R. (Westergren) (EDTA Whole Blood) (00--09mm/L)
Without Vitamin D3	P1	13.85	7695	65	23	2.8	3.2	0.2	41.3	88.3	28.3	31.2	4.86	3.12	8.3
	P2	14.6	8800	59	25	2.7	3.4	0.3	42.2	92.3	28.5	32.5	5.23	2.89	7.8
	P3	15.2	9385	63	30	3.8	3.9	0.5	44.2	91.5	29.3	33.5	5.86	3.75	6.5
	P4	13.69	7860	64	35	4.2	4.5	0.7	43.2	89.5	30.5	31.8	4.98	3.96	8.9
	P5	15.65	9900	68	34	4.5	4.8	0.6	48.2	92.5	31.6	32.5	5.12	3.85	7.6
	P6	14.2	8600	66	26	3	2	0.2	42.5	86.4	29.2	33.8	4.38	2.17	5.6
	P7	14.8	9600	58	29	2.5	2.8	0.8	41.8	88.4	30.2	34.8	4.96	2.95	4.5
	P8	14.4	10600	63	31	2.9	3.6	0.4	47.2	89.6	31.2	32.8	5.25	3.17	8.6
	P9	13.8	7600	69	32	3.7	3.9	0.5	42.6	90.6	31.6	33.1	4.47	2.75	7.5
	P10	15.2	8400	51	33	4.1	4.2	0.6	43.8	89.6	30.8	32.3	4.78	3.45	7.56
With Lichen Based Vitamin D3, test after 48 hrs.	P1	14.1	8300	62	23	2.8	2.1	0.2	37.5	84.65	31.2	32.5	4.38	2.48	12.5
	P2	14.7	9200	54	28	3.2	2.8	0.4	38.6	85.5	30.2	33.2	5.15	2.75	11.23
	P3	15	8700	61	29	3.1	3.2	0.3	41.3	87.5	30.4	31.6	4.48	2.65	11.45
	P4	13.55	9150	52	30	2.9	3.8	0.4	45.6	89.4	32.1	32.8	4.85	2.95	10.23
	P5	15.5	8650	61	32	3.4	3.5	0.3	41.3	85.3	31.2	32.4	4.56	3.12	11.56
	P6	15.5	8800	65	24	2.9	2	0.1	38.8	86.8	29.4	33.6	4.34	2.19	13.5
	P7	14.9	9900	55	27	2.6	2.9	0.7	42.4	88.9	30.3	34.9	4.91	2.89	10.58
	P8	14.6	9800	61	29	2.8	3.8	0.5	47.8	89.9	31.4	32.1	5.05	3.11	10.98
	P9	13.8	8000	67	28	3.6	4.1	0.6	41.2	90.8	31.4	33.3	4.41	2.71	11.23
	P10	15.1	8600	49	31	3.9	4.2	0.7	42.2	89.9	30.6	32.6	4.71	3.39	11.45

Table 4: Liver Function Test.

		S. Bilirubin (Total) (Serum Jendrassik & Grof Method) (0.00-1.00 mg/dl)	S. Bilirubin (Direct) (Serum Jendrassik & Grof Method) (UP to-00.30mg/dl)	S. Bilirubin (Indirect) (Serum, Calculated) (00-00.70 mg/dl)	S.G.O.T. {Serum, IFCC Method, Kinetic) (00.00-40.00IU/L)	S.G.P.T. {Serum, IFCC Method, Kinetic) (00.00-40.00 IU/L)	Alkaline Phosphatase (Serum IFCC Method, Kinetic)	Total Proteins Serum, Biuret Method) (6.50-8.50mg/dl)	Albumin (Serum, BCG Method) (3.50-5.50 mg/dl)	Globulin (Serum, Calculated) (2.30-3.50 mg/dl)	A:G Ratio (Serum, Calculated)
Without Vitamin D3	P1	0.64	0.12	0.52	22.98	24.18	78.77	7.46	4.35	3.11	1.3:1
	P2	0.61	0.14	0.54	23.98	27.45	78.77	7.75	4.15	3.01	1.3:1
	P3	0.58	0.16	0.56	28.75	30.28	78.77	6.46	3.85	2.65	1.4:1
	P4	0.69	0.17	0.58	27.65	29.88	78.77	7.66	4.78	3.45	1.3:1
	P5	0.56	0.19	0.59	31.45	33.18	78.77	6.86	4.98	3.12	1.4:1
	P6	0.71	0.11	0.61	33.41	26.18	78.77	7.81	4.15	2.98	1.4:1
	P7	0.73	0.17	0.62	23.65	24.88	78.77	6.95	4.25	2.88	1.4:1
	P8	0.72	0.22	0.45	35.67	25.18	78.77	6.32	4.17	3.01	1.3:1
	P9	0.48	0.18	0.53	27.65	28.78	78.77	7.16	3.98	3.24	1.2:1
	P10	0.52	0.23	0.47	36.68	26.78	78.77	7.02	4.89	3.35	1.4:1
With Lichen Based Vitamin D3, test after 48 hrs.	P1	0.62	0.14	0.53	23.18	24.78	78.98	7.66	4.25	3.21	1.3:1
	P2	0.62	0.16	0.56	23.78	27.35	78.69	7.65	1.25	3.12	1.3:1
	P3	0.59	0.18	0.58	28.55	30.48	77.77	6.36	3.75	2.55	1.4:1
	P4	0.67	0.19	0.61	27.68	29.78	78.46	7.36	4.38	3.35	1.3:1
	P5	0.53	0.21	0.62	31.65	32.18	78.17	6.76	4.88	3.32	1.4:1
	P6	0.73	0.14	0.64	33.64	26.25	78.98	7.61	4.05	2.78	1.4:1
	P7	0.71	0.15	0.58	23.45	24.68	78.57	6.75	4.15	2.98	1.4:1
	P8	0.74	0.24	0.47	35.57	25.28	78.67	6.12	4.27	3.11	1.2:1
	P9	0.46	0.21	0.55	27.45	28.58	78.67	7.26	3.88	3.14	1.2:1
	P10	0.54	0.24	0.48	36.61	26.68	78.57	6.92	4.69	3.25	1.4:1

Table 5: Lipid Profile Test.

		Total Lipids (Serum, Calculated) (400.00-700.00 mg/dl)	Serum Cholesterol (Serum, CHOD-PAP method (With LCF) (0.00-200.00 mg/dl)	HDL Cholesterol (Serum, Cholesterol esterase, oxidase method) (35.00-80.00 mg/dl)	LDL Cholesterol (Serum, Calculated) (100.00-129.00 mg/dl)	VLDL Cholesterol (Serum, Calculated) (20.00-40.00 mg/dl)	S. Triglycerides (Serum GPO - Trinder Method) (0.00-160.00 mg/dl)
Without Vitamin D3	P1	512	132	56	122	32	110
	P2	552	144	52	118	28	123
	P3	492	152	48	121	23	142
	P4	612	112	58	105	37	145
	P5	598	138	61	109	29	127
	P6	635	145	67	111	32	134
	P7	582	165	63	116	21	126
	P8	634	137	69	106	31	141
	P9	495	156	71	102	33	114
	P10	568	162	67	113	32	147
With Lichen Based Vitamin D3, test after 48 hrs.	P1	568	162	58	125	61	340
	P2	572	178	56	123	56	345
	P3	526	192	52	123	56	345
	P4	668	156	59	110	65	362
	P5	645	156	59	110	65	356
	P6	675	185	68	115	64	645
	P7	680	195	65	123	54	342
	P8	750	198	72	115	62	341
	P9	585	175	73	110	65	320
	P10	645	185	69	117	63	310

Table 6: Urine Examination.

		Routine Urine Analysis (20ML)						Microscopic Examination					
		Colour	Appearance	Spec. Gravity	Reaction	Albumin	Sugar	Pus Cells (1-2 /Hpf)	Rbc Cells	Ep Cells (1-2 /Hpf)	Casts	Crystals	Others
Without Vitamin D3	P1	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.2	NIL	1.42	NIL	NIL	NIL
	P2	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.25	NIL	1.46	NIL	NIL	NIL
	P3	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.32	NIL	1.48	NIL	NIL	NIL
	P4	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.28	NIL	1.41	NIL	NIL	NIL
	P5	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.35	NIL	1.38	NIL	NIL	NIL
	P6	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.36	NIL	1.42	NIL	NIL	NIL
	P7	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.29	NIL	1.34	NIL	NIL	NIL
	P8	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.33	NIL	1.38	NIL	NIL	NIL
	P9	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.18	NIL	1.31	NIL	NIL	NIL
	P10	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.24	NIL	1.31	NIL	NIL	NIL
With Lichen Based Vitamin D3, test after 48 hrs.	P1	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.22	NIL	1.39	NIL	NIL	NIL
	P2	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.26	NIL	1.48	NIL	NIL	NIL
	P3	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.35	NIL	1.51	NIL	NIL	NIL
	P4	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.32	NIL	1.43	NIL	NIL	NIL
	P5	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.31	NIL	1.42	NIL	NIL	NIL
	P6	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.29	NIL	1.44	NIL	NIL	NIL
	P7	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.31	NIL	1.43	NIL	NIL	NIL
	P8	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.32	NIL	1.39	NIL	NIL	NIL
	P9	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.23	NIL	1.42	NIL	NIL	NIL
	P10	YELLOW	CLEAR	Q.N.S	ACIDIC	NIL	NIL	1.29	NIL	1.43	NIL	NIL	NIL

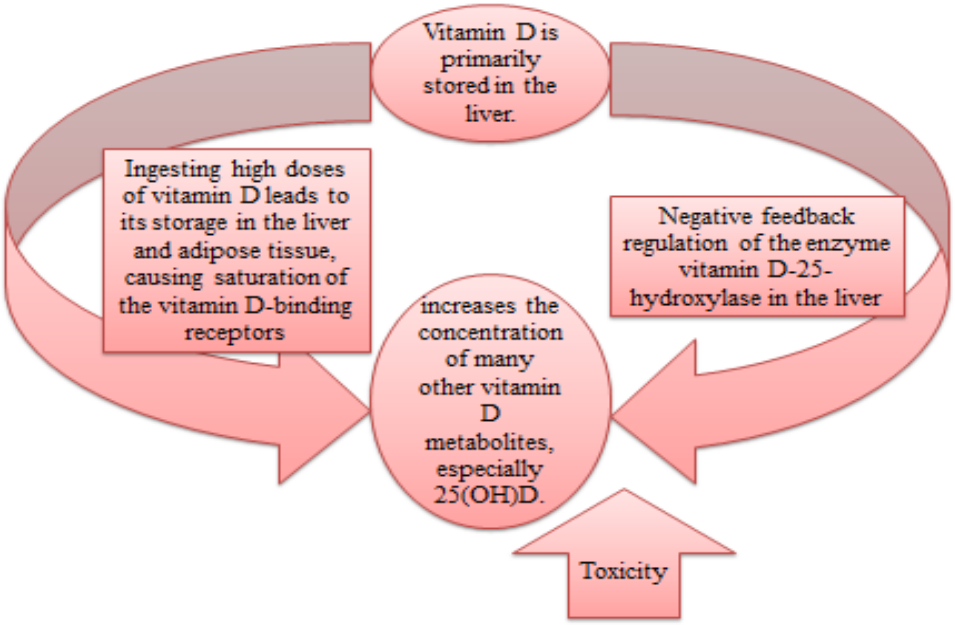

Figure 1: **Vitamin D toxicity.**

Table 7: **Immunoassay (Thyroid Stimulating Hormone).**

		Range (0.35-5.50u IU/mL)
Without Vitamin D3	P1	3.32
	P2	3.42
	P3	4.12
	P4	4.13
	P5	3.65
	P6	3.68
	P7	3.98
	P8	4.15
	P9	1.96
	P10	2.35
With Lichen Based Vitamin D3, test after 48 hrs.	P1	3.13
	P2	3.32
	P3	4.02
	P4	4.01
	P5	3.54
	P6	3.57
	P7	3.65
	P8	4.06
	P9	1.92
	P10	2.29

Conclusion

As a result of increased awareness and the common distribution of vitamin D, poisoning is still a problem and is anticipated to become more prevalent. Both the younger and older age groups are likely to have individuals who are more vulnerable. Simple laws aimed at guaranteeing the integrity of all vitamin D products, together with the restricted use of very high-dose formulations, may considerably reduce potential adverse effects because the majority of cases appear to have been simply preventable in hindsight.

Even though VDT with hypercalcemia is uncommon, it can be fatal if not properly recognised. Exogenous (iatrogenic) and endogenous VDT come in a variety of forms.

Exogenous VDT is most frequently caused by accidental overdoses from the use of pharmaceuticals. Intoxication is relatively uncommon, according to a review of VDT instances brought on by formulation or administration mistakes with vitamin D that led to a high dosage. However, individuals with a high level of VDT should always be taken into consideration as an alternative diagnosis.

Endogenous VDT is a significant clinical concern in several clinical situations. In granulomatous disorders, such as sarcoidosis and tuberculosis, or in lymphomas, ectopic synthesis of 1, 25$(OH)_2$D may lead to endogenous aetiologies. Numerous theories have been put forth by researchers to explain VDT, including the inhibition of 24-hydroxylase activity or increased activity of 1-hydroxylase, both of which would raise the concentration of the active vitamin D metabolite, the growth of VDRs, or the saturation of VDBP's capacity.

Although the general public is becoming more aware of the health advantages associated with vitamin D, the increased use of vitamin D-containing supplements could put them at risk for VDT. Therefore, those who self-administer vitamin D at doses greater than those prescribed for their age and body weight without medical supervision are encouraged to use caution.

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Conflict of Interest

The author declares no conflict of interest.

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