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Case Report

CASE PRESENTATION ON VITAMIN D DEFICIENCY-INDUCED FATTY LIVER DISEASE IN A 36-YEAR-OLD MALE

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ABSTRACT

Vitamin D deficiency has emerged as a significant contributing factor to the development of non-alcoholic fatty liver disease (NAFLD), a prevalent condition associated with metabolic dysfunction. This case presentation focuses on a 36-year-old male patient diagnosed with NAFLD likely induced by Vitamin D deficiency. The patient presented with symptoms of fatigue, right upper quadrant abdominal discomfort, and unexplained weight gain over the past year. Laboratory tests revealed elevated liver enzymes and a severely low Vitamin D level of 12 ng/mL. Imaging studies, including abdominal ultrasound, confirmed mild hepatic steatosis with grade 2 fatty liver. The patient's history of limited sun exposure and sedentary lifestyle were contributing factors to the deficiency. Management included Vitamin D supplementation, dietary changes, and regular physical activity. This case emphasizes the role of Vitamin D deficiency in the pathogenesis of NAFLD and underscores the importance of addressing this deficiency as part of a comprehensive treatment plan for improving liver health and preventing further progression.

Keywords: Vitamin D deficiency, Non-alcoholic fatty liver disease, Hepatic steatosis, Sedentary lifestyle.

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INTRODUCTION

Vitamin D deficiency has been increasingly recognized as a significant contributor to the development and progression of various metabolic diseases, including non-alcoholic fatty liver disease (NAFLD). NAFLD encompasses a spectrum of liver conditions, ranging from simple steatosis to more severe forms, such as non-alcoholic steatohepatitis (NASH) and cirrhosis. The patient is an office worker with a sedentary lifestyle of mostly at the desk work for about 6 h in a day. The patient has a history of mild obesity, which has been managed with diet and occasional exercise. No history of alcohol abuse or viral hepatitis. No significant family history of liver disease [1]. Vitamin D deficiency has been implicated in the development and progression of NAFLD through several interconnected mechanisms, including disruptions in lipid metabolism, insulin resistance, inflammation, and fibrogenesis. NAFLD is a growing global health concern characterized by excessive fat accumulation in the liver in individuals who consume little or no alcohol. It spans a spectrum from simple hepatic steatosis to NASH, fibrosis, and cirrhosis. Lower Vitamin D levels are commonly found in individuals with NAFLD, and more severe liver disease is often associated with deeper deficiency. Shared risk factors, such as obesity, insulin resistance, metabolic syndrome, or sedentary lifestyle, which contribute to both NAFLD and reduced Vitamin D status. Reverse causality, where liver dysfunction may impair Vitamin D metabolism, leading to low circulating levels.

Comparison with established NAFLD grading and staging systems

NAFLD is commonly assessed using various grading and staging systems that help define the severity of the disease. The most widely recognized grading systems include the NAFLD activity score (NAS) and the Kleiner scoring system, which categorize NAFLD based on histological features observed in liver biopsies.

NAS

Grade: The NAS provides a score ranging from 0 to 8, based on three histological features. Steatosis (fat accumulation): Graded from 0 (no steatosis) to 3 (more than 66% of hepatocytes affected). Lobular inflammation: Graded from 0 (none) to 3 (severe inflammation). Hepatocellular ballooning: Graded from 0 (none) to 2 (severe ballooning).

Staging

The staging of NAFLD progression uses the fibrosis score, where; Stage 0: No fibrosis, Stage 1: Portal fibrosis or mild fibrosis, Stage 2: Peri-portal fibrosis with more extensive involvement, Stage 3: Bridging fibrosis, where the liver architecture begins to be disrupted, and Stage 4: Cirrhosis with complete disruption of liver architecture and function.

Findings comparison

In the context of a clinical study, if the observed biopsy reveals steatosis graded as 2 (moderate fat accumulation), with mild hepatocellular ballooning (grade 1), and moderate lobular inflammation (grade 2), this would indicate a NAS of 5, suggesting moderate severity of NAFLD, and potentially a staging of fibrosis stage 1 or 2, depending on the degree of fibrosis. These findings would correlate with the transition from simple fatty liver to more advanced stages, potentially leading to fibrosis [2].

Kleiner scoring system

Fatty liver grading is similar, with scores for Steatosis: 0 (none)–3 (>66% hepatocytes affected), Ballooning: 0 (absent)–2 (severe), Inflammation: 0 (none)–3 (severe), Fibrosis: 0 (none)–4 (cirrhosis),

Comparison with findings

If the liver biopsy shows a score of 2 for steatosis (moderate), 1 for ballooning, and 2 for inflammation, and some initial fibrosis (stage 1), it would be classified as moderate NAFLD and at risk of progression if untreated. If the intervention impacts the fatty liver and reduces these scores (e.g., reducing inflammation and ballooning, or preventing further fibrosis), this would reflect a positive response to treatment.

Timeline of biochemical and symptomatic changes post-intervention

Post-intervention, both biochemical markers and clinical symptoms can reflect changes in the disease's progression. Below is an estimated

timeline of how biochemical and symptomatic changes might evolve in response to an intervention, for example, lifestyle modification, pharmacological therapy, or surgical interventions

Immediate changes (0–4 weeks)

Biochemical

Liver enzymes (aspartate aminotransferase [AST], alanine aminotransferase [ALT]): Typically, liver transaminases show the first improvement, usually decreasing within 1–4 weeks of intervention. A reduction in ALT and AST may be indicative of a reduction in liver inflammation. Lipid profile: A decrease in serum triglycerides and improvement in HDL cholesterol can be observed due to dietary or pharmacological interventions aimed at reducing fat accumulation.

Symptoms: Fatigue and malaise: These non-specific symptoms often improve early in the intervention phase as the liver starts to process and metabolize fats more effectively. Mild abdominal discomfort: Some patients may experience mild discomfort due to changes in liver function, but this generally resolves quickly.

Intermediate changes (1–6 months)

Biochemical

Improved liver function tests

AST and ALT should continue to trend downward. If steatosis is the main driver of NAFLD, markers of fat accumulation, such as the ratio of AST to ALT, may normalize.

Insulin sensitivity

For those with associated metabolic dysfunction, improvements in insulin resistance may be noted, often marked by a reduction in fasting blood glucose or hemoglobin. Fibrosis Biomarkers: Fibrosis markers (such as the enhanced liver fibrosis score,) may show a reduction, suggesting a reversal or stabilization of fibrosis, although this may take longer.

Symptoms

Decreased abdominal discomfort: As liver inflammation subsides, symptoms of bloating or discomfort may improve. Energy levels: Most patients report improved energy levels as their liver function stabilizes.

Long-term changes (6–12 months)

Biochemical

Stable liver enzyme levels: Liver enzymes (ALT, AST) should now be within normal range or near baseline. Improved lipid profile: Cholesterol and triglycerides may remain stable, reflecting the long-term benefit of dietary changes or lipid-lowering agents. Fibrosis regression: Studies show that fibrosis may regress in some patients with sustained lifestyle modification or pharmacological treatment. However, cirrhosis and severe fibrosis are less likely to fully reverse [3].

Symptoms

Resolution of symptoms: Most symptoms, such as fatigue, bloating, or discomfort, should resolve. However, patients with advanced fibrosis or cirrhosis might continue to have chronic issues. Improved quality of life: Long-term follow-up usually shows significant improvement in overall well-being, especially in patients who adhered to lifestyle modifications.

Lipid metabolism and insulin resistance

Vitamin D plays a crucial role in regulating lipid metabolism and insulin sensitivity. Its deficiency can lead to increased lipogenesis and decreased fatty acid oxidation in the liver, resulting in hepatic steatosis. Specifically, Vitamin D deficiency may upregulate sterol regulatory element-binding protein 1c and peroxisome proliferator-activated receptor gamma, both of which promote lipogenesis. In addition, Vitamin D deficiency can impair insulin signaling pathways, contributing to insulin resistance, a key factor in NAFLD pathogenesis.

Inflammation

Vitamin D has anti-inflammatory properties mediated through the Vitamin D receptor (VDR). Deficiency in Vitamin D can lead to increased activation of inflammatory pathways, such as nuclear factor kappa B and mitogen-activated protein kinases. This activation results in elevated production of pro-inflammatory cytokines, exacerbating liver inflammation and promoting the progression from simple steatosis to NASH.

Fibrogenesis

Vitamin D deficiency may contribute to liver fibrosis by promoting the activation of hepatic stellate cells (HSCs). Activated HSCs are the primary source of extracellular matrix production in the liver, leading to fibrosis. Vitamin D, through its receptor VDR, can inhibit HSC activation and proliferation. Therefore, a lack of Vitamin D removes this inhibitory effect, facilitating fibrogenesis.

Gut-liver axis

The gut microbiota and intestinal permeability are also influenced by Vitamin D status. Vitamin D deficiency can lead to dysbiosis and increased intestinal permeability, allowing bacterial endotoxins to enter the portal circulation and trigger hepatic inflammation – a process known as endotoxemia [4]. This mechanism further contributes to the inflammatory milieu in NAFLD.

Vitamin D deficiency can exacerbate NAFLD progression through multiple pathways, including dysregulation of lipid metabolism, induction of insulin resistance, promotion of inflammatory responses, facilitation of fibrogenesis, and disruption of gut-liver axis homeostasis. Addressing Vitamin D deficiency may therefore represent a potential therapeutic strategy in managing NAFLD.

CASE REPORT

The patient came to the outpatient department, presents with complaints of generalized fatigue, intermittent right upper quadrant abdominal discomfort, and a history of unexplained weight gain over the past 12 months, occasional cramps at the upper chondral region and calf muscle spasms when patient trying to stretch his body for minimal warm up physical activity. Recently, forehead facial melanoma and neck skin, mild insomnia, increasing fatigue, particularly during the past 6 months, which has significantly affected his daily activities. He also mentions mild, intermittent right upper quadrant pain, which he attributes to indigestion. Despite following a relatively balanced diet, he has experienced gradual weight gain. His symptoms worsened in the past 2 months. He denies any significant alcohol intake, smoking, or use of hepatotoxic drugs. He also reports no jaundice or changes in bowel movements. However, he admits to a largely sedentary lifestyle and minimal sun exposure, which could contribute to a potential Vitamin D deficiency.

Physical examination

On examination, patient is conscious, coherent, afebrile, vitals were blood pressure 130/80 mmHg, heart rate 78 bpm, respiratory rate 20 cycles/min, random blood sugar 127 mg/dL, temperature 98.7°F, oxygen saturation 99%, appears slightly overweight, but not acutely ill. Mild tenderness in the right upper quadrant, no signs of hepatomegaly or ascites. No palpable masses, no signs of jaundice or rashes, Mild muscle weakness and pain noted, especially in the lower limbs, likely at the left ankle region.

Laboratory investigations

Parameter	Result	After 6 months	Normal value
Hemoglobin	13.4	14.2	13.5–16.5 g
Total WBC count	13100	8650	4000–11000 cells/cumm
Neutrophils	88	62	40–70%
Lymphocytes	10	24	20–45%

Eosinophils	02	02	1–6
PCV	43	41	36–46%
Platelets	2.0	2.6	1.5–4.0 lakhs/cumm
RBS	120	114	80–140 mg/dL
S. creatinine	2.3	1.1	0.5–1.5 mg/dL
S. bilirubin	5.8	0.8	0.1–1.1 mg/dL
S. Na^+	125	146	135–155 mmoL/L
S. K^+	5.0	4.8	3.5–5.5 mmoL/L
AST	45	26	0–42 U/L
ALT	52	17	0–38 U/L
ALP	150	94	28–111 IU/L
Total proteins	6.0	7.2	6.0–8.5 g/dL
Albumin	2.8	4.6	3.5–5.0 g/dL
Globulin	3.2	3.8	
A/G	0.8	1.2	
Vitamin D	12	35	30–70
S. calcium	9.2	9.6	8.5–10.2 mg/dL
Viral markers	HBV, HCV	Non-reactive	Non-reactive
HbA1C	6.4	4.8	6–7

WBC: White blood cell, PCV: Packed cell volume, AST: Aspartate transaminase, ALT: Alanine aminotransferase, ALP: Alkaline phosphatase, HbA1C: Hemoglobin A1C, HBV: Hepatitis B virus, HCV: Hepatitis C virus, RBS: Random blood sugar, S. Na^+: Serum sodium ion, S. K^+: Serum potassium ion, S. creatinine: Serum creatinine, S. bilirubin: Serum bilirubin, S. calcium: Serum calcium

Lipid profile

Parameter	Result	After 6 months	Normal value
S. triglceride	375	128	60–160 mg/dL
Total cholesterol	153	160	130–250 mg/dL
HDL	34	42	30–60 mg/dL
LDL	119	85	0–150 mg/dL
VLDL	75	21	0–27 mg/dL
Cholesterol/HDL	4.5	3.6	0.7–4.3
LDL/HDL	1.2	2.02	0.4–3.1

LDL: Low-density lipoprotein, HDL: High-density lipoprotein, VLDL: Very low-density lipoprotein

Urine routines

U. Sugar	Nil
U. Albumin	Trace
Bile salts/bile pigments	Negative
U. Ketones	Negative
RBC	Nil
Pus cells	8–10/Hpf
Epithelial cells	2–4/hpf
Casts and crystals	Nil
Urine pH	6.2
Urine specific gravity	1.030 (1.010–1.025)

RBC: Red blood cells

Imaging

Ultrasound of the abdomen

Mild hepatic steatosis with Grade - II fatty liver. Small right renal calculi (2 mm). No evidence of cirrhosis or other structural abnormalities.

FibroScan (transient elastography)

7, a low level of liver stiffness, indicating early-stage fibrosis (F0-F1).

2D Echocardiogram

Normal chambers and valves with ejection fraction 64%.

Diagnosis

The patient was diagnosed with NAFLD likely exacerbated by Vitamin D deficiency. The diagnosis was based on clinical presentation, elevated liver enzymes, ultrasound evidence of hepatic steatosis, and low Vitamin D levels. Imaging Ultra sound abdomen Fig 1 and reduced hepatocellular injury in Fig 2.

DISCUSSION

Vitamin D is a fat-soluble vitamin with significant roles in bone metabolism and immune function. Recent studies suggest that Vitamin D deficiency is associated with an increased risk of metabolic syndrome, obesity, insulin resistance, and fatty liver disease [5]. Specifically, low levels of Vitamin D have been linked to the development of NAFLD, potentially through its effects on insulin sensitivity and the inflammatory process within the liver [6]. Several mechanisms may explain how Vitamin D deficiency contributes to fatty liver disease. Insulin resistance: Vitamin D plays a role in the regulation of insulin sensitivity. Deficiency may lead to increased insulin resistance, which is a key factor in the pathogenesis of NAFLD [7]. Inflammation and fibrosis: Vitamin D has anti-inflammatory properties and may modulate cytokine production. Reduced levels could contribute to increased hepatic inflammation, leading to fatty infiltration and progression to NASH. Lipid metabolism: VDRs are found in liver cells, and deficiency may disrupt normal lipid metabolism, contributing to lipid accumulation in hepatocytes [8].

In this case, the patient's low Vitamin D level, combined with clinical and imaging findings suggestive of NAFLD, supports the Vitamin D deficiency is contributing to the patient's liver disease. In addition, the patient's sedentary lifestyle and limited sun exposure may have exacerbated the deficiency, thereby accelerating the progression of fatty liver disease.

Management

Vitamin D supplementation

The primary intervention for Vitamin D deficiency is supplementation. The patient was prescribed 6 lakhs injection IM Stat and Vitamin D_3 60kone tablet/week and 250 IU with 500 mg calcium daily, advised to follow-up in 3 months for reassessment of Vitamin D levels.

Lifestyle modifications

Dietary changes, such as a diet low in saturated fats and high in antioxidants to reduce liver fat accumulation and Encouragement to engage in regular physical activity, aiming for at least 150 min of moderate-intensity exercise per week.

Monitoring

Regular follow-up with liver function tests and ultrasound is recommended to monitor for any progression of fatty liver disease. The patient will be reassessed for Vitamin D levels in 3 months.

With appropriate Vitamin D supplementation and lifestyle changes, the patient is expected to experience improvement in both Vitamin D levels and liver function. However, ongoing monitoring is required to prevent the progression of NAFLD to more severe forms like NASH or cirrhosis.

Expected timeline for key changes post-intervention

Time point	Expected changes
1–3 months	↓ALT/AST, ↑25(OH) D levels, initial weight loss, ↑energy levels
3–6 months	Improved lipid profile, ↓insulin resistance, ↓and hepatic steatosis on imaging
6–12 months	Potential fibrosis stabilization/regression, sustained symptom resolution, improved quality of life

ALT: Alanine transaminase, AST: Aspartate aminotransferase

Liver enzymes (ALT, AST, Gamma-glutamyl transferase)

Early (1–3 months): A reduction in ALT and AST levels is one of the first measurable signs of reduced hepatic inflammation and fat accumulation. Sustained improvement (6–12 months): Persistent normalization or downward trend of liver enzymes supports the resolution of steatosis and reduced hepatocellular injury.

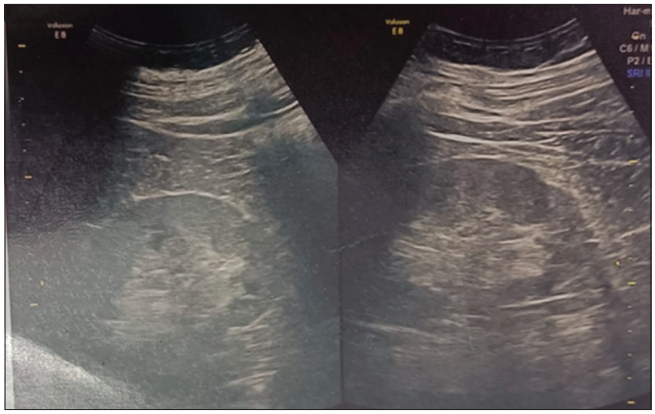

Fig. 1: Grade-II fatty liver with mild steatohepatitis

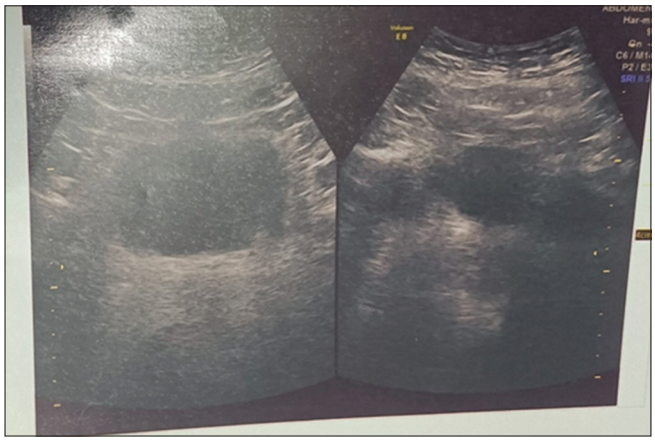

Fig. 2: Grade-I fatty liver with mild steatohepatitis after the treatment

Vitamin D status (25[OH]D levels)

Post-supplementation: Serum 25-hydroxyvitamin D (25[OH]D) levels are monitored to ensure that levels reach the optimal range (30–50 ng/mL). Improved Vitamin D status has been associated with decreased levels of pro-inflammatory cytokines (e.g., tumor necrosis factor-α, interleukin-6) and oxidative stress markers in NAFLD patients.

Metabolic and lipid profile

Insulin resistance: Reduced fasting glucose and homeostatic model assessment of insulin resistance scores indicate improved insulin sensitivity. Triglycerides and high-density lipoprotein (HDL): Lifestyle changes can improve dyslipidemia by lowering triglyceride levels and increasing HDL cholesterol [9].

Weight loss

Target ≥5–10% weight loss from baseline is associated with histological improvement in steatosis; ≥10% may lead to fibrosis regression. Measured regularly (monthly or quarterly), weight loss is a practical marker of adherence and effectiveness of lifestyle change.

Waist circumference and body mass index (BMI)

Reductions in waist circumference and BMI correlate with improved hepatic fat content and metabolic outcomes.

CONCLUSION

Vitamin D deficiency is a modifiable risk factor in the development of fatty liver disease. In this case, the 36-year-old male patient's liver disease appears to be significantly influenced by low Vitamin D levels. Addressing the deficiency through supplementation, alongside lifestyle modifications, may improve liver health and prevent further complications. This case underscores the importance of considering Vitamin D levels in the management of patients with NAFLD.

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AUTHORS CONTRIBUTIONS

RK and AK contributed to the idea of the study and work proposal and supervision; ST collected the patient data, consent for the study, documentation, and writing the manuscript. "All authors read and approved the manuscript."

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Written informed consent was signed by the study participant. The study approval was taken from the Institutional Ethical Committee (IEC), Amaravathi Institute of Medical Sciences, Amaravathi Hospital, Guntur, with Ethical Committee number -IEC/2022/09/AIMS/H.

CONFLICT OF INTEREST

Authors had no conflict of interest.

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REFERENCES

1. Warman NB, Shah FZ, Mohamad AF, Zainordin NA, Awang MH, Koshy M, *et al*. Vitamin D deficiency in patients with non-alcoholic fatty liver disease and type 2 diabetes mellitus. J ASEAN Fed Endocr Soc. 2024;39 Suppl 1:10. doi: 10.15605/jafes.039.S1.010
2. Kumar M, Parchani A, Kant R, Das A. Relationship between vitamin D deficiency and non-alcoholic fatty liver disease: A cross-sectional study from a tertiary care center in Northern India. Cureus. 2023;15(2):e34921. doi: 10.7759/cureus.34921, PMID 36938188
3. Permatasari D, Aini N, Yosmar R. Effects of post-chemotherapy drugs on the liver function tests in pediatric patients with acute lymphoblastic leukemia (all). Int J Appl Pharm. 2025;17(1):61-6. doi: 10.22159/ijap.2025.v17s1.09
4. Musso G, Gambino R, Cassader M, Pagano G. The role of vitamin D in nonalcoholic fatty liver disease: Mechanisms and therapeutic implications. Curr Diabetes Rep. 2010;10(2):135-41. doi: 10.1007/s11892-010-0120-0
5. Rosyid FN, Haryoto O, Kristinawati B, Rahman AF. The effect of supplementation of capsule with a combination of *Momordica charantia* fruit and *Moringa oleifera* leaves extract on the biomarker of liver and renal function in the limited healthy volunteers. Int J Appl Pharm. 2024;16(5):114-7. doi: 10.22159/ijap.2024v16s5.52468
6. Hsia SH, Gattu S. Vitamin D and fatty liver disease: A review of the literature. J Clin Med. 2018;7(12):486. doi: 10.3390/jcm7120486
7. Muthusamy S, Chidambaram M. Vitamin D deficiency and nonalcoholic fatty liver disease. J Clin Transl Hepatol. 2015;3(3):148-53. doi: 10.14218/JCTH.2015.00009
8. Laird E, Campbell M. Vitamin D and its role in nonalcoholic fatty liver disease. World J Hepatol. 2015;7(2):262-8. doi: 10.4254/wjh.v7.i2.262
9. Weng SW, Chou CK, Wang WH. Association between vitamin D and fatty liver disease: A review of clinical studies. J Nutr Biochem. 2018;62:1-8. doi: 10.1016/j.jnutbio.2018.01.010