How to Cite:
Haq, A. U., Shahab, K., Khattak, I. Q., Khan, F., Khan, A., Ullah, H., & Zeb, F. (2023). Deficiency of vitamin D in patients with chronic liver disease at department of medicine HMC Peshawar a multi center study. *International Journal of Health Sciences*, *6*(S10), 1306–1011. https://doi.org/10.53730/ijhs.v6nS10.13950

Deficiency of vitamin D in patients with chronic liver disease at department of medicine HMC Peshawar a multi center study

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***Abstract*---**Background: Multiple nutrients are deficient in people with chronic liver disease (CLD), but vitamin D insufficiency is more prevalent in CLD patients than in patients with other deficiencies. As a result of liver fibrosis, the liver's synthetic abilities are diminished, and this compromises activation through a decrease in vitamin D Binding Protein, which results in vitamin D insufficiency. The situation is further complicated by a decline in performance and nutritional deficiencies. The research examined the prevalence of vitamin D insufficiency in people with chronic liver disease and its relationship to the severity of the condition. Methodology: The Multi

center study conducted in department of Medicine at the Hayatabad Medical Complex in Peshawar carried out this. 71 CLD patients who satisfied the criteria were included in the research. Three groups of patients were divided based on their Child-Pugh scores. The patient's vitamin D levels were examined by hospital laboratories. Serum (26,OH)D concentrations below 30 nmol/L were considered to represent [vitamin D] insufficiency levels. The data were examined using SPSS version 22. Results: 71 CLD patients were assessed. In this study the (mean age) was 52.43 ± 12.190.years. The (male-to-female ratio) was 02.04:01. Hepatitis C was the underlying etiology in 58% of CLD patients. 41 patients (59%) had Child-Pugh Class C chronic liver disease. 43 patients (66%) had low vitamin D levels. Vitamin D insufficiency correlated with gender and (Child-Pugh Class). Conclusion: Patients with liver cirrhosis typically lack enough vitamin D. In our study low levels of vitamin D were identified in 66% of the patients. Inadequate vitamin D levels were more prevalent in females and those with severe liver fibrosis.

***Keywords*---**deficiency vitamin D, level vitamin D, fibrosis, chronic liver disease.

Introduction

Chronic liver disease (CLD) is a multi-nutrient deficient state. Patients with CLD are susceptible to micro and macronutrient deficiency ranging from protein and several vitamin deficiencies to minerals like zinc and selenium. (Deficiency of fat-soluble vitamins) is also frequently observed [01]. Vitamin D insufficiency is frequent in severe chronic liver disease patients, although people with less severe chronic liver disease may also be deficient [02]. Vitamin D deficiency increases mortality, morbidity, and CLD consequences such as recurring bacterial infections and portal hypertension problems [03]. CLD disease patients may have low vitamin D due to hepatic vitamin D metabolism alteration[04]. Under UV radiation, the skin synthesizes inactive vitamins D2 and D3, which the liver activates by hydroxylation. In liver fibrosis, the liver loses its capacity to hydroxyl ate the inactive form of vitamin D and consequently patients with Child-Pugh Class C have significant frequency of low vitamin D levels.[05,]. Vitamin-D deficient diet, decreased vitamin- D intestinal absorption, and sunshine reduction worsen the situation[06]. Vitamin D deficiency in chronic liver disease patients varies with fibrosis severity. Falak et al. found 55.2% vitamin D insufficiency in de compensated cirrhotic patients and 13.6% in compensated ones [07]. Arteh et al. found 92% of chronic liver disease patients had vitamin D insufficiency, with a pre-ponderance of Afro-American race. Our country has few studies regarding vitamin D insufficiency in chronic liver disease patients, and its relationship with liver disease status. This research addresses this information gap[08].

Methodology

The Multi-center study was carried out at the Department of medicine HMC hospital Peshawar total sample size 71 patients. The research, which was

conducted from December 2022 to August 2023 at the Medicine department of the Hayatabad Medical Complex, received approval from the institution's ethical review board.

Inclusion Criteria

CLD patients from 21 to 85years and of both genders fulfilling the study criteria were included. CLD was diagnosed if patients had one or more of the following: (1) If they have biochemical and synthetic function abnormalities suggestive of CLD along with risk factors for CLD.(2) Features of CLD such as surface nodularity, coarse heterogeneous echo texture, hypertrophic or atrophic liver segments on ultrasound[09].(3) Liver biopsy or medical records suggested CLD. Child-Pugh scores divided CLD patients into three groups. Patients with a Child-Pugh score of six or less were categorized as Class A, seven to nine as Class B, and higher than nine as Class C.

Exclusion Criteria

Patients with a history of vitamin-D deficiency, chronic renal illness, vitamin- D supplementation, and steroid use in the last six months were excluded.

Data Collection

Demographics including age, gender, the underlying cause of CLD, duration of CLD, and Child-Pugh Class were noted from patients' records. Relevant history of vitamin D deficiency like bone fractures was taken, followed by detailed physical examination for any signs of hypo vitaminosis D. Vitamin D level was determined in the hospital laboratory in the blood sample of the patient. Serum vitamin D 25,OH, D levels below 30 nmol/L are considered inadequate.

Data Analysis

IBM SPSS 28 statistical program analyzed the data. For qualitative variables such as gender, CLD etiology, Child-Pugh Class, and low vitamin D levels, frequencies and percentages were computed. Mean ± SD were calculated for quantitative variable such as Age, CLD duration, and blood vitamin D levels.To determine statistical significance, Chi-Square testing for categorical variables and Student T-tests for continuous variables were also utilized. For the correlation of vitamin D insufficiency with gender, age groups, and Child-Pugh Class, the Chi-Square test of independence with Cramer V nominal was used. Statistics were deemed significant at p-values under [0.05]

Results

71 patients included in this study The mean patient age was 52.42 ± 11.181 years. Range of patient’s age was between 21 to 85 years. 45 patients (69%) were men, and 26 (31%) were women. The male-female ratio was 2.4: 1. Table 1 highlights patient features.

Table 1
Characteristics and percentage n-71

[Characteristics]	[N] (%)
1.Age (Years) 2.Mean 3.Standard Deviation 4.Range	 52.42 ± 12.181 21-85
5.Gender 6..Male 7.Female	 45 (69%) 26 (31%)
8.Etiology Of CLD 9.Hepatitis C 10.Hepatitis B 11.Hepatitis C And Hepatitis B 12.Primary Biliary Cirrhosis 13.Miscellaneous	 40 (57) 16 (22) 06 (08) 03 (04) 07 (09)

41 patients (59%) had Child-Pugh Class C chronic liver disease, and 15 (21%) had both Class A and B. HCV was the most prevalent underlying cause of CLD (40 patients, 58%), followed by HBV in 16 (21%), HBV+HCV in 06 (08%), PBC in 03 (03%), and hemochromatosis ,NAFLD, Wilson or no underlying cause in 07 (09%) individuals despite extensive workup. The etiology of these 07 patients was labelled as Miscellaneous. 47 individuals 66%) had vitamin D insufficiency. 38 (52%) of 36 patients were aged 45-65, and 25 (50%) were vitamin D deficient. This study found no age-related vitamin insufficiency correlation (p = 0.816). Gender correlated with vitamin D insufficiency (p = 0.023). 31 men and 34 women were vitamin D deficient. Vitamin D insufficiency associated with Child-Pugh Class [p < 0.001]. (Child-Pugh Class) illness patients have more significant vitamin D insufficiency. 37 of 41 Child Class patients were vitamin D deficient.

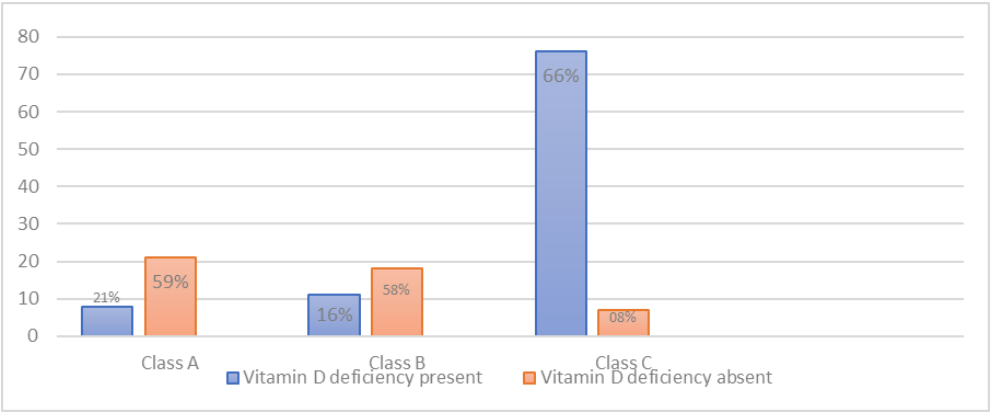

Figure 1. Vitamin D Deficiency in Different Child-Pugh Classes

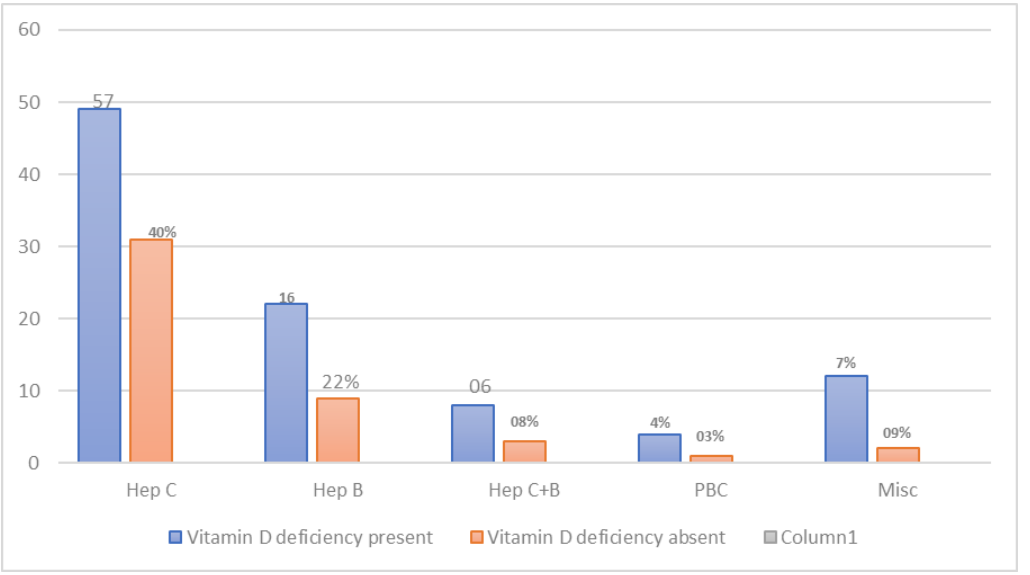

Figure 2. Based on the Causes of CLD, the Status of Vitamin D Deficiency

Discussion

Vitamin D is absorbed via the stomach from dietary sources to meet the body's needs. Second, exposure to UV light in the skin's epidermal cells leads to its predominate endogenous synthesis[09]. The liver uses a protein called vitamin D binding protein (DBP), an analog of albumin, to hydroxyl ate the inactive vitamin D that has thus been generated, activating it. The synthetic function of the liver is hampered in fibrotic disorders like chronic liver disease (CLD), where the normal parenchyma of the liver is replaced by fibrous tissue[10]. This results in a drop in DBP, which finally causes vitamin D deficiency[11].Vitamin D deficiency was seen in 67.4% of CLD patients in this research. In their study, Arteh et al. discovered a 92.4% vitamin D deficit. Similar to this, Falak et AL research .'s revealed that 76.5% of participants had insufficient levels of vitamin D.

Given that more women than males participated in these research, there may be a high prevalence of vitamin D inadequacy. Patients with chronic liver disease are not an exception to the general rule that gender influences vitamin D inadequacy. According to research by Johnson and colleagues, vitamin D insufficiency is more common in females with CLD [12]. The information we gathered shows that women with CLD had a higher prevalence of vitamin D insufficiency than men do (p 0.025). The high protein intake of the local people may lead to a vitamin D deficit. Women are less exposed to sunlight as a result of religious and socio-cultural norms, which also results in vitamin D deficiency. Patient Child-Pugh scores were negatively linked with vitamin D deficiency (p 0.001). The vitamin D deficiency increased as the stage of fibrosis advanced from Class A and B to Class C. As fibrosis worsens, liver synthesis declines, which lowers DBP and inhibits vitamin D activation. The Jamil et al. analysis found comparable patterns[13].

Conclusion

Low vitamin D levels are seen in the majority of CLD patients. Women and those with severe fibrosis are more likely to have vitamin D deficiencies. Large-scale multicenter studies should be conducted to assess vitamin D deficiency in CLD patients in our community. All CLD patients should have their vitamin D levels evaluated, and any deficiencies should be addressed very once.

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