compared to the TT genotype (198.68 vs. 177.85 mg/dL, p=0.010) and the AA genotype (196.81 vs. 178.58 mg/dL, p=0.006), respectively. In chronic patients, the GG+GA genotypes of *IL10* rs1800896 were associated with high insulin levels compared to the AA genotype (17.22 vs. 12.04 IU/mL, p=0.021).

Conclusions: The CC genotype of the *IFNL3* rs4803217 gene was associated with SC in patients from West Mexico. *IL10* and *IFNL3* polymorphisms increased TChol in SC patients. These results suggest an interaction between metabolic and immune factors in the outcome of HCV infection.

Ethical statement

The protocol was registered and approved by the Ethics Committee. The identity of the patients is protected. Consentment was obtained.

Declaration of interests

None

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25-hydroxyvitamin D deficiency as a factor associated with the development of Hepatic Encephalopathy in the Mexican population.

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Introduction and Objectives: Hepatic Encephalopathy (HE) is a common complication in patients with Chronic Liver Disease (CLD), and the development of this decompensation is multifactorial, including ammonia levels, inflammatory status, and sepsis, among others. A poorly studied factor in our population is the serum levels of 25-hydroxyvitamin D (25-OHD), which could act as a co-factor in HE. To assess if serum 25-hydroxyvitamin D (25-OHD) deficiency acts as a cofactor in the development of HE.

Materials and Patients: Observational, retrospective, analytical, case-control study; included subjects of both sexes, 18 years old and over, diagnosed with Chronic Liver Disease of different etiologies. Complete blood count, liver and kidney function, serum electrolytes, coagulation profile, and serum levels of 25-hydroxyvitamin D were recorded. They were evaluated using the West-Haven Criteria (WH).

Results: Independent samples T-test was used to compare differences between 25-hydroxyvitamin D levels in patients with and without HE. The association between 25-OHD deficiency and HE was assessed using a chi-square test, with a significance level set at alpha=0.05. Out of a total of 96 patients, 36.5% had HE. The mean 25-OHD level in the HE group was 18.78 ± 8.56, compared to 22.77 ± 9.94 in the group without HE. The T-test was significant: T (1=2.072), p =0.041. Among patients with deficiency, 20/35 (57.1%) had EH, while 22/61 (36.1%) did not have HE. The chi-square test for the association between deficiency and HE was positive, with a value of (1) =4.015, p =0.045.

Conclusions: A causal relationship between 25-hydroxyvitamin D (25-OHD) deficiency and the development of HE cannot be attributed, as this is multifactorial. However, 25-OHD deficiency is common in patients with Chronic liver disease, and our study demonstrates that this deficiency acts as a cofactor, as there is a significant difference between the groups. It is necessary to validate these findings in the future through multivariate analysis to confirm our results.

Ethical statement

The protocol was registered and approved by the Ethics Committee. The identity of the patients is protected. Consentment was obtained.

Declaration of interests

None

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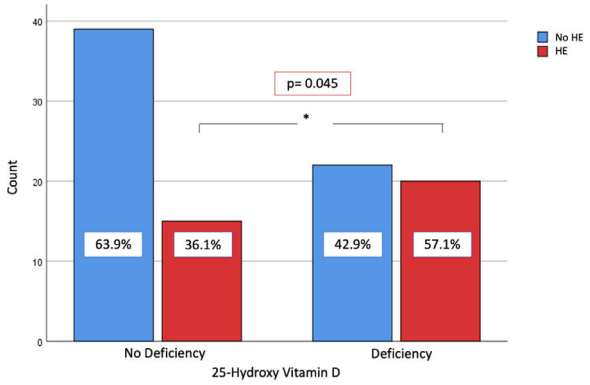

Figure 1. Percentage of patients with 25-OHD deficiency and HE

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Incidence and Associated factors to development of hyponatremia in a cohort of ambulatory patients with compensated liver cirrhosis

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Introduction and Objectives: Hyponatremia is associated with ascites, hepatic encephalopathy, primary bacterial peritonitis, and increased mortality. However, the information about incidence and factors associated with hyponatremia in ambulatory patients with compensated cirrhosis is scarce. The aim of the study was to estimate the incidence and associated factors to the development of hypervolemic hyponatremia.

Materials and Patients: Ambulatory patients with compensated cirrhosis seen at Medical Center Siglo XXI were selected. All variables included in Child-Pugh Index and in the MELD Score and the types of treatment diet were analyzed. Hyponatremia was considered when