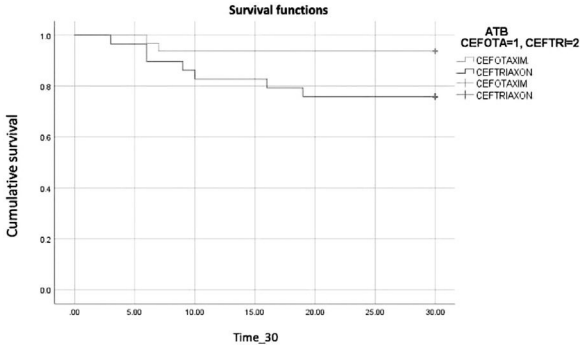

Figure 2. Kaplan Meyer survival curve with respect to type of antibiotic.
Source: Data extracted from records of the Gastroenterology Service of the Hospital General de México "Dr. Eduardo Liceaga".

https://doi.org/10.1016/j.aohep.2025.101863

The role of the prognostic nutritional index as a prognostic factor in patients with hepatocellular carcinoma.

Vilma Hernandez-Garza, Berenice Lorenzo-Valle, Kenia Michel Bastida-Guadarrama, Santiago Camacho-Hernandez, Fátima Higuera-de-la-Tijera

Department of Gastroenterology and Hepatology, General Hospital of Mexico "Dr. Eduardo Liceaga", Mexico City, Mexico

Introduction and Objectives: Immune-nutritional status has been demonstrated to be associated with prognosis in patients with various malignancies. The aim of this study is to determine the association of the nutritional prognostic index (PNI) with survival and assess the correlation between PNI and clinic-pathological parameters in HCC patients.

Materials and Patients: An observational, retrospective, descriptive and case series study was performed. We included 69 patients from a third-level hospital with a diagnosis of HCC from February 01, 2019 to July 24, 2023 and studied their survival 12 months after diagnosis. We determined the association between PNI status and their clinic-pathological characteristics and evaluated the impact of PNI on the HCC patient survival. The following formula was used to determine PNI: $10 \times$ serum albumin (g/dL) $+ 0.005 \times$ total lymphocyte count (per μL). Clinic-pathological characteristics related to disease prognosis included age, sex, body mass index (BMI), alpha-fetoprotein (AFP) value, the presence or not of cirrhosis, tumor size, ECOG score, and BCLC grade. Qualitative data are expressed as percentages and quantitative data as mean±SD. Statistical comparison was performed with two-tailed unpaired Student's t-test or chi-square and Fisher's exact test. Statistical significance was defined as $P<0.05$.

Results: Of the 69 patients diagnosed with HCC, most of the studied population was found to involve males at 56.52% while females were reported at 43.48%, with an age range of 26-81 years and a median age of 61.84, 61.84±9.8 (59.53-64.15). Of the total sample, 10.14% were patients with no diagnosis of cirrhosis while 89.86% were patients with some degree of cirrhosis (Child Pugh Stages: A 37.68%, B 43.48%, C 8.70%). The results indicated that low PNI is associated with poor prognosis while high PNI was found to be beneficial for survival with a 95 % CI=35.48±8.41 (32.42-38.54), 95% CI=40.86±5.42 (39.19-42.54) p=0.0019, respectively. It is also associated with more favorable outcomes, such as lower AFP, lower ECOG grade and lower BCLC staging (p=0.0371, p=0.0303, p= 0.002), respectively. However, we found that age, sex, presence or absence of cirrhosis, BMI and tumor size were not statistically significantly associated with the PNI value.

Conclusions: PNI is an independent predictive indicator of survival and is significantly associated with serum AFP, ECOG score, and BCLC stage in patients with HCC.

Ethical statement: The research was carried out in accordance with the Helsinki Declaration of the World Assembly 2013.

Declaration of interests: None.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

https://doi.org/10.1016/j.aohep.2025.101864

Clinical and demographic characteristics of liver transplant recipients who develop steatosis in the liver allograft.

Edgar A. Quezada-Cornejo[1], María F. Hernández-Torres[1], Pedro A. López-Hernández[1], Alma L. Kuljacha-Gastelum[2]

[1] *Department of Gastroenterology, High Specialty Unit 25, Monterrey, NL, Mexico*
[2] *Gastroenterology, Hepatology & Liver Transplant, Ángeles Valle Oriente Hospital, Monterrey, NL, Mexico*

Introduction and Objectives: Liver steatosis (LS) can develop in liver transplant (LT) recipients, with different studies reporting prevalence of 30-60%, our country has a high prevalence of the components of metabolic syndrome. The objective of this study is to describe the characteristics of liver transplant recipients who develop steatosis.

Materials and Patients: Retrospective, transversal and descriptive study in which 28 LT recipients with diagnosis of LS after LT were included, data was retrieved from patients clinical file and the following data were considered: age, sex, history of obesity, arterial hypertension, diabetes mellitus (DM), dyslpidemia and metabolic syndrome (MS) prior and after LT. Other data included were etiology of cirrhosis, immunosuppressive treatment and liver biochemistry at the moment of diagnosis.

Results: A statistic sample of 28 LT recipients who developed LS after LT was analyzed, 12 were male (42.9%) and 16 female (57.1%) with a medium age of 52 (27-74). The medium of post-LT years at diagnosis was 8 years (1-19). Etiology of cirrhosis was autoimmune in 14 (50%) patients, viral in 6 (21.4%), steatosis in 4 (13.8%) and alcohol in 4 (13.8%), the diagnostic method was imaging in 15 (53.6%) patients and biopsy in 13 (46.4%). The medium body mass index (BMI) was 28.6 (22-39), presence of pre-LT DM in 4 (13.8%) patients and post-LT DM in 15 (53.6%), pre-LT and post-LT obesity was found in 5(10.7%) and 15 (53.6%) patients, respectively, pre-LT and post-LT arterial hypertension in 3 (10.7%) and 11 (39.3%) patients respectively, pre-LT and post-LT dyslipidemia in 0 (0%) and 22 (78.6%) respectively, pre-LT and post-LT MS in 0 (0%) and 15 (53.6%) respectively. 24 (85.7%) patients used prednisone at diagnosis with a medium dose of 11.8 milligrams (5-30). Previous to diagnosis, 24 (85.7%) received tacrolimus and 23 (82.1%) sirolimus. Liver biochemistry showed the following mediums: ALT 85.3 (16-406), 50.5 (14-273), ALP 139.8 (38-519), GGT 237 (11-2296) and TBIL 0.7 (0.2-1.5).

Conclusions: This study highlights the frequency with which metabolic comorbidities after LT presented in comparison to the ones presented before LT, especially DM, obesity and dyslipidemia. Concluding, LT recipients should be tightly monitored for these metabolic parameters to prevent LS in a timely manner.

Ethical statement: The identification of patients was protected; informed consent was obtained.

Declaration of interests: None.

Funding: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Characteristics of LT recipients who develop steatosis	
Variable	Total (%)
Sex	
Male	12 (42.9)
Female	16 (57.1)
Age (médium)	52
Etiology	
Autoimmune	14 (50)
Viral	6 (21.4)
Steatosis	4 (13.8)
Alcohol	4 (13.8)
Diagnostic method	
Imaging	15 (53.6)
Biopsy	13 (46.4)
BMI (médium)	28.6
DM	
Pre-LT	4 (13.8)
Post-LT	15 (53.6)
Obesity	
Pre-LT	5 (17.9)
Post-LT	15 (53.6)
Arterial hyertension	
Pre-LT	3 (10.7)
Post-LT	11 (39.3)
Dyslipidemia	
Pre-LT	0 (0)
Post-LT	22 (78.6)
MS	
Pre-LT	0 (0)
Post-LT	15 (53.6)

Table 2.

Liver biochemistry of LT recipients who develop steatosis	
Variable	Medium
ALT	85.3
AST	50.5
FA	139.8
GGT	237
BT	0.7

ALT, Alanine aminotransferase; AST, Aspartate aminotransferase; BT, Total bilirubin; FA, Alkaline phosphatase; GGT, Gamma-glutamyl transferase.

https://doi.org/10.1016/j.aohep.2025.101865

Ischemic Hepatitis and Cardiac Tamponade, a rare association.

María E. Hernández-Ortega,
Viridiana Ramírez Villagrán, Thania B. Zurita-Cruz,
Oscar J. Tercero-Colmenares

Internal Medicine, General Hospital "Dra. Matilde Petra Montoya Lafragua" ISSSTE, Mexico

Introduction and Objectives: Ischemic hepatitis transiently elevates aminotransferases due to reduced oxygen delivery to the liver. The most common cause is heart failure[1]. Cardiac tamponade is an accumulation of pericardial fluid that can cause hemodynamic compromise[2]. The association of both is unusual, which is why it is important to identify them.

Materials and Patients: A 50-year-old patient with a history of type 2 diabetes, systemic arterial hypertension and chronic kidney disease, presented in November 2023 due to hypotension with data of low output during a hemodialysis session, adding dyspnea on minor exertion and abdominal pain located in the right hypochondrium. Upon admission with hemodynamic instability, it was decided to start vasopressor support. In laboratory studies, it presents elevated aminotransferases (Alanine aminotransferase at 1947 U/L and aspartate aminotransferase at 2649 U/L), lactate at 5 mmol/L, lactic dehydrogenase at 2166 U/L and elevated INR at 3.15. An ultrasound of the liver and bile ducts was performed, reporting parenchyma with increased echogenicity and pericardial effusion. An evaluation was requested by Cardiology, performing a transthoracic echocardiogram, showing severe pericardial effusion with a separation of up to 34 mm in the basal region. Pericardiocentesis was performed with the extraction of 850 milliliters of pericardial fluid. As part of the approach, viral and autoimmune etiology was ruled out as a cause of liver disease. PCR for Mycobacterium tuberculosis in the pericardial fluid was requested with a negative report and no malignancy data in the pericardial effusion approach. Patient with clinical improvement and progressive decrease in transaminase levels until normalization.

Results: Ischemic hepatitis has been associated with cardiovascular diseases. The pathogenesis of ischemic hepatitis appears to occur as a result of two mechanisms, when the liver that is at risk is subsequently exposed to systemic hypoperfusion and ischemia, ultimately resulting in a marked but transient elevation of aminotransferases[3]. The diagnosis is largely clinical and uses three criteria, a clinical setting of cardiac, circulatory, or respiratory failure, transient increase in serum aminotransferase activity, and exclusion of other causes of liver cell necrosis, especially viral hepatitis or induced drugs hepatitis[1]. Other abnormal laboratory findings may be found in patients with ischemic hepatitis, such as increased lactic dehydrogenase levels, reduced prothrombin activity, increased serum creatinine, serum bilirubin, and serum lactate levels, due to an abnormal hepatic clearance. Non-invasive imaging options, such as abdominal ultrasound, may aid in the diagnosis of ischemic hepatitis. Dilatation of the inferior vena cava and suprahepatic veins due to passive congestion suggests this. However, the diagnostic utility of ultrasound has not yet been validated[1].

Conclusions: Ischemic hepatitis is a cause of elevated aminotransferase levels, a consequence of a serious underlying disease that leads to a >50% in-hospital mortality rate[3]. The only recognized treatment is to correct the predisposing condition. Timely recognition is vital, as delaying diagnosis can worsen outcomes[4].

Ethical statement: The standards of the Declaration of Helsinki were taken into account. This study is considered risk-free, following the Regulations of the General Health Law on Health Research, Second Title, Chapter I, Article 17, Section II, published in the Official Gazette on January 6, 1987.

Declaration of interests: None.