Declaration of interests: None.

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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.

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Ischemic Hepatitis and Cardiac Tamponade, a rare association.

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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.

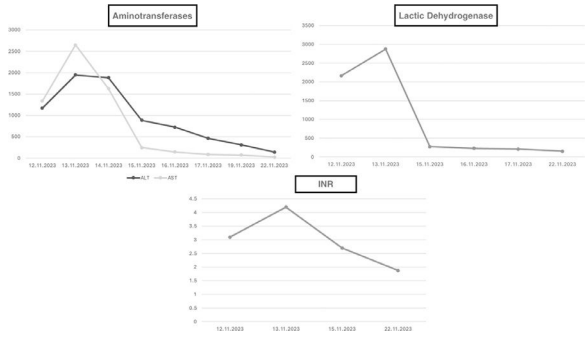
Results: Ischemic hepatitis has been associated with cardiovascular diseases. The pathogenesis of ischemic hepatitis appears to occur as a result of two mechanisms, when the liver that is at risk is subsequently exposed to systemic hypoperfusion and ischemia, ultimately resulting in a marked but transient 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.

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



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Novel bacterial cluster "Prevotella, Bacteroides and Suterella" associated with mortality in Mexican patients with acute-on-chronic liver failure (ACLF) and clinical utility of systemic hs-CRP and IL-6: A frontier approach involving next-generation sequencing at the intestinal level in a cohort by alcoholic etiology.

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Introduction and Objectives: ACLF is characterized by acute decompensation of cirrhosis, organ failure, and high short-term mortality. Several studies have demonstrated the relevance of intestinal microbiota (IM) in the pathophysiology of cirrhosis. To date, there are no studies in the Mexican population focused on IM in alcohol-associated ACLF and its relationship with mortality and inflammatory markers.

Aim: To analyze the composition and diversity of IM in patients with alcohol-associated cirrhosis and ACLF, healthy controls, and its correlation with inflammatory markers.

Materials and Patients: Cross-sectional study, which included 22 decompensated patients with ACLF, 16 decompensated patients without ACLF (CD) and 18 healthy individuals (HI), recruited at the Hospitales Civiles de Guadalajara. Fecal IM was characterized by NGS of the 16S-rRNA gene. Systemic levels of high-sensitivity C-reactive protein (hs-CRP) and interleukin 6 (IL-6) were quantified by ELISA, and bioinformatics analysis of IM was performed using the QIIME2 package. Quality filtering, which includes removal of chimeras and non-biological sequences, was performed using the DADA2 algorithm. Resulting ASVs were taxonomically assigned through a self-trained naïve Bayesian classifier, against the SILVA database. Furthermore, α and β diversity analyses, relative abundances, and ANCOM-BC compositional analysis were performed in the QIIME2 package. Predictive values and associations were performed using ROC curves and Spearman correlations, respectively.

Results: ACLF and CD patients showed significantly lower α-diversity compared to CS. The comprehensive bacterial taxonomy profile in ACLF was significantly dominated by pathogenic/inflammatory genera such as Escherichia/Shigella, Enterobacter and Prevotella. In contrast, we observed a depletion of Bacteroides compared to CD. Interestingly, the subanalysis of MI in ACLF patients categorized at 7 and 90 days of mortality showed consistency with the enrichment of the Prevotella, Bacteroides and Suterella cluster. The Proteobacteria/Firmicutes ratio as a potential marker of dysbiosis, was significantly elevated in ACLF patients. Serum levels of hs-CRP and IL-6 were potentially increased in ACLF, in comparison to CD and CS. hs-CRP correlated positively with IL-6 and the Proteobacteria/Firmicutes ratio and negatively with α-diversity. IL-6 levels were positively correlated with MELD-Na. Finally, ROC curve analyses showed that hs-CRP allows discrimination of infections in patients with CD with a cut-off point >70.7 mg/L (AUROC: 0.75, with 90% sensitivity and 68.9% specificity). IL-6 allows discrimination of hepatic encephalopathy (HE) in patients with CD and ACLF with a cut-off point >7051.1 pg/mL (AUROC: 0.67, with 81.4% sensitivity and 45.8% specificity).

Conclusions: The dysbiotic/proinflammatory profile of IM in ACLF correlated with the potential increase in systemic inflammation. The bacterial cluster "Prevotella, Bacteroides and Suterella" represents a hallmark of mortality within 7 and 90 days. IL-6 and hs-CRP allow discrimination of HE and infections in patients with alcohol-associated cirrhosis.

Ethical statement: The study was conducted in accordance with the latest update of the Declaration of Helsinki and the Regulations on Human Studies in Health Matters of the Mexican Republic.

The protocol was approved by the ethics committees of the Civil Hospitals of Guadalajara (010/20 and 00012) and the Ethics, Research and Biosafety Committee of the University Center for Health Sciences of the University of Guadalajara (22-96).

Declaration of interests: None.

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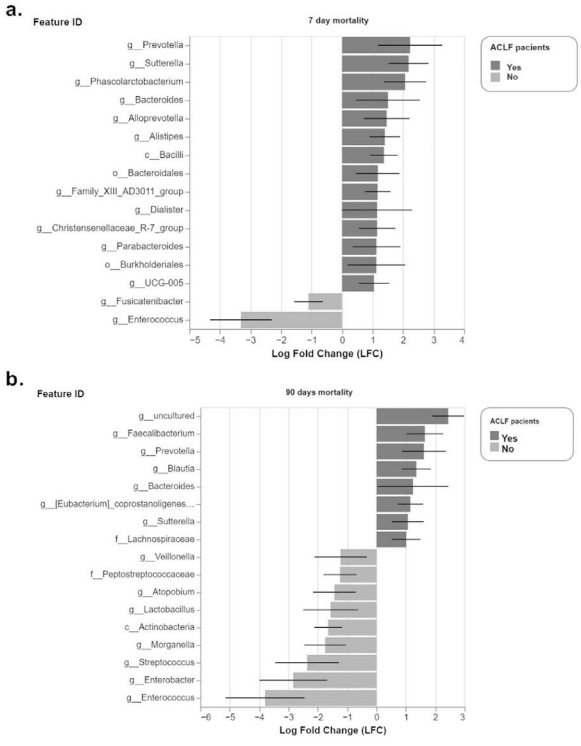

Figure 1. ***Analysis of fecal microbiota from patients with ACLF and its association with mortality.*** a) and b) Differential bacterial