TACE	19 (52.7)
Ablation	4 (11.1)
TACE + Ablation	4 (11.1)
Medical	1 (2.7)
Medical and radiological	3 (8.3)
Radiological and transplant	4 (11.1)
Transplant	1 (2.7)
Treatment response evaluation	
complete	4 (11.1)
Partial	9 (25)
Stable	11 (33.3)
Progression	10 (27.7)

BCLC, Barcelona Clinic Liver Cancer; HCC, Hepatocellular carcinoma; MAFLD, Metabolic dysfunction-associated fatty liver disease; MELD, Model for End-Stage Liver Disease; MELD-Na, Model for End-Stage Liver Disease-Sodium; TACE, Transarterial chemoembolization.

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Pirfenidone limits the progression of malignancy and slows the development of fibrosis in experimental hepatocarcinoma.

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Introduction and Objectives: Hepatocarcinoma (HCC) is the fourth cause of cancer death in Mexico, derived from fibrotic, metabolic and inflammatory alterations, modifiable by pirfenidone (PFD), which has shown beneficial effects at these levels. Our aim is to demonstrate the hepatoprotection of PFD in a model of HCC progression.

Materials and Patients: To evaluate the effect of PFD in a setting similar to that of patients at risk for HCC, we developed an experimental model of neoplastic progression, with damage induction for 9 weeks, followed by free progression of the disease. Male Fischer-344 strain rats (n=18) were divided into three groups: CTL: untreated control; HCCp: damage progression group (generated by administration of diethylnitrosamine (DEN) 50 mg/kg and 2-Acetaminofluorene (2AAF) 25 mg/kg weekly for 9 weeks and damage progression); and HCCp/PFD: damage progression group plus administration of PFD 300 mg/kg daily starting from week 9. The weight of the animals in the different study groups was recorded, and morphological and histopathological analyses of the liver were performed. H&E, Masson's Trichrome (MCT) and Sirius Red (SR) staining were performed, and GPC-3 and Ki-67 proteins were analyzed by immunohistochemistry. This was done in order to evaluate the presence and severity of fibrosis, malignancy and proliferation markers. Data were analyzed by ANOVA followed by Tukey's post-hoc tests to identify differences between study groups. Comparisons with p values ≤ 0.05 were considered significant.

Results: Treatment with PFD did not produce a difference in weight between the groups. However, it caused a tendency to decrease in body weight, net weight and relative liver weight. The morphological analysis of the liver of the HCCp/PFD group showed surface characteristics, coloration and consistency similar to the control, in addition to the evident attenuation in the progression of cancerous nodules, with a 34.02% reduction in the total tumor incidence. At the tissue level, PFD decreased the accumulation of extracellular matrix and collagen I and III deposition. The fibrotic bridges present in the HCCp/PFD group are incipient and of interstitial disposition, contrary to the intensity and tissue restriction shown in the HCCp group. In addition, dysplastic changes were limited after PFD treatment, with a decrease in hyperchromasia and nuclear pleomorphism, fewer cells with loss of polarity and nucleus/cytoplasm ratio, together with a decrease in necrotic cells, as well as a decrease in ductal reaction and destruction of portal triads compared to the damage group. Finally, in the HCCp/PFD group, the expression of both GPC-3 and Ki-67 was reduced, slowing tumor progression.

Conclusions: PFD administration had a hepatoprotective effect on tumor progression and slowing of fibrosis in our experimental model based on our results, we can conclude that PFD could work at the level of primary prevention of HCC in patients with chronic liver disease.

Ethical statement: Our research with animal models is governed by the highest ethical and animal welfare standards. The use of animals was scientifically justified, minimizing their suffering and providing them with adequate living conditions. All regulations were complied with, and transparency and accountability in research were continuously re-evaluated and maintained.

Declaration of interests: None.

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The neutrophil-to-lymphocyte ratio (NLR) can predict the presence of bacterial infections in hospitalized patients with decompensated cirrhosis.

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Introduction and Objectives: Liver cirrhosis can increase susceptibility to bacterial infections, which are often underestimated due to subtle or absent symptoms and the absence of additional biochemical markers. Early identification of these infections is limited in these patients. The objective is to determine the effectiveness of the Neutrophil-to-Lymphocyte Ratio (NLR) as a predictor of bacterial infections in hospitalized patients.

Materials and Patients: The aim of this retrospective, observational, analytical, cross-sectional study was to validate a prognostic index for patients diagnosed with liver cirrhosis and hospitalized from October 2023 to March 2024. The study included variables such as age, sex, etiology of liver disease, decompensation events leading to hospitalization, and biochemical data to calculate MELD, Child-Pugh, MELD-Na scores, and the degree of acute-on-chronic liver failure upon admission using the EASL-CLIF-ACLF, European Association for the Study of the Liver - Chronic Liver Failure Acute-on-Chronic Liver Failure score. Additionally, the presence of bacterial infection was determined through laboratory tests, imaging studies, and corresponding cultures. The Neutrophil-to-Lymphocyte Ratio was established by dividing the respective total cell counts. The study summarized the variables using descriptive statistics and constructed the area under the curve (AUC-ROC) with 95% confidence intervals. A p-value <0.01 was considered significant.

Results: A total of 183 patients were included in the study. There were 93 (50.8%) men, with an average age of 55.8 $\pm$ 10 years. The