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DEMOGRAPHIC, CLINICAL, AND BIOCHEMICAL CHARACTERISTICS IN PATIENTS WITH PRIMARY BILIARY CHOLANGITIS WITH AN UNFAVORABLE BIOCHEMICAL RESPONSE TO URSODEOXYCHOLIC ACID IN A TERTIARY CARE HOSPITAL

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Introduction and Objectives: Ursodeoxycholic acid (UDCA) is the standard treatment for primary biliary cholangitis (PBC), leading to improvement in liver biochemical parameters in most patients. However, up to one-third fail to achieve an adequate biochemical response, which has been associated with more rapid progression to cirrhosis and other complications. Identifying clinical and biochemical factors associated with an unfavorable response may help optimize therapeutic strategies and enable early risk stratification.

This study aimed to identify clinical, demographic, and biochemical characteristics associated with an inadequate biochemical response to UDCA in patients with PBC.

Materials and Methods: We conducted a retrospective, observational, analytical, case-control study with longitudinal follow-up in 73 patients with confirmed PBC treated with UDCA for at least 12 months at a tertiary care hospital (2022–2024). Biochemical response was evaluated using Paris II, Barcelona, and Globe criteria. Variables analyzed included age, sex, BMI, time since diagnosis, liver enzymes (ALP, AST, ALT), bilirubin, albumin, platelet count, liver fibrosis by elastography, autoantibody profiles (AMA, SP100, GP210), and metabolic comorbidities. Statistical analysis was performed using measures of central tendency and dispersion, as well as absolute and relative frequencies. Group comparisons were conducted using Student's t-test, with statistical significance set at $p < 0.05$.

Results: Among the 73 patients, 52% (n=38) were classified as non-responders. Non-responders were older (55 ± 9.2 vs. 51 ± 8.5 years; p=0.04) and had significantly higher baseline bilirubin (2.3 vs. 0.9 mg/dL; p<0.01) and ALP (361 vs. 240 U/L; p<0.01). Fibrosis on elastography was more frequent in non-responders (47% vs. 21%; p=0.01). Autoantibody positivity was also higher among non-responders (38% vs. 22%; p=0.05).

Conclusions: These results highlight key markers of unfavorable UDCA response and support the need for personalized monitoring and consideration of alternative therapies in high-risk PBC patients.

Conflict of interest: None

Clinical and Biochemical findings	Non-Responders (n = 38)	Responders (n = 35)	p-value
Mean age (years)	55 ± 9.2	51 ± 8.5	0.04
Total bilirubin (mg/dL)	2.3	0.9	<0.01
Alkaline phosphatase (U/L)	361	240	<0.01
Hepatic fibrosis (elastography)	n = 18 (47%)	n = 7 (21%)	0.01
Positive autoantibodies	n = 14 (38%)	n = 8 (22%)	0.05
Female sex	84%	86%	0.78
BMI (kg/m^2)	26.7 ± 4.3	25.9 ± 3.8	0.43
AST (U/L)	56 ± 12	54 ± 11	0.50
ALT (U/L)	49 ± 10	46 ± 9	0.28

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LIPID PROFILE-RELATED MARKERS AS PREDICTORS OF 28- AND 90-DAY MORTALITY IN PATIENTS WITH ALCOHOL-RELATED DECOMPENSATED CIRRHOSIS (DC) AND ACUTE-ON-CHRONIC LIVER FAILURE (ACLF): FIRST STUDY IN PATIENTS FROM WESTERN MEXICO

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Introduction and Objectives: DC and ACLF represent critical stages of alcohol-related chronic liver disease. Infections substantially increase mortality; therefore, the search for prognostic markers is important to predict clinical outcomes. Lipid profile has been shown to be associated with immunomodulatory processes and liver dysfunction; however, their role has been little investigated in patients from western Mexico. The objective was to evaluate the lipid profile in patients with alcohol-related DC and ACLF, and its association with infections and mortality.

Materials and Methods: Analytical cross-sectional study. Serum samples from 91 patients (DC=30, ACLF=61) were analyzed, plus 33 healthy controls (HC). Lipid profile quantification was performed using automated methods. ROC curves and Kaplan-Meier analyses were made using GraphPad. Approval number: 00012. No conflicts of interest are reported.

Results: ACLF group showed a significant decrease in triglycerides, LDL-c, and VLDL-c compared to DC and HC. Importantly, LDL-c and VLDL-c were effective to discriminate DC from ACLF (AUROC=0.73 and 0.71). The significant decrease in HDL-c with infection, and 28- and 90-day mortality in both groups DC and ACLF groups showed an AUROC of 0.72 for infection vs. non-infection, plus 0.98 and 0.75 for 28- and 90-day mortality, respectively. In ACLF, VLDL-c <8 mg/dL was associated with a 25% survival rate at 28 days.

Conclusions: Triglyceride, LDL-c, and VLDL-c levels progressively decline with the severity of cirrhosis. LDL, VLDL, and potentially HDL cholesterol are emerging as practical biomarkers for discriminating infections and mortality in DC and ACLF. These findings demonstrate the impact of lipid profiles on the prognosis and stratification of these patients.

Conflict of interest: None