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Metabolic Profile Alterations in the Progression of ALD- and NAFLD-Induced Liver Cirrhosis

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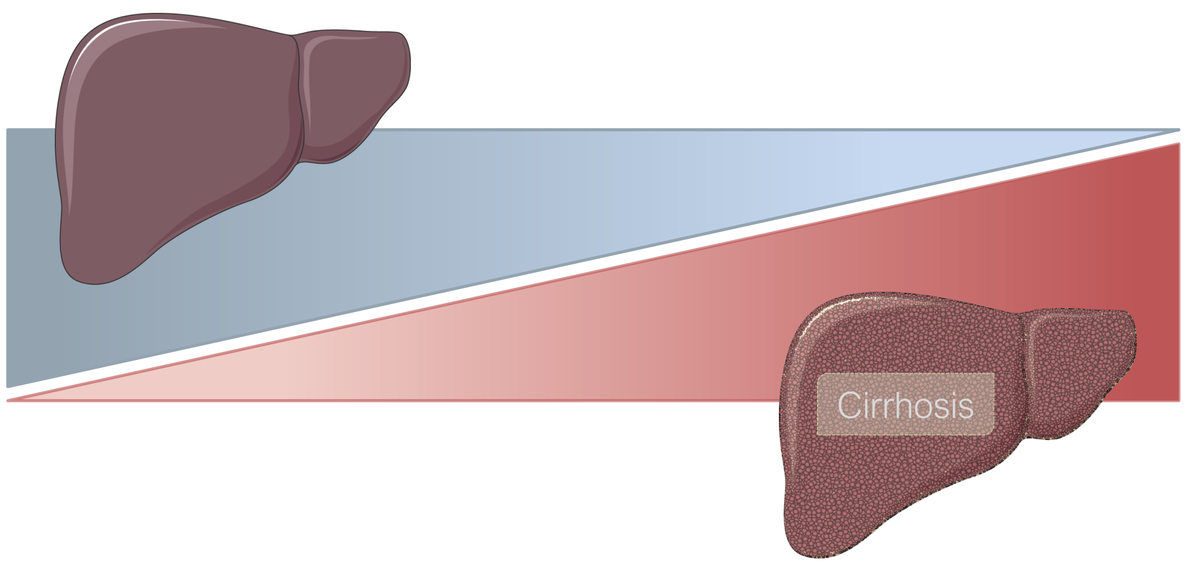
INTRODUCTION & AIM

This study aimed to characterize the metabolomic profile alterations in patients with liver cirrhosis resulting from alcoholic liver disease (ALD, n=31) and non-alcoholic fatty liver disease (NAFLD, n=8) compared to matched controls

METHOD

We conducted a study of 39 cirrhotic patients and age-, gender-, and BMI-matched controls (n=59) (p>0.05 for all), which included pre-cirrhotic patients and healthy individuals. Metabolite level in the blood serum was determined by high-performance liquid chromatography and tandem mass spectrometry. In order to find out the relationship between certain metabolites and the presence of liver cirrhosis, a binary logistic regression model was used to determine the odds ratio (OR) and 95% confidence interval (CI)



RESULTS & DISCUSSION

This study employed targeted metabolomic profiling to analyze 96 serum metabolites in patients with NAFLD and ALD. The analysis of the obtained data allowed us to identify a spectrum of metabolites associated with liver cirrhosis in the outcome of ALD and NAFLD ($p < 0.05$) and multidirectional shifts in the concentrations of biologically active molecules. Fibrosis progression was associated with significant metabolite alterations: increased levels of kynurenine (OR 2.93 [1.56-5.51]), acetyl-carnitine (OR 1.08 [1.01-1.17]), epinephrine OR 26.5 [5.7-123.4], dimethylarginine OR 20.2 [2.5-164.8], and methionine OR 1.10 [1.05-1.16], along with decreased indole-3-carboxaldehyde OR 0.04 [0.003-0.45]), serotonin (OR 0.038 [0.01-0.15]), norepinephrine (OR 0.25 [0.11-0.56]), taurine (OR 0.98 [0.97-0.99]), and carnosine OR 0.55 [0.34-0.90])

CONCLUSION

Comprehensive metabolomics studies remain challenging due to their high cost, leading most liver disease studies to focus on individual metabolite groups. In contrast, our targeted analysis of 96 serum metabolites provides a broad, multifactorial view of the metabolic alterations that occur in the context of ALD and NAFLD cirrhosis. The obtained data, which suggest enhanced oxidative stress, sympathetic activation, and mitochondrial dysfunction in progressive fibrosis, not only confirm previous findings but also uncover novel, previously invisible metabolic pathways and interactions