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Summary

IMPACT OF HEPATIC AND SYSTEMIC INFLAMMATION ON THE DYNAMIC COMPONENT OF PORTAL HYPERTENSION DURING DISEASE REGRESSION IN ALCOHOL-RELATED CIRRHOSIS

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Inflammation remains a critical determinant of the dynamic component of portal hypertension in both mouse models and abstinent patients with alcohol-related liver cirrhosis despite significant improvements during disease regression.

KEY POINTS

- In mouse models of cirrhosis regression, increased intrahepatic expression of inflammatory genes (Tnfα, Il6, Il1β, Cxcl1, Mcp1) was significantly associated with higher-than-expected portal pressure levels beyond what would be predicted by the degree of fibrosis alone.
- In 128 abstinent alcohol-related liver cirrhosis patients, longer abstinence duration correlated with lower HVPG, liver stiffness, IL-6, and CRP, yet patients with higher systemic inflammation (IL-6, CRP) demonstrated higher HVPG than predicted by liver stiffness measurement.
- Decreased complement factors C3c and C4 were associated with higher-than-expected HVPG in patients, with the negative C3c correlation present only in short-term abstinence (≤6 months) while systemic inflammation effects were primarily seen in long-term abstinence (>6 months).
- The study used linear modeling to separate static (fibrosis-related) from dynamic (inflammation-related) components of portal hypertension by comparing predicted versus measured portal pressure in both experimental systems.
- This work is relevant to researchers investigating portal hypertension mechanisms and clinicians managing patients with regressing liver disease who wish to understand factors beyond fibrosis that influence portal pressure.

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