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Summary

JAK2 INHIBITION ATTENUATES PORTAL HYPERTENSION IN METABOLIC DYSFUNCTION-ASSOCIATED LIVER

Shiyao Chen

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Ruxolitinib, a JAK2 inhibitor, significantly reduced portal pressure in a mouse model of MASLD-associated portal hypertension by modulating endothelial dysfunction and endothelial-to-mesenchymal transition.

KEY POINTS

- In mice fed a high-fat diet for 12 weeks, ruxolitinib treatment (60 mg/kg by gavage every two days for 3 weeks) significantly reduced directly measured portal pressure and the spleen-to-body weight ratio.
- Ruxolitinib treatment improved ALT, AST, and TC levels and decreased perisinusoidal and perivascular fibrosis on histopathological staining.
- Electron microscopy revealed restoration of endothelial fenestration, and ruxolitinib downregulated endothelial damage markers, mesenchymal cell markers, and EndMT-related regulatory factors, indicating alleviation of endothelial dysfunction.
- The study proposes that MASLD-associated portal hypertension may not depend solely on structural alterations like fibrosis but correlates with endothelial dysfunction, which JAK2 inhibition can target.
- The findings position ruxolitinib as a promising candidate for clinical management of non-cirrhotic portal hypertension, particularly in MASLD-associated portal hypertension.

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