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Summary

THE IMPORTANT ROLE OF HYALURONAN SYNTHASE 2 IN HEPATOCYTES IN OXALIPLATIN-INDUCED PORTAL HYPERTENSION

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Oxaliplatin-induced portal hypertension is mediated by MAPK/ERK pathway activation, leading to hepatocyte HAS2 upregulation and sinusoidal endothelial cell damage in both human patients and rat models.

KEY POINTS

- Liver samples from oxaliplatin-treated patients showed hepatic sinusoidal dilatation, and a six-week oxaliplatin treatment in rats induced portal hypertension with sinusoidal endothelial activation and perivenous monocyte-macrophage infiltration, though classical sinusoidal obstructive syndrome was absent.
- The ERK pathway and hyaluronan synthase 2 (HAS2) were upregulated after oxaliplatin treatment in both rats and patients, with the MAPK/ERK cascade identified as central to oxaliplatin-induced portal hypertension.
- Oxaliplatin-treated hepatocytes released mediators that activated liver sinusoidal endothelial cells, causing upregulation of von Willebrand factor and tissue factor, increased migration, and ERK pathway activation.
- Hyaluronic acid treatment increased sinusoidal endothelial cell migration, confirming an important role of HAS2 in oxaliplatin-induced pathology through a hepatocyte-to-endothelial cell signaling mechanism.
- This research addresses the increasing incidence of portal hypertension in oxaliplatin-treated cancer patients and may inform prevention strategies for this serious complication.

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