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Summary

ANTICOAGULATION ATTENUATES LIVER FIBROSIS VIA TARGETING NEUTROPHIL EXTRACELLULAR TRAPS

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Preclinical study in a mouse model of thioacetamide-induced liver fibrosis found that the anticoagulant edoxaban reduced collagen deposition, fibrosis-related gene expression, neutrophil infiltration, and markers of neutrophil extracellular traps (NETs).

KEY POINTS

- Mice treated with edoxaban (30 mg/kg daily for 3 weeks) showed significantly decreased hepatic collagen deposition compared to controls as measured by Masson's trichrome and Sirius Red morphometry.
- Edoxaban treatment significantly downregulated fibrosis-related genes Col1a1, Fn, and Acta2, and reduced inflammation-related gene expression.
- Differential gene expression analysis revealed enrichment in the NETs formation pathway in the treatment comparison.
- Edoxaban-treated mice showed significantly reduced neutrophil infiltration and lower expression of NETs markers including myeloperoxidase, neutrophil elastase, and citrullinated histone H3.
- The authors conclude that anticoagulation may alleviate liver fibrosis by targeting neutrophil extracellular traps, suggesting a novel therapeutic mechanism for liver fibrosis treatment.

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