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Summary

THERAPEUTIC TARGETING OF SECRETED OSTEOPONTIN ATTENUATES THE FIBROTIC PHENOTYPE IN EXPERIMENTAL CHRONIC PANCREATITIS

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Neutralizing antibody against secreted osteopontin significantly reduced pancreatic fibrosis, stellate cell activation, and pro-fibrotic gene expression in a mouse model of chronic pancreatitis.

KEY POINTS

- Anti-osteopontin antibody treatment (200µg, 3×/week) significantly decreased serum osteopontin levels (Δ = -98514, p = 0.0209) and reduced collagen deposition on histology (Δ = -1.648, p = 0.0064) in mice with cerulein-ethanol-induced chronic pancreatitis.
- Pancreatic stellate cell activation was significantly reduced, with decreased α-SMA expression by immunohistochemistry (Δ = -3.636, p = 0.0004) and by qPCR (Δ = -72.91, p = 0.0028).
- Pro-fibrotic gene expression was significantly downregulated, including Col1a1 (Δ = -6.467, p = 0.0077), Col3a1 (Δ = -5.097, p = 0.0026), CTGF (Δ = -3.112, p = 0.0147), and MMP2 (Δ = -2.659, p = 0.0500).
- Therapeutic benefit was observed in both early (3-week preventive treatment) and established (6-week therapeutic treatment) chronic pancreatitis models.
- The study identifies secreted osteopontin as a potential therapeutic target in chronic pancreatitis, for which no curative therapy currently exists.

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