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Summary

INHIBITION OF PANCREATIC ENDOTHELIAL CYCLOOXYGENASE-2 AMELIORATED SEVERE ACUTE PANCREATITIS

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Mechanistic study demonstrates that COX-2 is specifically expressed in pancreatic microvascular endothelium during severe acute pancreatitis, and its inhibition with parecoxib or endothelial-specific genetic knockout improves survival, reduces pancreatic necrosis and hemorrhage, and protects against neutrophil recruitment, endothelial senescence, mitochondrial dysfunction, and microvascular thrombosis.

KEY POINTS

- Parecoxib intervention or genetic ablation of endothelial ptgs2 significantly improved survival rates and attenuated pancreatic necrosis, hemorrhage and inflammatory cell infiltration in rat and mouse models of severe acute pancreatitis.
- COX-2 was specifically expressed in pancreatic microvascular endothelia, not in acinar cells, during severe acute pancreatitis, while normal pancreas does not express COX-2.
- COX-2 inhibition reduced neutrophil recruitment, decreased expression of adhesion molecules VCAM-1 and ICAM-1 and chemokines CXCL1, CXCL2 and CXCL8, and suppressed the NF-κB pathway.
- COX-2 inhibition protected endothelia from TNF-α-induced dysfunction, mitigating markers of cellular senescence including p21, p16, p53 and phosphorylated γ-H2A.X, and preserved mitochondrial membrane potential by reducing mitochondrial permeability transition pore opening.
- Microvascular thrombosis and parenchymal necrosis were markedly reduced by COX-2 inhibition.
- The authors conclude that down-regulation of COX-2 expression with COX-2 inhibitors as early as possible may be beneficial for prevention and treatment of severe acute pancreatitis.

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