

Gut Barrier Dysfunction in Experimental Acute Pancreatitis

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Abstract

Bacterial infections, frequently caused by bacteria of enteric origin, are often seen during the progression of severe acute pancreatitis, concomitant with the potential development of multiple organ dysfunction. In the present paper, the complex mechanisms of gut barrier dysfunction and gut origin sepsis in acute pancreatitis are discussed.

The individual parts of the gut barrier, e.g. the immunological, bacteriological and morphological (mucus, epithelium, endothelium and interstitium) components, seem to interact and depend on each other. Pancreatitis-induced hypovolaemia due to endothelial barrier leakage and gut arteriovenous shunting causes intestinal ischaemia and reperfusion injury with concomitant gut barrier dysfunction. Gut endothelial barrier dysfunction probably plays a central role. Potential molecular mechanisms could, among others, be associated with alterations in intracellular signal transduction, intercellular signalling and expression of adhesion molecules on endothelial cells.

The mechanisms underlying gut barrier dysfunction in acute pancreatitis are thus complex and still not fully elucidated. Knowledge about the regulating events will probably allow future pharmacological therapy for prevention and treatment of the severe complications of acute pancreatitis, including gut barrier dysfunction.

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