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Factors involved in the pathogenesis of the acute pancreatitis

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Abstract
Pathophysiological and molecular research have marked the understanding of the primary events taking place in triggering acute pancreatitis, although the early diagnosis of pancreas diseases in general, continues to be a source of frustration in modern medicine. This presents the news about pathogenesis (co-localization theory, auto-activation theory of the tripsynogen), location of early events (acinar pancreatic cells which are the "key" involved: muscarinic receptors, acinar membrane, role of ionized calcium, the phenomenon of apoptosis), extracellular events in initiation of acute pancreatitis with the granting of a central place to enzyme activation and systemic inflammatory response. Aspects of early microvascular changes, disturbances of ischemia-reperfusion and systemic microvascular abnormalities are so important that justifies therapeutic concept of microcirculatory protection. Participation of monocyte/macrophage system, excessive activation of leukocytes that involving activation and release of lysosomal enzymes and oxygen free radicals associated with ischemia-reperfusion mechanism are defining for pathogenesis of acute pancreatitis.
Key words: pathogenic theory, enzymatic activation, local injury, muscarinic receptors, acinar membrane, cytokines, ionized calcium, ischemia-reperfusion, oxidative stress

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