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Ulinastatin promotes T lymphocyte apoptosis in rats with severe acute pancreatitis via mitochondrial pathways

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ABSTRACT. We explored the influence of ulinastatin on apoptosis of T lymphocytes in rats with severe acute pancreatitis (SAP) and the effect of ulinastatin on mitochondrial apoptosis pathways in spleen lymphocytes. Thirty-six Wistar rats were randomly divided into three groups (N = 12): a sham operated group, a SAP group, and an ulinastatin-treated SAP group. The SAP model was established by injecting 5% sodium taurocholate into the intrapancreatobiliary duct. Study rats were sacrificed after 24 h, and splenic lymphocytes were then collected. CD_4^+ and CD_8^+ T lymphocytes were labeled by direct immune fluorescence assays; the percentage of apoptotic cells, mitochondrial membrane potential levels, and mitochondria permeability transition pore opening levels were measured by flow cytometry. In the ulinastatin-treated SAP group, the ratio of CD_4^+/CD_8^+ T lymphocytes was significantly higher than that in the SAP group, and the apoptosis percentage of CD_4^+ T lymphocytes was significantly decreased. The percentage of lymphocytes with an abnormal opening of the mitochondrial permeability transition pore and lymphocytes

with decreased mitochondrial membrane potential in the ulinastatin-treated SAP group were significantly lower than that in the SAP group. Ulinastatin can directly enhance immunological function and attenuate immune suppression in SAP rats through inhibiting the apoptosis of CD_4^+ T lymphocytes. These study findings demonstrate that therapeutic effects may occur through inhibiting the apoptosis induced by mitochondrial signaling pathways.

Key words: Ulinastatin; Severe acute pancreatitis; Immune suppression; Cell apoptosis; Mitochondrion