Early Gut Barrier Dysfunction in Patients with Severe Acute Pancreatitis Attenuated by Continuous Blood Purification Treatment

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ABSTRACT

Objective Early gut barrier dysfunction is one of the critical pathophysiologic disorders in patients with severe acute pancreatitis (SAP) and plays important role in the development of sepsis and multiple organ failure. The aim of this study was to investigate the effect of CBP on gut mucosal dysfunction. Methods Patients with SAP were randomized to receive 24h of CVVH (n=33) or without CVVH (n=30). Blood sample were taken from the patients at 0, 6, 12, 24 h during CVVH therapy or intensive care unit resuscitative therapy, The levels of serum DAO and endotoxin were measured respectively by a spectrophotometric assay. The epithelial permeability, TER and F-actin rearrangement were assessed using cultured Caco-2 cell monolayer; iNOS mRNA expression and NO production in monolayer were detected by RT-PCR and spectrophotography, respectively.

Results Patients with SAP had increased levels of serum DAO, endotoxin and epithelial permeability when compared to normal controls, and the increase was more pronounced in patients with organ dysfunction (P<0.01). Transepithelial electrical resistance (TER) decreased in patients with SAP especially in patients with organ dysfunction (P<0.05). F-actin rearrangement, cell-cell junction loosed and iNOS mRNA upregulation was found in all patients. After CBP treatment, APACHE II score and SOFA score improved significantly, levels of serum DAO, endotoxin and epithelial permeability decreased, and TER increased in all patients (P<0.05); CBP also significantly attenuated reorganization of actin and downregulated iNOS mRNA expression and NO production (P<0.05). Conclusions Early gut barrier dysfunction is present in patients with SAP and may be correlate with the disease severity. CBP can not only improve the general conditions but also effectively improve gut barrier dysfunction. The beneficial effect of CBP on gut barrier function is associated with improvement of cytoskeletal instability, at least partly, by downregulating iNOS through remove excess pro-inflammatory factors.