

FUNDAMENTAL SCIENCES

ORAL PRESENTATIONS

250. TREATMENT OPTIONS IN SYSTEMIC INFLAMMATORY RESPONSE SYNDROME

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Introduction: Patients subject to major surgery, suffering sepsis, major trauma, or following cardiopulmonary bypass exhibit an ‘acute phase’ inflammatory response. When the inflammatory response becomes uncontrolled, a Systemic Inflammatory Response Syndrome (SIRS) ensues. The etiology of systemic inflammatory response syndrome (SIRS) is broad and includes infectious and noninfectious conditions, surgical procedures, trauma, medications, and therapies. There are 3 stages in development of SIRS. In the first stage, following an insult, cytokines are produced at the site. In the second stage, small quantities of local cytokines are released into the circulation, improving the local response. This acute phase response is typically well controlled by the release of endogenous antagonists, the goal being homeostasis. In the final stage if homeostasis is not restored, a significant systemic reaction occurs.

Materials and methods: Collecting data and research results from published cohort studies, double blinded placebo RCT, prospective survey with emphasis on intensive care unit patients.

Discussion results: The goal of this project was to establish the effectiveness of new treatment strategies in treatment of SIRS. Mainly these are based on physiological aspect and pharmacological one. From physiological treatment can be mentioned Early Goal-directed therapy based on optimising cardiac pre-load and contractility, delivery of necessary oxygen and insulin therapy. Van den Berghe et al showed that insulin therapy reduces by 34% the in-hospital mortality and incidence of fatal infection in diabetic and non-diabetic critically ill patients.

Pharmacological therapies are based on treatment with monoclonal antibodies, activated Protein C, corticosteroids, antioxidants, serine protease inhibitors as well as filtration and adsorptive therapies (activated Charcoal; Immobilized antibody systems). Infusion of Activated Protein C reduces the absolute risk in mortality to 6.1%.Corticosteroids showed a decrease of pro-inflammatory cytokines(IL-6, TNF α , and E-selectin) and increase of anti-inflammatory cytokines(IL-10). However, the negative side in using corticosteroids are the pulmonary dysfunction and prolonged time to extubation, as well as a lot of exclusion criteria for using them. Monoclonal antibodies HA-1A (human monoclonal IgM antibody) showed a reduction from 49% to 30% in 28 day mortality in patients with gram-negative bacteremia. The most efficient therapies are filtration and adsorptive therapies. Immobilized antibody systems assure near-complete removal of TNF α from human plasma as well as reduction to 70% of cytokine levels (IL-1 β , IL-1Ra, IL-6, IL-8 and TNF α). Activated Charcoal absorb almost 100% of plasma LPS, IL-Ra, IL-1 β , IL-8, IL-1a and IFN- γ and 40% of TNF α . In 5 of the 8 adult patients in ICU, reported resolution of sepsis.

Conclusion: The true incidence of systemic inflammatory response syndrome (SIRS) is unknown. However, the occurrence of SIRS was characterized by a significantly elevated release of IL-6 and IL-8, with subsequent increase in the leukocyte count, C-reactive protein (CRP), and procalcitonin. Prognosis depends on the etiologic source of SIRS, as well as on Associated comorbidities. Strategies targeting purported triggers, early mediators and even physiological responses to inflammation have largely been unsuccessful to date. Some of the most prominent areas of research relates to the initiators and modulation of the pro-inflammatory cascade, methods of extracting pro-inflammatory cytokines and how genetic polymorphisms may influences the natural history of SIRS in patients.. However, some encouraging data exists with adsorptive strategies to attenuate the hyper-cytokinaemia Associated with SIRS. Activated Charcoal and Polymyxin B hemofiltration systems have promising features in this respect, but we look forward to the generation of more exhaustive and definitive research in the future.

Key Words: Systemic Inflammatory Response Syndrome, Cytokines, Treatment Strategies.

251. THE ROLE OF VASCULAR ENDOTHELIAL GROWTH FACTORS AND NEOVASCULARIZATION IN THE DEVELOPMENT OF RECURRENT VARICOSE VEINS AFTER SURGERY.

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Introduction: Varicose disease remains an actual pathology due to high incidence, possible complications and also damage to the quality of life. The basic treatment of varicose veins is surgical one. According to retrospective observations, about 35% of patients over 2-5 years after the surgery develop recurrent varicose veins with pathologic reflux at the sapheno-femoral junction (SFJ). Relapsed venous reflux at SFJ can lead to severe venous insufficiency and recurrent venous disease.

Materials and methods: The study included 26 patients with venous disease in the basin of great saphenous vein in both legs, taking part to class C2-C3, according to CEAP classification (Clinical-Etiology-Anatomy-Pathophysiology). To the patients with a defect in the SFJ and a great saphenous vein reflux, confirmed by Doppler Duplex scanning, was performed the crossectomy and striping in combination with mini-phlebectomy to remove the dilated veins. In all patients were performed both methods of prevention of the phenomenon of neovascularization: anatomical barrier and selective crossectomy, either on the left or right leg. Vascular endothelial growth factors (VEGF-C/VEGF-D) were determined by immunohistochemical methods through monoclonal antibodies.

Results: One month later after the surgery have been effectuated Duplex scanning to all patients, for the control of performed crossectomy, which demonstrated a lack of residual affluents. One year after the surgery in 15.2% of patients were detected visible varices at the thigh, while the phenomenon of neovascularization, confirmed by Doppler Duplex scanning, was detected in 22.9% patients. After two years the frequency of phenomenon of neovascularization was 34.5% (23% selective crossectomy and anatomical barrier-11.5%). In these patients, plasma levels of VEGF-C/VEGF-D was increased,