IS-88 Pelvic Vasculer Dysfunction in Patients with Chlamydial Pelvic Inflammatory Disease

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[Objective] Chlamydia trachomatis (C.t.) is a common cause of pelvic inflammatory disease (PID). Another Chlamydia, C. pneumoniae, has been recently indicated as an etiologic agent of coronary artery disease. In this pathogenic process, infected monocyte/macrophage in chlamydial pneumonia plays pivotal role, transfers the pathogen to coronary vascular endothelium and triggers atherosclerosis. Following this analogy, C. t. may cause pelvic vascular abnormality. In this study we tried to elucidate pelvic endothelial dysfunction as an early event of atherogenesis of patients with chlamydial PID. [Methods] To evaluate endothelial dysfunction, we have established measurement of mean diameter (Md) of vessel lumen from real-time recording throughout cardiac cycles. We examined the femoral artery at rest, and during reactive hyperemia in two groups: 10 controls without vascular risk factors and 10 chlamydial PID patients. [Results] The baseline Md in the control and chlamydia groups were 7.02 ± 0.54 and 7.32 ± 0.44, respectively. During reactive hyperemia the Md increased to 7.85 ± 0.32mm in the control group, which indicates flow-mediated dilatation in all observed arteries. In chlamydia group, reactive dilatation (Md = 7.66 ± 0.37 mm) was absent ($p < 0.001$). [Conclusion] Using this dynamic method, we have detected presence of the endothelial dysfunction in chlamydial PID patients.

IS-89 Gender Differences of Sensitivity to A Thromboxane A_2 Mimetic U46619 in Isolated Rat Femoral Arteries

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[Objective] We have reported that sensitivity to a thromboxane A_2 mimetic U46619 decreases with age in rat femoral arteries and the sensitivity is affected by fetal and maternal environment. The aim of this study is to investigate the mechanisms of gender difference of the sensitivity in isolated rat femoral arteries. [Methods] Femoral arteries of male and female adult Wistar rats were dissected after overdose pentobarbitone administration (ip), and mouted on wire-myograph as a ring preparation. After setting a physiological pressure, tensions were measured as mN/mm under isometric condition. Cumulative concentration doses of U46619 ($1 \times 10^{-9} - 5 \times 10^{-6}$ M) were examined with or without 0.1 mM L-NAME (nitric oxide synthase inhibitor), 10μM indomethacin (cyclooxygenase inhibitor) or 25 mM potassium (inhibiting endothelium-derived hyperpolarizing factor). pEC_{50} ($-\log M$) of each response-curve was calculated. The values were expressed as mean ± SEM and compared by Mann-Whitney U-test. Significancy is accepted when $p < 0.05$. [Results] Female showed higher sensitivity to U46619 than male (6.79 0.12 vs 6.36 0.09). Indomethacin reduced both sensitivity and abolished the gender difference (male, 6.380.16 vs female, 6.290.15). L-NAME and 25 mM potassium also abolished the difference. [Conclusion] Gender difference of sensitivity to U46619 is enhanced in female and mediated by enthothelial dilatory system.

IS-90 Fetal Growth Retardation May Result from Apoptosis

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Objectives: Signals that trigger cells to undergo apoptosis utilize shared pathways, whether the initiating event is receptor mediated or a response to an exogenous stimulus. In 1997 Smith et al. described increased apoptosis in placentae from third trimester pregnancies complicated by intrauterine growth restriction. Hypoxic stress is a strong stimulus for intrauterine growth restriction and severe cases of preterm infants will show significant changes in Doppler flow velocities. Our purpose is to investigate the possible role for apoptosis in the pathophysiological mechanism of intrauterine growth restriction in preterm infants. **Methods:** Placental samples were obtained from preterm deliveries between 28 and 29 weeks of gestational age with and without intrauterine growth retardation, complicated with severe Doppler flow results. All infants were delivered by cesarean section. Light microscopy was used to describe ramification and vascularisation according to the gestational age, TUNEL (terminal deoxynucleotidyl transferase-mediated deoxyuridine phosphate nick end-labeling) staining were used to confirm the occurrence of apoptosis. **Results:** We found a strong expression of apoptosis in placentae from preterm infants with intrauterine growth restriction complicated with severe Doppler flow compared with placentae of normal developed preterm infants. **Conclusions:** These results suggest that apoptosis may play a key-role in the pathophysiological mechanism of intrauterine growth restriction. In this study we are now analyzing the details of receptor-mediated pathways, the Bcl-2 regulators and the caspases and substrates involved in the placental apoptosis of preterm infants.