PB1955 | Tripeptides Affected Hemostas in Various Pathologies

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Background: For modern medicine and pharmacology, the most important are the problems of finding new drugs that are safe and without side effects. The use of drugs based on regulatory peptides can significantly help in solving these problems. However, until recently, few such peptides have been found.
Aims: To study the effect of small regulatory peptides, which are fragments of many natures of compounds, such as collagen, neurohormones, vasopressin, oxytocin, luliberin on platelet aggregation. Data on the effect of tripeptides on aggregation are presented on immobilization stress models and on the development of experimental inflammation.
Methods: Experiments were carried out on white mongrel male rats weighing 180-200 g body. A pro-inflammatory agent was used to simulate experimental peritonitis - 40% sodium thioglyaolate solution 4g / kg, administered intraperitoneally. We are using peptide Pro-Gly-Pro (PGP) fragment of collagen in dose 3.7 mkg/kg intramuscularly for prophylactic and treatment inflammation. Platelet aggregation (PA) was recorded on a aggregometer. Inductor aggregation - ADP -10 mM. In the next series of experiments, animals were subjected to immobilization stress for 60 minutes and determined the effect of C-terminal tripeptides of neurohypophysial hormones on PA. All experiments were performed according to the documents of the European Science Foundation.
Results: The development of inflammation causes an increase in PA with a maximum effect 2 hours after administration of thioglycolate. Prophylactic peptide administration significantly reduced PA, whereas peptide did not show any statistically significant results on the background of an already developed inflammation. C-terminal tripeptides of neurohypophysial hormones Pro-Arg-Gly and Pro-Leu-Gly also had a protective effect on PA against immobilization preventing the development of a prothrombotic stress effect.
Conclusions: Thus, we can note that the tripeptide of various origin can have a prophylactic protective effect, reducing PA in inflammation and immobilization stress.

PB1956 | Expression Profiles of the Internal Jugular and Saphenous Veins: Focus on Hemostasis Genes

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Background: The ample heterogeneity of veins, related to their specific role and position should modulate the transcriptional profile of anticoagulants and procoagulant genes which contribute to the "in situ" hemostasis balance, and could modulate the ability of the individual vascular bed to counteract prothrombotic stimuli. The internal jugular vein (IJV) has a major role in cerebral venous return towards the heart, and differs from saphenous vein (SV) for morphological and hemodynamic characteristics.
Aims: To compare the IJV and SV transcriptomes, which could participate in venous bed specificity and vulnerability to thrombus formation.
Methods: Microarray-based transcriptome analysis in wall and valve specimens from IJV and SV collected during surgical reconstruction of IJV by patch angioplasty in multiple sclerosis patients with impaired brain outflow. Multiplex antigenic assay in paired jugular and peripheral plasma samples.
Results: 3375 differentially expressed transcripts in walls defined distinct venous expression profiles. The "complement and coagulation cascade" emerged among the enriched pathways. In IJV, upregulation of genes for coagulation inhibitors (TFPI, PROS1), activated protein C pathway receptors (THBD, PROCR), fibrinolysis activators (PLAT, PLAUR), and downregulation of the fibrinolysis inhibitor (SERPINE1) and of contact/amplification pathway genes (F11, F12), would be compatible with a thromboprotective profile in respect to SV. Further, in SV valve the prothrombinase complex genes (F5, F2) were up-regulated and the VWF showed the highest expression. Differential expression of several VWF regulators (ABO, ST3GAL4, SCARA5, CLEC4M) was also observed. Among other differentially expressed hemostasis-related genes, heparanase (HPSE)/heparanase inhibitor (HPSE2) were up-/down-regulated in IJV, which might support procoagulant features and disease conditions. For several proteins, encoded by differentially expressed genes, the jugular plasma levels were lower and highly correlated with peripheral levels.
Conclusions: The IJV and SV rely on differential expression of many hemostasis and hemostasis-related genes to balance local hemostasis, potentially related to differences in vulnerability to thrombosis.