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AICE Associazione Italiana dei Centri Emofilia

HDTM Hrvatsko Društvo za Transfuzijsku Medicinu

SETS Sociedad Española de Transfusión Sanguínea y Terapia Celular

SISET Società Italiana per lo Studio dell'Emostasi e della Trombosi

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ABSTRACT BOOK

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cells. In fact, activated cells were characterized by a) increased platelet deposition and fibrin formation, and b) thrombin generation, compared with unstimulated cells. Activation of TNFα-treated endothelial cells was also confirmed by assessing the upregulation of CD106 and TF.

Conclusion. Our results support the use of ECFCs for patient-specific in vitro investigation of ED, thus paving the way for their application in personalized modeling of vascular diseases.

PO036 - Hemostasis gene expression of the internal jugular and saphenous veins

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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. The differential vulnerability to thrombus formation between IJV and SV is potentially reflected in mRNA profiles.

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. 3,375 differentially expressed transcripts in walls defined distinct venous expression profiles. The "complement and coagulation cascade" emerged among the enriched pathways (P<1.0E-4). 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, PAI-1) and of contact/amplification pathway genes (F11, F12), would be compatible with a thromboprotective profile in respect to SV. In the SV valve the VWF showed the highest expression, and differential expression of several VWF regulators (ST3GAL4, SCARA5, CLEC4M) was observed.

For several proteins (soluble Thrombomodulin, Protein S, PAI-1), encoded by differentially expressed genes, the jugular plasma levels were lower (p=0.006, p=0.011, p=0.039, respectively) and highly correlated with peripheral levels (p<0.001).

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.