P.025: ABSENCE OF SYNDECAN-1 RESULTS IN INCREASED INFARCT HEALING AND DEPRESSED CARDIAC FUNCTION AFTER ACUTE MYOCARDIAL INFARCTION

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VALIDITY OF THE TENSIOSCINIC DEVICE TO MEASURE ARTERIAL STIFFNESS IN PATIENTS ON HEMODIALYSIS

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Assessment of arterial stiffness in dialysis patients has prognostic significance. The TensiClinic device uses oscillometrically obtained wave forms to calculate pulse wave velocity (PWV) and augmentation index (AI). Our objective was the assessment of the validity of measurements of TensiClinic (PWV1, AI1) compared to that of the validated Pulsheen tonometer (PWV2, AI2). We measured PWV and AI in duplicate, before and after hemodialysis, in 94 hemodialysis patients. Reliability of a given device was assessed by calculating the standard deviation of the difference (SDD) between the first and second measurement. Validity of TensiClinic was evaluated by comparing its results to that obtained by the PulsePen device, using correlation analysis and Bland-Altman plots.

Pre- and postdialysis SDD for AI P and AI T were 0.87 ± 0.12% and 1.419 ± 0.18% m/s, respectively. Pre- and postdialysis SDD for A0 and AI were 0.87 ± 0.54% and 1.81 ± 0.74%, and 0.79 ± 0.40% and 3.54 ± 2.69%. Mean predialysis PWV was 10.95 ± 2.89 m/s and PWV2 was 9.07 ± 2.36 m/s. Postdialysis PWV was 11.59 ± 2.92 m/s and PWV2 was 10.37 ± 3.24 m/s. Significant correlation was found between predialysis PWV and PWV2 (r = 0.28, p < 0.05). After dialysis, correlation was not significant (r = 0.16). Mean predialysis AI0 was 23.12 ± 12.27% and AI2 21.77 ± 10.04%. Postdialysis AI0 and AI2 were 22.01 ± 12.54% and 4.36 ± 37.81%, respectively. Significant correlation was found between pre- and postdialysis AI0 and AI2, r = 0.40 and 0.26, respectively.

PWV and AI measurements with PulsePen are more reproducible compared to TensiClinic. Poor correlation between the results obtained by the two devices may limit the use of TensiClinic in hemodialysis patients.

THE RELATIONSHIP BETWEEN PERIPHERAL ARTERY PULSE WAVE VELOCITY AND VASODILATOR FUNCTION IN HYPERTENSIVE PATIENTS

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Pulse wave velocity is commonly employed as a summary descriptor of physical arterial characteristics and can be measured rapidly using several commercially available devices. While brachial artery vasodilator function remains a more established index of endothelial health, its potential for assessment of arterial stiffness and both are predictors of cardiovascular events, no method is currently available to measure local PWV non-invasively. The common carotid artery (CCA) of 4 young subjects were measured with multiple M-line ultrasound, resulting in 14 distension waveforms spaced over 17 mm. PWV was determined by applying linear regression to the foot of the distension waveforms and the corresponding echo line position. The PWV was accepted for further analysis if the correlation coefficient of both parameters was above 0.95.

Results: The local PWV measurement method had a good intra-subject coefficient of variation (CV) of <3%. The average PWV was 3.5 ± 0.7 m/s with an inter-subject CV of 20%. One subject, with high blood pressure (140/90 mmHg), had a PWV of 4.6 ± 0.2 m/s.

Conclusion: Local PWV can be measured with good precision using multiple M-line ultrasound. The inter-subject variation (CV > 20%) exceeds the intra-subject variation (CV ≤ 5%), enabling distinction between individual differences. More measurements are required to evaluate the accuracy of the local PWV method in vivo.

ABSENCE OF SYNDECAN-1 RESULTS IN INCREASED INFARCT HEALING AND DEPRESSED CARDIAC FUNCTION AFTER ACUTE MYOCARDIAL INFARCTION

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Syndecan-1 (Synd1) has been implicated in angiogenesis during tumor formation and wound healing. To investigate the unknown role of Synd1 in cardiac healing, remodeling and function, acute myocardial infarction (AMI) was performed in Synd1-deficient (Synd1 KO) and wild type (WT) mice.

Cardiac function and structure did not differ between sham-operated WT and Synd1 KO mice at 14 days. However, significantly less necrotic cardiomyocytes remained in the infarct of Synd1 KO versus WT mice (5% necrotic cardiomyocytes in Synd1 KO and wild type (WT) mice). Cardiac function and structure did not differ between sham-operated WT and Synd1 KO mice at 14 days. Increased number of CD45-staining leucocytes and CD31 staining capillaries in Synd1 KO versus WT mice (CD45; 1068 ± 283 in WT vs 2076 ± 193 in KO; CD31; 138 ± 5 in WT vs 233 ± 19 in KO, n = 5, p < 0.05) suggesting accelerated infarct healing in absence of Synd1 at 14 days. Increased number of CD45-staining leucocytes and CD31 staining capillaries in Synd1 KO versus WT mice (CD45; 1068 ± 283 in WT vs 2076 ± 193 in KO; CD31; 138 ± 5 in WT vs 233 ± 19 in KO, n = 5, p < 0.05) confirmed depressed cardiac function in Synd1 KO mice, resulting in a 70% higher lung to body weight index in Synd1 KO as compared to WT mice.

In conclusion, absence of Synd1 results in increased inflammation, angiogenesis and accelerated healing after AMI, leading to increased infarct healing and decreased cardiac function.

A PIVOTAL ROLE OF THE ANTI-ANGIOGENETIC FACTOR THROMBOSPONDIN-2 IN THE PROGRESSION OF CARDIAC HYPERTROPHY TO FAILURE.

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Introduction: Thrombospondin-2 has been implicated in angiogenesis during wound healing and tumour formation, but its role in cardiac hypertrophy and function during cardiac overload is unknown.

Methodology: First, micro-array analysis of cardiac biopsies taken in hypertensive renin-overexpressing rats at the compensated phase (10 weeks) revealed that increased cardiac expression of the matricellular protein TSP2 identified the failure-prone hearts. Subsequently, thrombospondin-2 (TSP2) knockout and their wild type littermates were subjected to Angiotensin II infusion (0.5 mg/g/day). After detailed histomorphometric analysis, hearts were taken out and prepared for further histological and molecular analysis.

Results: TSP2 immunostaining in WT mice revealed increased expression in areas of active remodeling after AngII infusion, with a maximum at 4 and 7 days and declining at 14 days. When TSP2 knockout (KO) mice were subjected to Ang II infusion, 70% of TSP2 KO mice succumbed due to fatal cardiac rupture. The surviving TSP2 KO mice showed severe cardiac failure, as indicated by decreased fractional shortening and increased diastolic dimensions, whereas cardiac rupture, dilatation or dysfunction were absent in TSP2 WT mice. Ultrastructural analysis of Angii treated TSP2-KO hearts revealed oedema and disruption of the extracellular matrix, associated with increased activity of the collagen degrading enzymes MMP-2 and MMP-9.

Conclusion: The placental-derived matricellular protein TSP2 and SPARC during cardiac overload or ischemia protects against adverse cardiac remodeling, thereby preventing cardiac dilatation, failure or fatal cardiac rupture.