Impact of plasma protein deposition on Staphylococcus aureus adhesion to tissues used for RVOT reconstruction

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Introduction: Congenital heart disease encompasses anatomical defects, such as malformations on the right ventricular outflow track (RVOT) that can be surgically corrected using a cryopreserved pulmonary homograft (CPH) in first line and as an alternative xenografts such as bovine jugular vein (BJV) conduits which are also available stent-mounted for percutaneous catheter interventions. Although a good therapeutic alternative several clinical studies report an increased risk of stenosis, immunogenicity, thrombus formation or even infective endocarditis of BJV xenografts. In this study we investigate the role of plasma protein deposition on Staphylococcus aureus adhesion to several tissues used for RVOT reconstruction.

Methods: Similar tissue pieces prepared as for clinical use (CPH, BJV conduit, pericardium patch and decellularized pericardium patch) were incubated for 2h at 37°C with 30 μg/ml of fluorescently labelled Fibrinogen or 50 μg/ml of fluorescently labelled immunoglobulin (obtained from frozen pooled human plasma) resuspended in 200g/L of human albumin. Then, S. aureus 8325-4 (fluorescently labelled) adhesion to the same tissues was assessed under flow conditions (10 min at 1000s⁻¹) after 2h of incubation at 37°C with PBS or frozen human pooled plasma using a micro-parallel flow chamber. Protein deposition and bacterial adhesion was assessed and quantified by fluorescence microscopy.

Results: Pericardium patch presented significant higher protein deposition (P < 0.05) compared to BJV and CPH wall and leaflet. Although not significant, there is a trend to higher fibrinogen deposition on BJV tissue compared to CPH. On the opposite, CPH presented higher (P < 0.05) immunoglobulin deposition compared to BJV tissue. After incubation with frozen human pooled plasma S. aureus adhesion under flow conditions increased (P < 0.05) compared PBS.

Conclusions: Our results indicate that plasma protein deposition modulate S. aureus adhesion to the tested tissues. Plasma protein adhesion might also play an important role after tissue implantation in vivo and may contribute to the onset of inflammation and infection as a clinical complication. In the future we will focus on the impact of plasma protein deposition on platelet adhesion and thrombus formation, endothelial cell and inflammatory cells adhesion and how these can modulate associated S. aureus adhesion and infection.