Fever following interventional or surgical pulmonary valve replacement - SIRS like humeral and cellular immune response in the context of treating RVOT dysfunction in congenital heart defects

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Objective: Pulmonary valve implantation (PVI) led to a substantial increase in life expectancy for many patients with congenital heart disease. Transcatheter pulmonary valve implantation (TPVI) has evolved to an alternative when suitable, reached worldwide clinical acceptance and routine procedure status. By the help of TPVI, dysfunction of right ventricular outflow tract (RVOT) could be successfully treated in more than 3000 patients worldwide so far. Currently used pulmonary valve conduits possess xenograft materials (porcine or bovine). Procedure-induced and / or xenograft-tissue-induced febril systemic inflammatory response syndrome (SIRS) could provoke a subsequent dysfunction of the engrafted valve. Hence, in this study, the immune response is analyzed in detail before and after TPVI and surgical pulmonary valve replacement (SPVI).

Methods: At defined time points before and after RVOT intervention (preliminary stent implantation (n=20); TPVI (n=20) or SPVI (n=10)), clinical data and blood samples of adults and infants (median age: 17 years) are compared regarding changes in general-clinical (heart and breathing frequencies, temp.), unspecific para-clinical (CBC, LDC, CrP) and specific para-clinical parameters (interleukin (IL) 12p70/1beta/2/6/8/10, TNFa, PCT, leukocyte analysis by flow cytometry).

Results: After ethics board approval 20 percutaneous and 10 surgical treated patients were included in the study from August 2013 to November 2014. First results of analysis of immune response reveal an increase of IL 6 and IL 8 concentrations within the first 72 h after the procedure (pre-stenting and both types of pulmonary valve implantation). Granulocytes count, especially neutrophils, raises 24 h after percutaneous or surgical valve implantation. Monocyte count does not change after each treatment option. Lymphocytes counts decrease within the first 24 h after both treatment options. This is explained by T cell diminishment.

Conclusion: The analysis of specific and unspecific parameters of humeral and cellular immunity leads to the characterization of the febril immune response after TPVI or SPVI. The number of included patients limits statistical relevancy by now, but trends of specific cellular populations changes are pointed out. Further investigations will be realized to understand this phenomenon in detail and to what extent this is provoked by xenograft material or intervention itself.