THE INFLUENCE OF A SYSTEMIC INFLAMMATORY RESPONSE SYNDROME ON PROGNOSIS AFTER TRANSCATHETER AORTIC VALVE IMPLANTATION

ACC Poster Contributions
Ernest N. Morial Convention Center, Hall F
Sunday, April 03, 2011, 3:30 p.m.-4:45 p.m.

Session Title: Aortic Valve Disease and its Treatment
Abstract Category: 19. Valvular Disease
Session-Poster Board Number: 1049-78

Authors: Jan-Malte Sinning, Viktoria Adenauer, Hannah Steinhäuser, Alexander Ghanem, Christoph Hammerstingl, Georg Nickenig, Nikos Werner, Medizinische Klinik II, Universitätsklinikum Bonn, Bonn, Germany

Background: A systemic inflammatory response syndrome (SIRS) is induced in nearly all patients undergoing open-heart surgery. The aim of this study was to elucidate the influence of a SIRS on prognosis after transcatheter aortic valve implantation (TAVI).

Methods: TAVI was performed with the 18F-CoreValve™ prosthesis via transfemoral access in 92 consecutive patients with a mean age of 80.7±6.3 years (STS mortality score 9.4±7.1%, logistic EuroSCORE 30.0±17.5%). Proinflammatory cytokines [Interleukin-6 (IL-6), Interleukin-8 (IL-8)] and acute-phase proteins [C-reactive protein (CRP), procalcitonin (PCT)] were measured at baseline, 1h, 4h, 24h, 48h, 72h, and 7days after TAVI.

Results: SIRS occurred in 26/92 patients: 15 patients (58%) with SIRS died during follow-up. The incidence of SIRS was related to acute kidney injury (AKI) (63 vs. 21%; P<0.001) and repeated rapid pacing after valve-deployment (50 vs. 25%, P=0.02), but independent from procedure time. During the first 24-48h, patients with AKI showed a significant increase of IL-6 (P=0.01), IL-8 (P=0.02), CRP (P=0.01), and PCT levels (P=0.01). The occurrence of SIRS increased 30-day mortality (HR 6.7, 95% CI: 1.4-33.4, P=0.02) and 1-year mortality (HR 3.3, 95% CI: 1.5-7.3, P=0.002) after TAVI.

Conclusions: Post-procedural occurrence of SIRS is a strong predictor of 30-day and 1-year mortality after TAVI. Our results suggest that SIRS might be result of renal ischemia-reperfusion injury and plays an additional role in the pathogenesis in AKI.