Renal failure: a non-cardiac source of high sensitivity cardiac troponin T

A. Papachristou[1], C. Puelacher[2], N. Glarner[2], I. Strebel[2], J. Steiger[3], M. Diebold[3], G. Lurati Buse[4], D. Bolliger[5], L.A. Steiner[5], L. Gurke[6], T. Wolff[6], E. Mujagic[6], D.M. Gualandro[2], C. Mueller[2], T. Breidthardt[1]

[1]University Hospital Basel, Cradiovascular Research Institute Basel and Department of Cardiology, Division of Internal Medicine, Basel, Switzerland; [2]University Hospital Basel, Cardiovascular Reasearch Institute Basel and Department of Cardiology, Basel, Switzerland; [3]University Hospital Basel, Clinic for Transplantation Immunology and Nephrology, Basel, Switzerland; [4]University Hospital Duesseldorf, Department of Anaesthesiology, Duesseldorf, Germany; [5]University Hospital Basel, Clinic for Anaesthesia, Intermediate Care, Prehospital Emergency Medicine, and Pain Therapy, Basel, Switzerland; [6]University Hospital Basel, Department of Vascular Surgery and Renal Transplantation, Basel, Switzerland

Funding Acknowledgement: Type of funding sources: Public Institution(s). Main funding source(s): Swiss National Science FoundationSwiss Heart Foundation

Background: Circulating high sensitivity cardiac troponin T (hs-cTnT) levels are frequently elevated in patients with end-stage renal disease (ESRD). The underlying pathophysiology is largely unknown. Currently, accumulation of hs-cTnT due to impaired renal clearance, as well as increased production caused by chronic cardiomyocyte injury is being discussed.

Purpose: The aim of this study was to assess the relative contribution of impaired renal clearance as a non-cardiac source of elevated hs-cTnT concentrations, using renal transplantation as an in vivo model of rapidly improved renal function and on a short-term basis rather unchanged cardiac pathology.

Methods: This single-centre study was a secondary analysis within a prospective active surveillance study program for perioperative myocardial infarction/injury (PMI). 42 consecutive high-risk patients undergoing renal transplantation without evidence of PMI were included. Serial creatinine and hs-cTnT (Elecsys, Roche) measurements were performed pre-transplant (baseline) and post-transplant on day (d) 1, between d2 and d5, and between d14 and d180. The effect of time and creatinine on hs-cTnT was estimated with a log-level non-linear mixed-effects model, where time and creatinine were treated as the fixed effects and subject as the random effect. Natural cubic splines were used to account for nonlinearity in the fixed effects.

Results: Baseline median serum creatinine concentration was 616 umol/L [interquartile range (IQR) 477–825], and significantly fell to 425 umol/L (IQR 313–619) on d1, 285 umol/L (IQR 194–509) on day2–5, and 116 umol/L (IQR 100–166) on d14–180 ($p<0.001$, $p<0.001$, and $p=0.043$, respectively; Figure 1A).

Pre-transplant hs-cTnT concentrations were above the 99th percentile (14ng/L) in all patients, median hs-cTnT concentration was 50 ng/L (IQR 35–70). In parallel to the fall in serum creatinine from baseline to d1, hs-cTnT concentrations significantly fell to 28 ng/L (IQR 15–40) on d1 ($p<0.001$), and then remained constant on d2–5 (27 ng/L (IQR 18–35)), and on d14–180 (24 ng/L (IQR 19–28); Figure 1B).

The mixed-effect model showed a significant decrease of hs-cTnT between baseline and d1 ($p<0.001$), whereas no significant change between d1 and d2 ($p=0.82$) occurred (Figure 2).

Conclusion: In contrast to the continuously falling serum creatinine levels, hs-cTnT concentrations reduced by about 50% only within the first 24 hours with a functional graft and then remained elevated above the 99th percentile. This suggests, that ESRD is a non-cardiac source of elevated circulating hs-cTnT concentrations, which contributes about 50%, while the other 50% seem related to chronic cardiomyocyte injury. Further studies assessing the long-term effect of renal transplantation on hs-cTnT levels and cardiac function are needed.

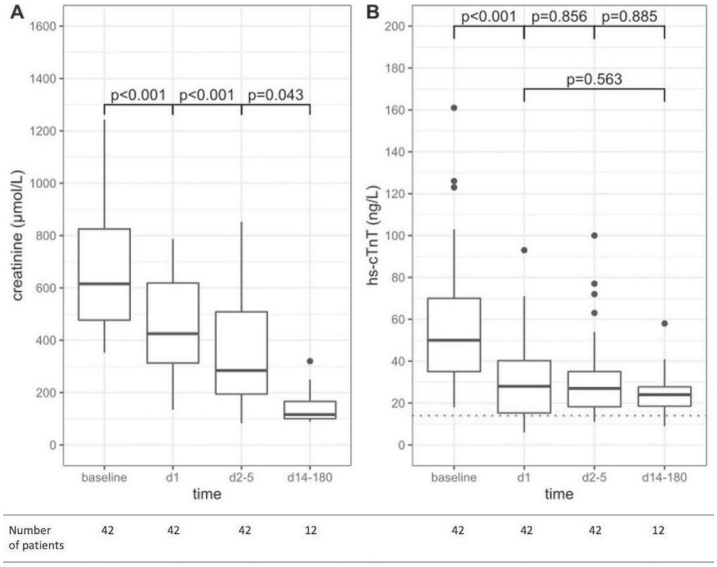

Figure 1: Serum creatinine **(A)** and hs-cTnT **(B)** concentrations pre-and post-transplantation

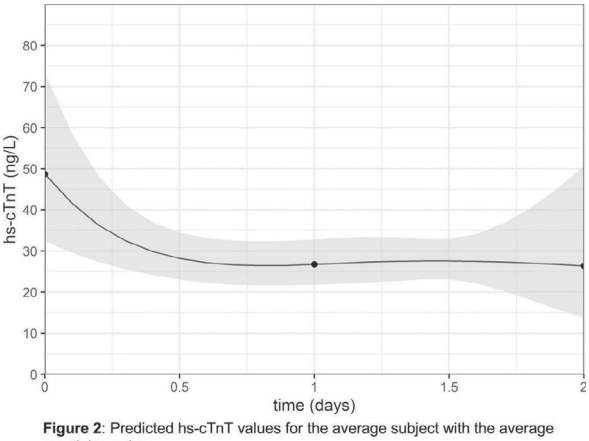

Figure 2: Predicted hs-cTnT values for the average subject with the average creatinine value.

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