Hemodynamic status during targeted temperature management and association with cerebral and cardiovascular death after out-of-hospital cardiac arrest.

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Background: After resuscitation from out-of-hospital cardiac arrest (OHCA), patients suffer from the post-cardiac arrest syndrome (PCAS) characterized by brain injury, a sepsis-like inflammatory response and hemodynamic instability with impaired vasoregulation.

Purpose: This study investigates hemodynamic status and inflammation in relation to cause of death as a post hoc analysis of a large trial cohort of PCAS patients.

Methods: Single-center substudy of 152 patients included in the prospective, randomized Targeted Temperature Management-trial. Hemodynamic evaluation was performed by serial measurements by pulmonary artery catheter. The inflammatory response was assessed by Interleukin-6 (IL-6) 48 hours after admission.

Measurements and Main Results: Patients were stratified according to survival status after 180 days and deceased patients were further stratified according to cause of death: Survivors (n=101 (66%)), cerebral death (n=39 (26%)), cardiovascular death (n=12 (8%)). The hemodynamic profile (mean arterial pressure, heart rate, stroke volume, cardiac index, central venous saturation and arterial lactate) of survivors and cerebral deaths was similar during TTM and rewarming (figure). Patients dying from a cardiovascular cause had a significantly different hemodynamic profile than the other groups (figure). In univariate logistic regression, cardiovascular death was associated with lower mean MAP (odds ratio: 0.89 (0.79-0.99), p=0.03 per mmHg increase), lower mean central venous saturation (odds ratio: 0.89 (0.82-0.98), p=0.01 per % increase), lower stroke volume (odds ratio: 0.96 (0.92-0.99), p=0.02 per ml increase), higher mean heart rate (odds ratio: 1.06 (1.02-1.09), p=0.004 per beat increase) and higher mean lactate (odds ratio: 1.82 (1.26-2.62), p=0.001 per mmol/L increase). No hemodynamic parameters were significantly associated with cerebral death. IL-6 was significantly higher in the cardiovascular death group (Survivors, median (inter-quartile range): 104 (63-202) pg/mL, cerebral death: 149 (89-265) pg/mL, cardiovascular death: 1232 (132-1765) pg/mL, p<0.0001), however not different between survivors and cerebral deaths (p=0.11).

Conclusions: Patients dying from cardiovascular failure after OHCA have elevated IL-6 and severely affected hemodynamic status during TTM. Patients dying of anoxic brain injury have relatively little inflammation and hemodynamic instability, suggesting that severe anoxic brain injury can be an isolated phenomenon without the other aspects of the PCAS. Future studies of OHCA could individualize treatment depending on which part of the PCAS that is pronounced.
Hemodynamic status during Targeted Temperature Management (0-28 hours) and the following 20 hours stratified according to cause of death. Difference between groups during all time points is signified by: $p_{\text{group}}$. Statistically significant difference at single time points is signified by: *.