Activation of the kynurenine pathway predicts mortality and neurological outcome in cardiac arrest patients: A validation study

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Activation of the kynurenine pathway (KP) has been shown to predict outcome in cardiac arrest (CA) patients. We validated these findings in a Swiss cohort.; We measured admission tryptophan and kynurenine levels in 270 consecutive CA patients (38 in-hospital CA) and investigated associations with in-hospital mortality and neurological outcome at hospital discharge.; 120 of 270 (44%) patients died in the hospital. Compared to survivors, non-survivors showed higher median initial kynurenine levels (5.28 μmol/l [IQR 2.91 to 7.40] vs 3.58 μmol/l [IQR 2.47 to 5.46]; $p < 0.001$) and a higher median kynurenine/tryptophan ratio (0.10 μmol/l [IQR 0.07 to 0.17] vs 0.07 μmol/l [IQR 0.05 to 0.1]; $p < 0.001$). In a model adjusted for age, gender and comorbidities, kynurenine (OR 1.16, 95% CI 1.05 to 1.27; $p = 0.001$) and kynurenine/tryptophan ratio (OR 1.19, 95% CI 1.08 to 1.31; $p = 0.003$) were significantly associated with mortality. Results were similar for neurological outcome.; Our findings validate a previous study and show associations of the activation of the KP with unfavorable outcomes after CA. Future studies should evaluate whether therapeutic modulation of the KP may impact clinical outcomes after CA.

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