

ACUTE KIDNEY INJURY PREDICTS NEUROLOGICAL OUTCOME & MORTATLITY AFTER CARDIAC ARREST

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INTRODUCTION

Acute kidney injury (AKI) has been associated with inferior short- and long-term mortality after cardiac arrest. Although AKI is accepted as a consequence of severe illness in critically ill patients, an abundance of epidemiological data demonstrates that AKI itself contributes to this elevated mortality rate and acts as an independent risk factor. However, the impact of AKI on neurological recovery and short- and long-term outcomes remains scarcely described. Therefore we evaluated the incidence of AKI in cardiac arrest patients and characterized their neurological outcome.

PATIENTS AND METHODS

In our single center study, we consecutively enrolled 515 out- and in-hospital cardiac arrest (OHCA/IHCA) patients from 2006 to 2013, who were admitted to our intensive care unit for at least 72 hours, and treated with therapeutic temperature management. Patients were grouped with respect to the presence of AKI defined by the KDIGO guidelines 2012. We assessed patient characteristics, comorbidities, cardiac arrest details, and therapeutic interventions for all patients and analyzed neurological recovery, long-term neurological outcomes, and mortality. Neurological recovery was classified according to the widely-used Pittsburgh Cerebral Performance Category (CPC).

RESULTS

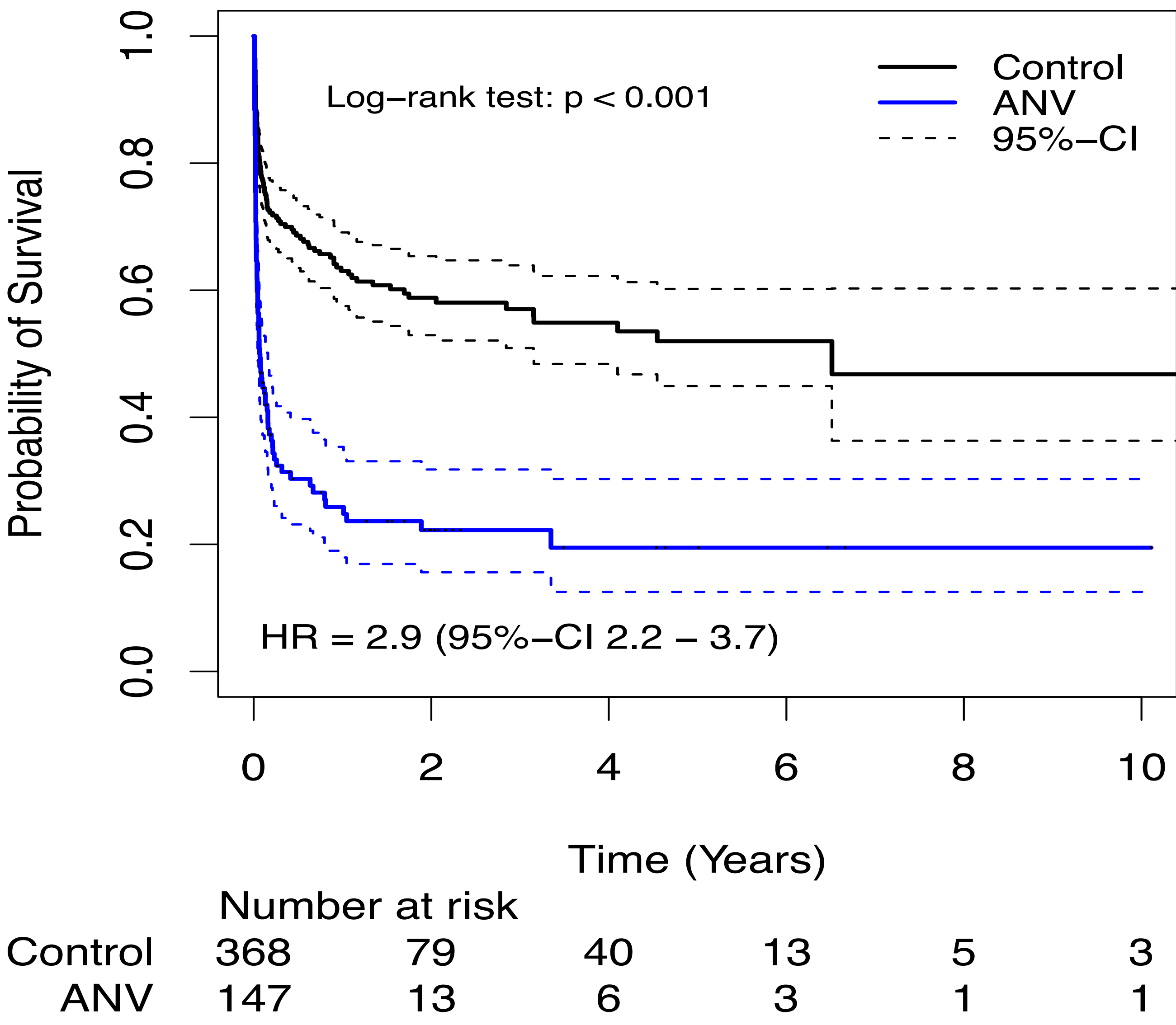


Figure 1: Kaplan-Meier plot of patient probability of survival after cardiac arrest with (ANV) or without AKI (=Control)

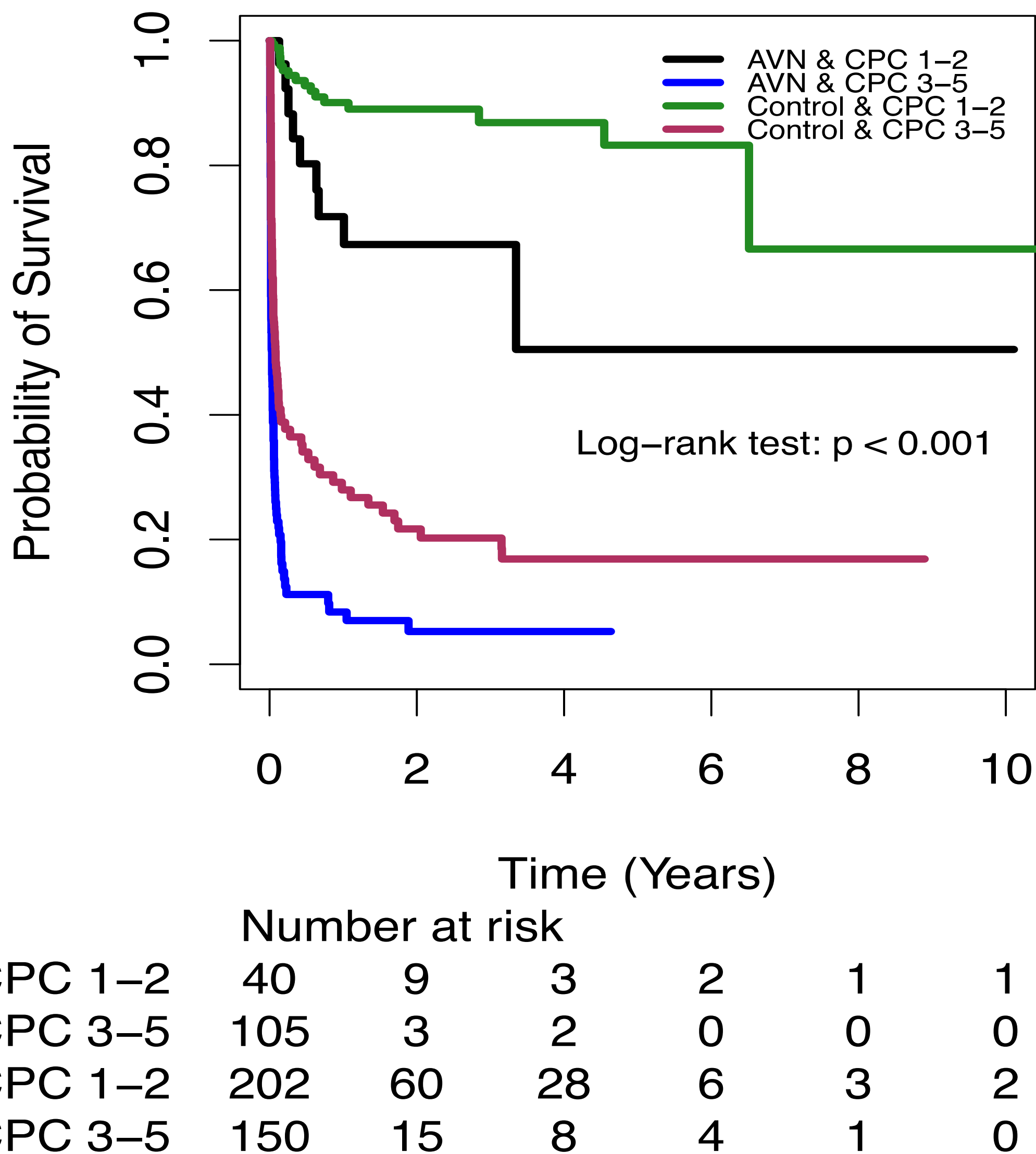


Figure 2: Kaplan-Meier plot of patient probability of survival after cardiac arrest with or without AKI (ANV) in relation to good neurological outcome (CPC 1-2) vs. poor neurological outcome (CPC 3-5)

147 of 515 patients (28.5%) developed AKI during the intensive care unit period. Patients who developed AKI showed inferior neurological outcomes with a median CPC score of 5 compared to patients without AKI with a median CPC score of 2 after cardiac arrest (Figure 1 and 2). Median survival for patients with AKI after cardiac arrest was 1 month compared to 78 months for patients without AKI (Figure 1+2). Independent risk factors for the development of AKI after cardiac arrest were age (p=0.002), time to return of spontaneous rhythm (p=0.006), and the initial APACHE-II-Score (p<0.001) (Figure 3). Age, female sex, time to ROSC and AKI were predictive factors of mortality after cardiac arrest (p<0.05) (Table 1).

Variables	HR	Lower HR CI	Upper HR CI	p-value
AKI KDIGO	2,365	1,739	3,218	<0.001
Age (years)	1,018	1,007	1,03	0,001
Time to ROSC (min)	1,018	1,006	1,029	0,002
Gender female	1,48	1,071	2,046	0,017
NSE at + 72 hours (µ/L)	1,002	1,001	1,002	<0.001
First Rhythm Asystole	2,876	2,083	3,971	<0.001
First Rhythm PEA	1,659	1,066	2,583	0,025

Table 1: Cox regression analysis for survival and AKI after cardiac arrest

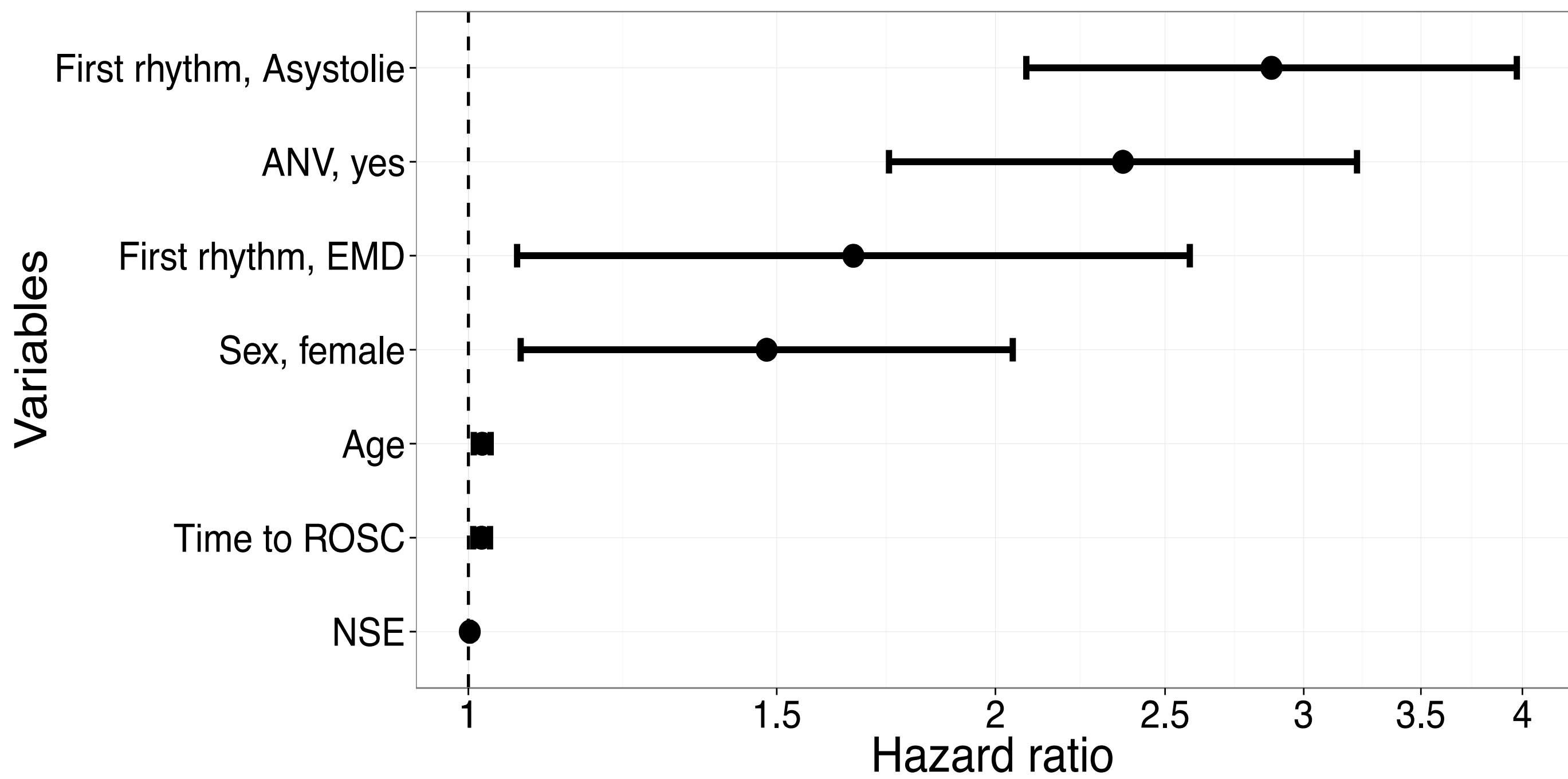


Figure 3: Hazard Ratio for AKI concerning first rhythm, AKI (acute kidney injury), Gender, Age, Time to ROSC (=return of spontaneous circulation), NSE (=neuron specific enolase).

CONCLUSIONS

Our data suggest that AKI is an independent risk factor for poor neurological outcome after cardiac arrest. In addition, AKI predisposes to a higher mortality among those who survived cardiac arrest. Here, AKI may reflect both, more severe chronic comorbid conditions and the extend of the cardiac arrest. This supports the importance of closely monitoring, diagnosing and treating patients for AKI and considering the medical decisions taking and implications on further medical assistance beyond discharge.

