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ORIGINAL SCIENTIFIC PAPER
Acute Coronary Syndromes

Long-term outcomes after acute kidney injury in myocardial infarction complicated by cardiogenic shock: a retrospective, observational study

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Aims

The recent DanGer shock trial found reduced mortality, but increased risk of acute kidney injury (AKI) in patients treated with a microaxial flow pump after an acute myocardial infarct with cardiogenic shock. AKI has previously been associated with increased short-term mortality, whereas data on long-term outcomes are sparse. We aimed to describe the frequency of AKI and associated risk factors as well as long-term mortality and morbidity.

Methods and results

A retrospective observational study comprising patients admitted with acute myocardial infarction cardiogenic shock in Denmark between 2010 and 2017 with data on kidney function from the RETROSHOCK cohort. National health registry data enabled 10-year follow-up to assess mortality and morbidity. Kaplan-Meier estimates and competing risks regression were used to evaluate the association of AKI with the incidence of short- and long-term mortality, chronic kidney disease (CKD) and dialysis. Among 1473 patients, 44% developed AKI, 25% required renal replacement therapy (RRT). AKI development was associated with increasing age, diabetes, low ejection fraction and high lactate levels on admission ($P < 0.05$). Thirty-days mortality as well as mortality at 1–, 5–, and 10-years follow-up was significantly increased in patients with AKI; at 10 years follow-up mortality was increased by more than 30% ($P < 0.001$). The 10-year cumulative incidence of both CKD and dialysis, accounting for the competing risk of death, was significantly higher in patients treated with RRT during admission ($P < 0.001$).

Conclusion

AKI was associated with increased short- and long-term mortality and morbidity, including CKD and dialysis, but not new cardiovascular events.

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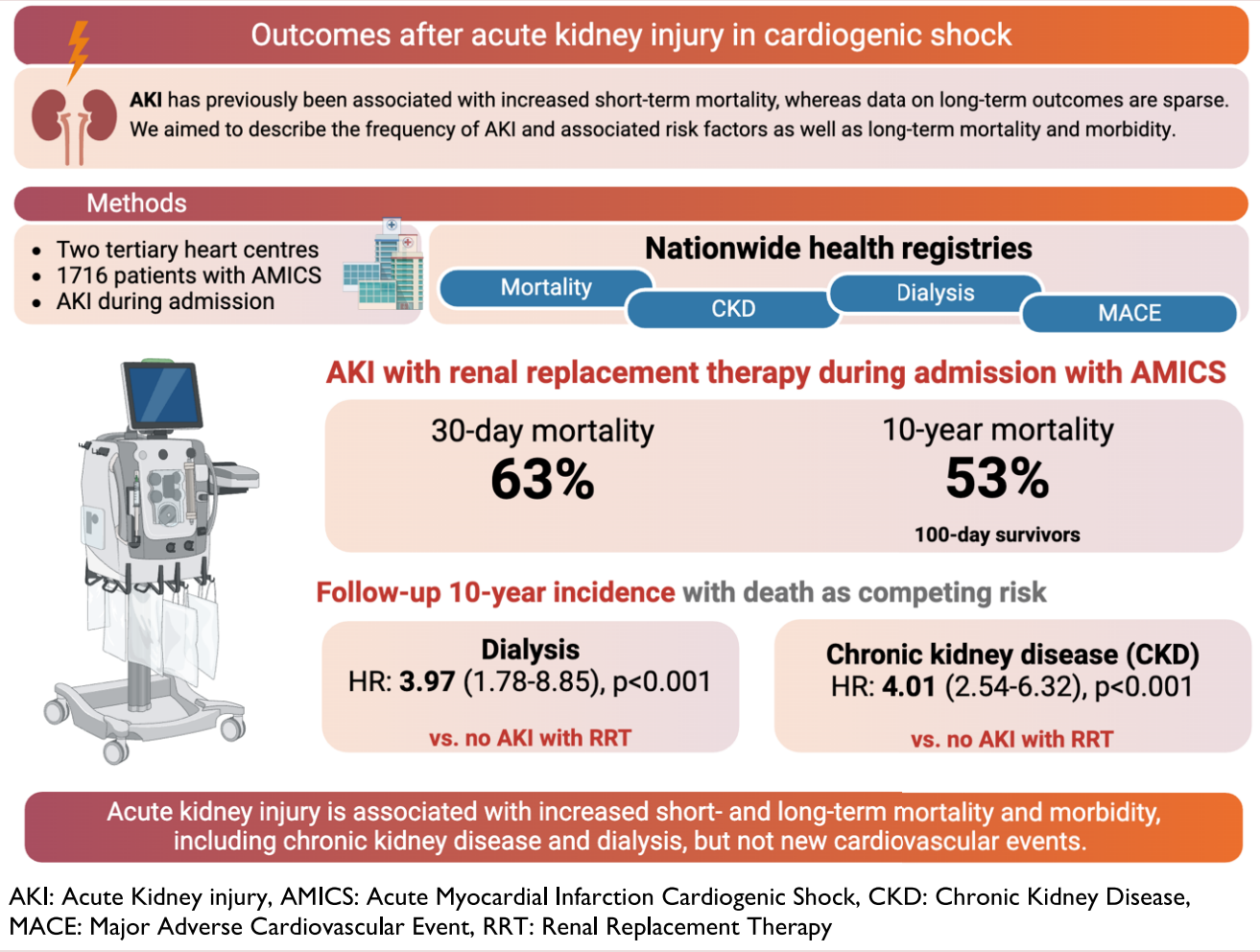
Graphical Abstract



AKI: Acute Kidney injury, AMICS: Acute Myocardial Infarction Cardiogenic Shock, CKD: Chronic Kidney Disease, MACE: Major Adverse Cardiovascular Event, RRT: Renal Replacement Therapy

Keywords Acute myocardial infarction • Cardiogenic shock • Acute kidney injury • Intensive care • Renal replacement therapy • Chronic kidney disease

Introduction

Cardiogenic shock caused by acute myocardial infarction (AMI-CS) is a condition with an abrupt decline in cardiac output leading to organ hypoperfusion, often involving the need for intensive care therapy and a high mortality risk.[1–6] The kidneys are especially sensitive to hypoperfusion, resulting in a high incidence of acute kidney injury (AKI) and need for renal replacement therapy (RRT) in this population.[2,7] Development of AKI has multifactorial causes, but has consistently been associated with increased short- and long-term mortality, especially in more severe stages of kidney dysfunction.[1,2,8,9] Recently the DanGer Shock trial reported improved survival with the use of a micro-axial flow pump (mAFP) compared with standard care in AMI-CS patients.[10] AKI was an important risk factor in DanGer, but a survival benefit was seen despite a significantly increased occurrence of AKI in the mAFP-group, and at 6-months follow-up all survivors were free of dialysis.[7,10] Whether the transient AKI exposes patients to long-term risk of CKD and increased mortality has mainly been based on registry data,[2,8] where AKI not requiring RRT will serve as a control together with patients without AKI, which may obscure the true impact of AKI. By using data from the clinical registry RETROSHOCK, we intended to describe the frequency of AKI in a well-defined Danish AMI-CS population. In addition, we wanted to describe short- and long-term outcomes after AKI and to evaluate long-term morbidity in terms of chronic kidney disease (CKD), need for dialysis and new cardiovascular events.

Methods

Ethical considerations

The study was conducted in accordance with applicable legislation and guidelines, with approval from the relevant institutional review board. Due to the retrospective nature of the study, patient consent was waived.

Table 1 Baseline characteristics

n	No AKI 821	AKI without RRT 276	AKI with RRT 376	*P*-value
Age, years	65 (12)	68 (11)	66 (11)	**<0.001**
Male gender	634 (78%)	201 (73%)	290 (77%)	0.26
BMI, kg/m^2	26.1 (4.1)	26.7 (4.8)	26.9 (4.5)	**0.02**
Medical history				
Hypertension	384 (49%)	149 (57%)	192 (53%)	**0.05**
Myocardial infarction	103 (13%)	56 (21%)	54 (15%)	**0.006**
Diabetes	109 (14%)	63 (24%)	96 (27%)	**<0.001**
Symptoms at presentation				
Dyspnea	156 (19%)	83 (30%)	119 (32%)	**<0.001**
OHCA	443 (54%)	109 (40%)	131 (35%)	**<0.001**
Status at arrival				
LV Ejection fraction, %	31 (12)	27 (12)	25 (13)	**<0.001**
Heart rate, min^{-1}	82 (24)	93 (33)	90 (25)	**<0.001**
Systolic BP, mmHg	84 (15)	84 (17)	82 (15)	0.11
Arterial lactate, mmol/L	5.8 (4.3)	6.8 (4.5)	7.9 (5.2)	**<0.001**
Revascularization				
PCI	726 (96%)	225 (95%)	303 (90%)	**<0.001**
Emergency CABG	24 (3%)	11 (5%)	35 (10%)	**<0.001**
Coronary culprit lesion				
Left Main	61 (8%)	23 (10%)	71 (21%)	
LAD	355 (47%)	117 (49%)	134 (40%)	
LCx	123 (16%)	28 (12%)	37 (11%)	
RCA	215 (29%)	69 (29%)	95 (28%)	

Data are presented as number and percentage (%). Statistically significant values are marked in bold numbers.
AKI, acute kidney injury; BMI, body mass index; BP, blood pressure; CABG, coronary artery bypass surgery; LCX, left coronary artery; LAD, left anterior descending artery; OHCA, out of hospital cardiac arrest; PCI, percutaneous coronary intervention; RCA, right coronary artery.

This is a retrospective observational study using data from the RETROSHOCK cohort, which comprises 1716 patients admitted to two Danish tertiary cardiac centres, covering more than 50% of the Danish population. Patients were admitted with AMI-CS during the period of 1 January 2010 to 31 December 2017.[4]

AMI-CS patients were identified from the Danish National Patient Registry as previously described in detail.[4] Patients who had received chronic haemodialysis prior to admission were excluded from analyses.

In patients identified as potential AMI-CS patients a full review of the electronic medical record was performed to identify patients with CS that fulfilled all of the following criteria: (A) age ≥18 years; (B) persistent hypotension with systolic blood pressure ≤90 mmHg for >30 min or use of vasoactive drugs; (C) signs of impaired organ perfusion (at least one of the following: altered mental status excluding medically-induced sedation; cold/clammy skin; oliguria; arterial lactate ≥2.5 mmol/L); and (D) documented reduction in left and/or right ventricular function in the absence of hypovolemia, sepsis, anaphylaxis, pulmonary embolism or primary valve dysfunction. The diagnosis of AMI was made at the discretion of the treating physician based on the current definition of myocardial infarction.

Individual electronic hospital medical records of patients fulfilling the AMI-CS definition were reviewed for patient demographics, medical history, hemodynamic and clinical data. Clinical data included interventions performed, location of culprit lesion, use of mechanical circulatory assist device and type, use of mechanical ventilation and use of vasoactive drugs. Emergency medical service patient charts were also evaluated for prehospital management.

Primary outcomes were AKI, in-hospital mortality, long-term kidney disease, major adverse cardiovascular event (MACE) associated with CKD and long-term mortality.

Signs of AKI were evaluated through chart review and assessment of laboratory results. Patients were classified during chart review in three groups based on RIFLE-criteria to align with previous cardiogenic shock studies, using increase in *P*-creatinine and use of RRT.[11] Systematic information on urine output was not obtained. Based on these criteria patients were classified into three groups: No AKI, AKI without RRT, AKI with RRT.

Data on all-cause mortality and long-term kidney outcomes were obtained from Danish nationwide registries. All Danish citizens are registered in the Civil Registration Registry with a unique personal number that allows identification across several national registries and contain data on survival status.[12] Data on long-term kidney outcomes were collected from the Danish National Patient Registry, which holds information on all hospital in- and outpatient contacts since 1977 coded with one primary diagnosis according to the International Classification of Diseases 10th edition (ICD-10) at discharge and if relevant one or more secondary diagnoses.[13] Collection of long-term kidney outcomes was arbitrarily initiated 100 days after admission for AMI-CS to exclude transient RRT during admission for AMI-CS. Contacts associated with dialysis, CKD, stroke, AMI, percutaneous intervention, coronary artery bypass grafting, aortic stenosis, implantation of a cardioverter defibrillator or pacemaker were counted for up to 10 years (referred to herein as MACE, Supplementary material online, *Table S1*). Mortality was assessed at 30 days (all ICU patients), 1, 5, and 10 years (100-day survivors to exclude patients who died in relation to

Table 2 In-hospital management and duration

n	No AKI 821	AKI without RRT 276	AKI with RRT 376	*P*-value
Mechanical ventilation	662 (81%)	238 (87%)	354 (95%)	**<0.001**
Days on ventilator	2 (1–5)	3 (1–5)	5 (2–11)	**<0.001**
Vasoactive medication				
Norepinephrine	560 (70%)	233 (85%)	360 (96%)	**<0.001**
Epinephrine	124 (16%)	87 (32%)	208 (56%)	**<0.001**
Dopamine	109 (14%)	63 (24%)	96 (27%)	**<0.001**
Dobutamine	27 (3%)	18 (7%)	36 (10%)	**<0.001**
Milrinone	144 (18%)	84 (31%)	185 (51%)	**<0.001**
Levosimendan	36 (5%)	43 (16%)	96 (26%)	**<0.001**
Mechanical circulatory support				
IABP	89 (11%)	26 (10%)	62 (17%)	**0.007**
Impella	39 (5%)	36 (13%)	123 (33%)	**<0.001**
VA-ECMO	12 (2%)	6 (2%)	37 (10%)	**<0.001**
Outcome				
In-hospital mortality	251 (31)	167 (62)	241 (66)	**<0.001**
ICU days (all)	3 (1–6)	3 (1–6)	6 (2–13)	**<0.001**
ICU days (hospital survivors)	3 (1–5)	5 (3–9)	13 (7–23)	**<0.001**

Statistically significant values are highlighted i bold numbers. IABP, intra-aortic balloon pump; ICU, intensive care unit; VA-ECMO, veno-arterial extracorporeal membrane oxygenation.

index admission). Follow-up was available until 1 January 2024, whereafter any remaining patients were censored.

Statistical analyses

Data are presented as mean (±SD) for continuous data, and nominal data are given as number (frequency in percentage). Comparison between groups was either performed as the Pearson χ^2 test or one-way ANOVA, depending on variable types. Factors associated with AKI were assessed with logistic regression analysis based on data available after initial treatment in the catheterisation laboratory.

Differences in mortality were assessed using the log-rank test between each AKI classification group. Cox regression models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for mortality, both unadjusted and adjusted for factors known to be associated with mortality. Cumulative incidence functions were used to evaluate the risk of CKD, dialysis, or MACE over a 10-year follow-up period. Gray's test was used to compare cumulative incidence functions across AKI classification groups, accounting for death as a competing risk. Subdistribution hazard ratios and their 95% CIs were estimated using Fine and Gray regression to quantify the association of AKI with the risk of CKD, dialysis, and MACE, while adjusting for competing risks. A *P*-value <0.05 was considered statistically significant.

Results

Among 1716 patients in the RETROSHOCK cohort, 186 patients died before admission to the ICU, and data on AKI were not available in 57 patients leaving 1473 patients for the analysis. There were 652 patients (44%) developing AKI, and in 376 patients (25.5%), RRT was established.

Risk factors for AKI in AMI-CS patients included increasing age, diabetes, elevated lactate level and lower LVEF upon arrival. In contrast, out-of-hospital cardiac arrest (OHCA) was associated with a lower risk of developing AKI (see Supplementary material online, *Table S2*).

Patient characteristics according to AKI classification are listed in *Table 1*.

Of note, diabetes was almost twice as common in AKI patients, but systolic blood pressures upon admission were comparable and independent of AKI development.

During ICU admission, the need for vasoactive drugs and mechanical circulatory support was higher in patients developing AKI and with increasing severity of AKI.

AKI was associated with significantly higher in-hospital mortality and, among survivors, a significantly longer length of stay in the ICU (*Table 2*).

Kaplan-Meier analysis (*Figure 1*) for all-cause mortality split according to AKI status demonstrated a lower 30-day mortality in AMI-CS patients without AKI (37%), whereas mortality was 66 and 63% for AMI-CS patients with AKI ± RRT, respectively ($P < 0.001$).

In unadjusted Cox regression analysis, AKI was associated with a significantly higher risk of 30-day mortality compared with no AKI. The HR was 2.61 (95% CI 2.16–3.16, $P < 0.001$) in patients with AKI without RRT and 2.36 (95% CI 1.98–2.82, $P < 0.001$) in patients requiring RRT.

After adjusting for factors associated with mortality, including age, gender, diabetes, body mass index (BMI), history of hypertension, prior ischaemic heart disease, prior stroke, LVEF and lactate levels, the association between AKI and mortality remained significant. The adjusted HR was 2.22 (95% CI 1.67–2.96, $P < 0.001$) for AKI without RRT and 2.23 (95% CI 1.73–2.89, $P < 0.001$) for AKI with RRT.

In a model incorporating an interaction term between AKI classification and OHCA status, AKI remained significantly associated with mortality in patients without OHCA (HR 2.60, 95% CI 1.76–3.83, $P < 0.001$ for AKI without RRT and HR 2.49, 95% CI 1.75–3.55, $P < 0.001$ for AKI with RRT). The interaction term was not statistically significant, indicating that the association between AKI and mortality did not differ significantly between patients with and without OHCA.

Long-term outcomes in relation to AKI during admission and being alive at 100 days, demonstrated that AKI was associated with significantly increased mortality up to 10 years. After ten years differences in mortality were even more pronounced, with a relative increase of more than 30% in AKI patients (*Table 3*; $P = 0.005$).

Ten years after hospital discharge, the cumulative incidence of CKD or the need for chronic dialysis, with death as a competing risk, was more common in patients with AKI (*Table 4*), but only significantly in AKI patients requiring RRT during index admission [HR_{CKD}: 4.01 (2.54–6.32); $P < 0.001$] and [$HR_{dialysis}$ 3.97 (1.78–8.85); $P < 0.001$].

In adjusted models accounting for age, gender, diabetes, BMI, history of hypertension, prior ischaemic heart disease and stroke, the association

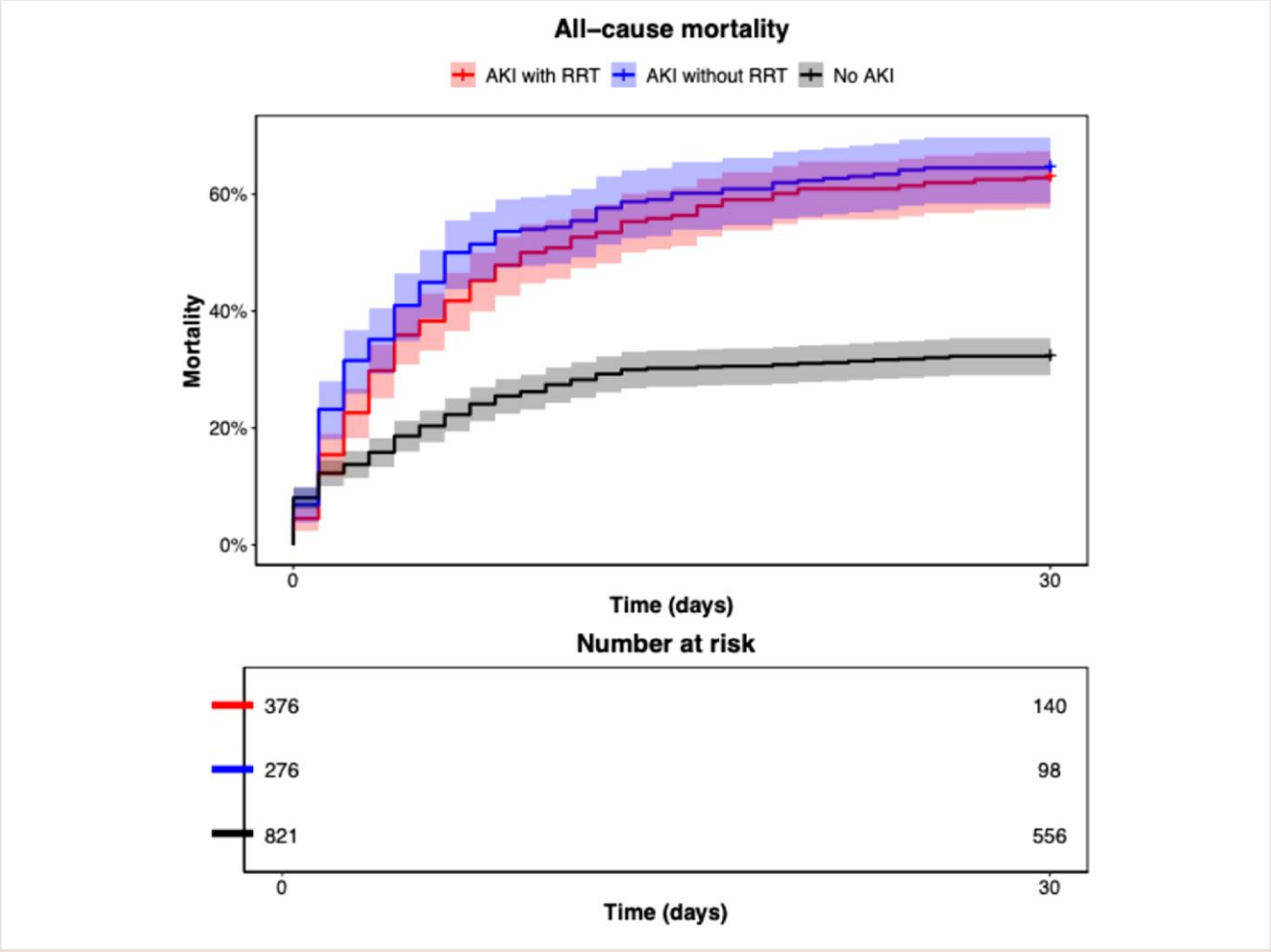


Figure 1 All-cause 30-day mortality from time of admission stratified by AKI status in patients with AMI complicated by cardiogenic shock.

Table 3 Long-term mortality

	100-day survivors ($n = 741$)			
	1 year	5 years	10 years	*P*-value
No AKI	5.4% (3.5–7.3)	20% (16–23)	39% (34–43)	0.005
AKI without RRT	8.0% (2.1–14)	23% (14–31)	50% (36–61)	
AKI with RRT	9.2% (3.9–14)	26% (18–33)	53% (41–62)	

a: Log-rank test at end of follow-up.

remained significant for AKI with RRT [HR_{CKD}: 3.97 (2.97–6.74), $P < 0.001$; $HR_{dialysis}$: 2.97 (1.4–7.72), $P < 0.001$], while AKI without RRT was not significantly associated with increased risk of CKD or dialysis (*Table 4*). Adding arterial lactate and left ventricular ejection fraction to the adjusted analyses did not alter the conclusions.

In a sensitivity analysis of non-OHCA patients, AKI requiring RRT remained strongly associated with an increased risk of CKD and dialysis, both in unadjusted and adjusted models (see Supplementary material online, *Table S3*).

There were no significant differences in the occurrence of new cardiovascular events between patients with and without AKI (see Supplementary material online, *Figure S1*).

Discussion

In this population of AMI-CS patients, AKI was seen in 44%. AKI occurrence was related to age, diabetes and severity of haemodynamic instability, including need for vasoactive and mechanical support. Development of AKI was associated with increased short- and long-term mortality for up to 10 years. Among AMI-CS survivors, development of CKD and need for chronic dialysis occurred more frequently in patients needing RRT during hospitalisation. The occurrence of new cardiovascular events during 10-year follow-up was high but independent of AKI development.

AKI in AMI-CS partly reflects the cardiorenal syndrome. The kidneys and the heart are co-dependent, in terms of heart failure causing kidney injury and vice versa.[1] The underlying mechanisms in AMI-CS are hypoperfusion, ischemia-reperfusion injury and inflammation causing tubular necrosis. Fluid-retention and fluid administration may lead to venous congestion that may further augment kidney injury, as it has previously been established that positive fluid balance is associated with AKI and increases mortality.[1,7,8,14,15]

Table 4 Cumulative 10-year incidence and subdistribution hazard of CKD, dialysis or a cardiovascular event with death as competing risk, among patients alive at 100 days post discharge ($N = 741$).

	Cumulative incidence % (95% CI)		
	Chronic kidney disease	**Dialysis**	**Cardiovascular events**
No AKI ($n = 534$)	7.9% (5.5–10.1), $n = 40$	2.3 (1.0–3.6), $n = 12$	48.8 (44.3–53.3), $n = 249$
AKI without RRT ($n = 87$)	11.7% (4.8–18.6), $n = 11$	2.3 (0.0–5.5), $n = 3$	56.0 (44.7–67.4), $n = 46$
AKI with RRT ($n = 120$)	27.9% (19.4–36.4), $n = 33$	8.4 (3.4–13.4), $n = 11$	50.3 (40.8–59.9), $n = 58$
P-value (Gray's Test)	*<0.001*	*0.003*	Ns

	Hazard ratio (95% CI) Unadjusted		
No AKI ($n = 534$)	**Chronic kidney disease Reference**	**Dialysis Reference**	**Cardiovascular events Reference**
AKI without RRT ($n = 87$)	1.66 (0.86–3.20), $P = ns$	1.42 (0.41–4.97), $P = ns$	1.11 (0.82–1.49), $P = ns$
AKI with RRT ($n = 120$)	4.01 (2.54–6.32), $P < 0.001$	3.97 (1.78–8.85), $P < 0.001$	1.04 (0.79–1.38), $P = ns$

	Hazard ratio (95% CI) Adjusted for sex, age, BMI, history of diabetes, hypertension and AMI.		
No AKI ($n = 417$)	**Chronic kidney disease Reference**	**Dialysis Reference**	**Cardiovascular events Reference**
AKI without RRT ($n = 70$)	1.50 (0.71–3.19), $P = ns$	0.85 (0.20–3.60), $P = ns$	1.22 (0.88–1.71), $P = ns$
AKI with RRT ($n = 94$)	3.97 (3.97–6.74), $P < 0.001$	2.97 (1.14–7.72), $P < 0.001$	1.05 (0.75–1.46), $P = ns$

Thus, the development of AKI has multifactorial aetiologies relating to both the duration and severity of compromised hemodynamics, but also depends on the type of management AMI-CS patients require. Most AMI-CS patients undergo emergency coronary angiography, which can potentially trigger AKI. The use of vasoactive therapy is also associated with AKI development. Probably not due to the drugs on their own, but merely reflecting the severity of haemodynamic instability, which is also reflected in the SCAI distribution. We have previously reported SCAI-classes from the RETROSHOCK cohort, where RRT frequency increased from 19% in SCAI class C to 55% in SCAI class E.[16] Similar observations were done in the DanGer Shock Trial, where >50% of patients were SCAI-Class D or E when developing AKI.[7]

The vascular effects of diabetes and increasing age with reduced functional reserve seem to make the kidneys more vulnerable to these influences. Interestingly, a lower risk of AKI was seen in OHCA-patients (35%), which is coherent with the frequency of AKI in other observational OHCA studies.[17–19] This may partly relate to a faster haemodynamic recovery with improved cardiac output and kidney perfusion in these patients.[20]

When trying to reconcile observations from the RETROSHOCK cohort into the existing literature, there is substantial evidence on the impact of AKI on outcome. Quite unanimously the occurrence of AKI has been associated with increased mortality,[2,7,14,21,22] and mortality is more than twice as high in patients requiring RRT—an observation confirmed in the present study. Randomized trials can provide information on the effect of certain interventions or management strategies. Despite the significantly improved outcome in terms of survival with a mAFP in the DanGer Shock Trial, it came at a cost, namely a markedly increased risk of AKI, including need for RRT.[10] In contrast, in the ECLS trial, there was no impact of VA-ECMO on the occurrence of AKI nor survival.[5] A subgroup analysis from the DanGer shock trial found suction events and pump speed with signs of haemolysis as well as duration of support to be associated with AKI,[7] and generally mAFPs seem to increase inflammation more than ECMO,[23] indicating that other factors will harm the kidneys despite increased perfusion. This may explain the high need for RRT in the Impella-treated patients in RETROSHOCK, but it may also merely reflect more severely compromised hemodynamics since the causal relationship cannot be deduced due to the observational study design. The RETROSHOCK database represents all comers with AMI-CS, also including patients non-eligible for RCTs, and thereby gives a better reflection of everyday outcomes and consequences of developing for instance AKI.

Long-term outcomes above 1 year have only been sparsely studied.[2,9,24] Lauridsen et al.[2] found that 5-year mortality remained significantly higher with a relative mortality risk of 1.7 in RRT-treated patients,[2] similar to the present study (1.5). The impact of AKI *per se* in this study was seen in the group of patients with AKI without RRT, with increased mortality at 1-, 5-, and 10-year follow-up.

AKI was previously considered a self-limiting condition, as creatinine often normalize before discharge. However, recent studies suggest that even minor episodes of AKI are associated with progression to CKD.[25,26] In the meta-analysis by Abdala et al.,[26] seven studies described the AKI to CKD transition in various critical medical conditions. AKI had a hazard ratio above three for CKD development.[26] We observed the highest occurrence of CKD of 28% in patients treated with RRT, which was four times as frequent than in patients without AKI. Development of CKD is associated with increased risk of cardiovascular events and mortality,[27,28] and may eventually progress to end-stage requiring dialysis or transplantation.[29] In the present study,

chronic dialysis at 10-years follow-up was almost four times as common in patients needing RRT during index admission. We found no difference in the occurrence of cardiovascular events after 10 years. Nevertheless, in the registry study by Lauridsen et al.[2] cause of death in patients requiring RRT during admission for cardiogenic shock was in 61% of cases related to cardiovascular disease as compared to 51% in patients not requiring RRT.[2] Unfortunately, we do not have access to the cause of death after hospital discharge and can therefore not confirm nor defer this observation.

Limitations of the present study relate to the retrospective observational design. We can merely demonstrate associations, not whether there is a cause-effect relationship. Due to the high mortality in cardiogenic shock, the number of survivors is relatively limited, and MACE at follow-up was based on ICD-10 with its inherent limitations. Therefore, the variation in observed outcomes may be biased. However, the development of AKI was consistently associated with an increased mortality rate both short- and long-term.

Data on urinary output was not available, which might have affected the occurrence of AKI in the present study. However, in cardiogenic shock patients, serum creatinine has been established as the strongest predictor for both RRT and mortality.[1]

There is currently no preventive intervention to mitigate AKI. However, the time period from AKI to CKD transition leaves the possibility to prevent progression to CKD.

Future studies should focus on measures in preventing AKI, attenuating the severity of AKI and avoiding the progression to CKD to further improve prognosis in AMI-CS.

Supplementary material

Supplementary material is available at *European Heart Journal: Acute Cardiovascular Care* online.

Author contribution

M.B., J.B.K., K.K.J., O.H., C.H., J.J., L.O.J., L.H., H.S., E.F., J.E.M., and H.B.R.

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Data availability

Due to General Data Protection Regulation restrictions on personal data, the data supporting this article cannot be publicly shared. It may be accessed upon reasonable request to the corresponding author.

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