Predictors and Prognostic Impact of Early Acute Kidney Injury in Cardiogenic Shock: Results from a Monocentric, Prospective Registry

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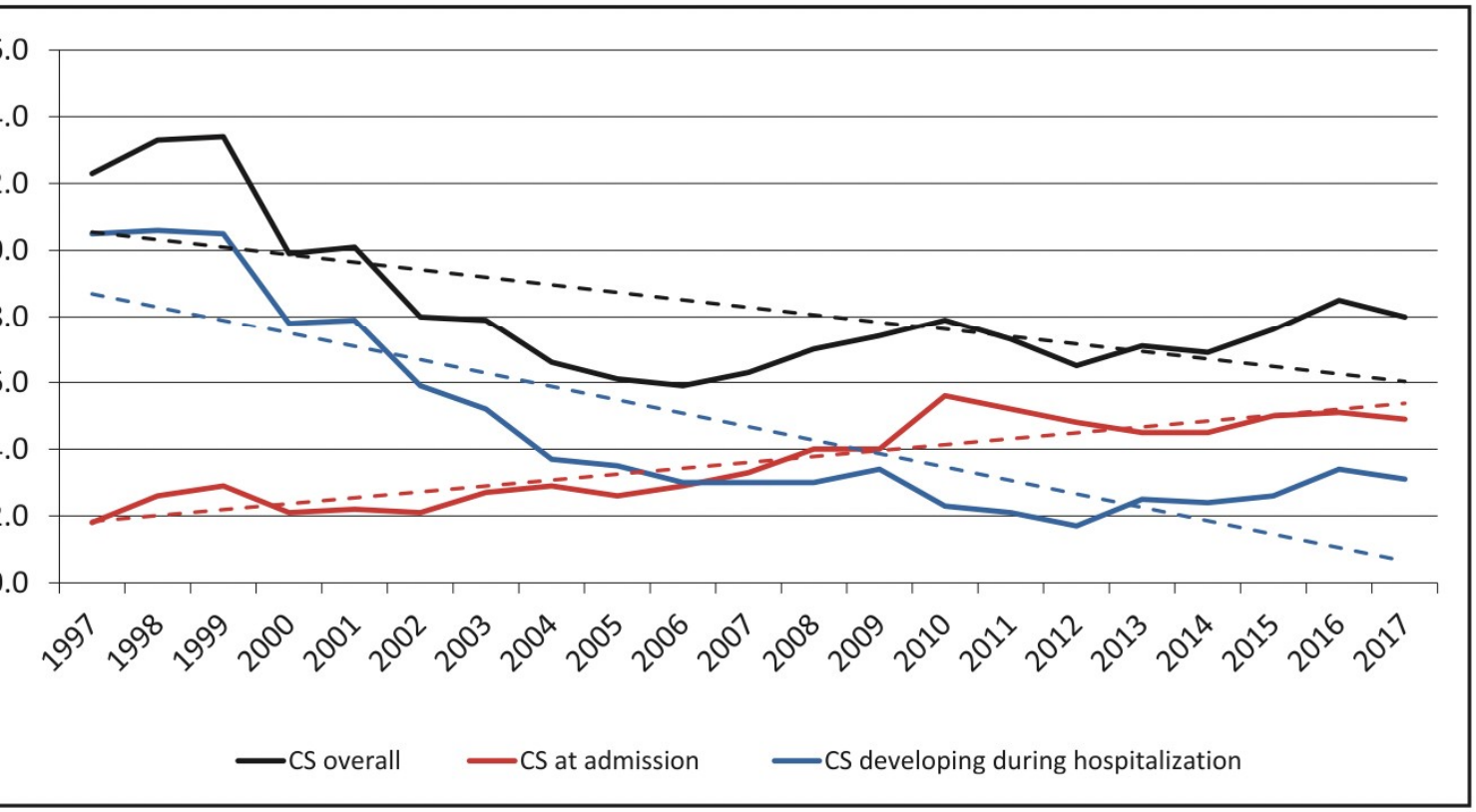
cidence of CS due to AMI



- The risk for CS following acute myocardial infarction (AMI) has significantly decreased from 8.7% 7.3% from 1997 to 2017 ($p < 0.0$
- In contrast, the risk of CS due to heart failure (HF) is steadily increasing.

Hunziker L et al; Circ Cardiovasc Interv. 20

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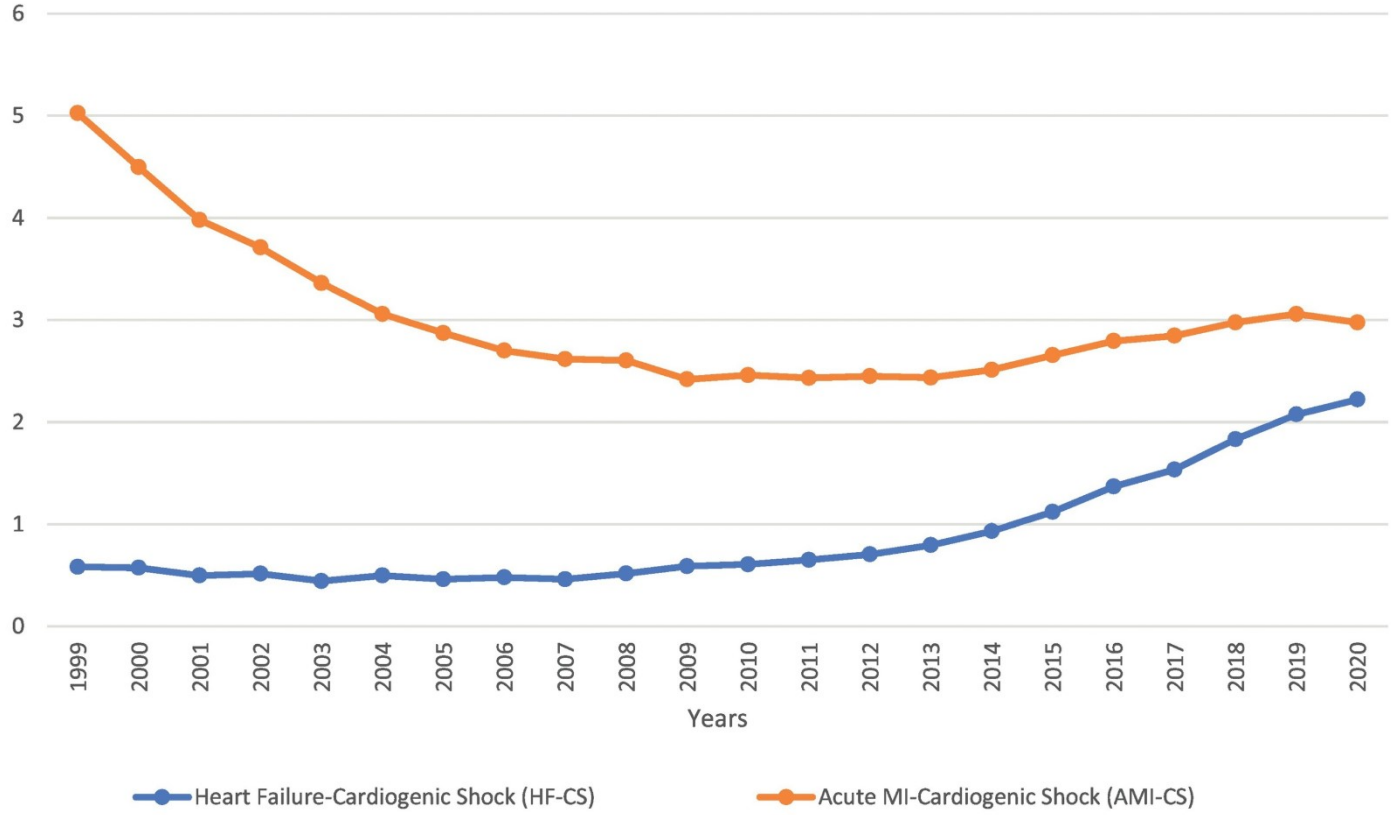
ortality trends im AMI- and HF-related Cardiogenic Shock



- Age-adjusted mortality (AAMR) f
  AMI-CS decreased significantly f
  1999 to 2009 (-6.9% [95%CI -7.7
  −6.1]) then stabilized from 2009 t
  2020.

- HF-CS associated AAMR rose
  steadily from 2009 to 2020 (+ 13
  [95%CI 11.4,15.2].

Ghajar A et al; Int J Cardiol. 2022

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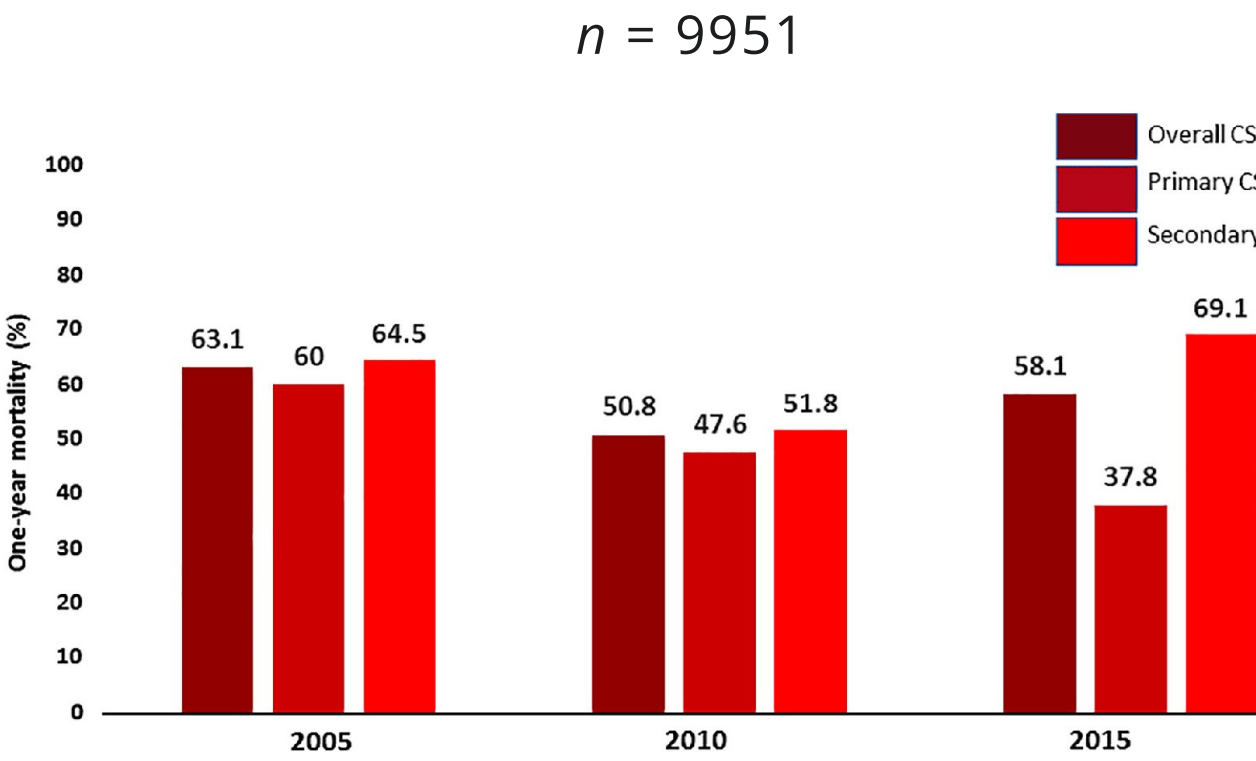
Cardiogenic shock related mortality remains high at about 40-60% at 30 days.

Recent studies investigating the prognosis of CS were commonly restricted to patients with AMI-CS.

Data investigating predictors of outcomes in CS of any cause remains limited.



Aissaoui N et al; Eur J Heart Fail. 2020.

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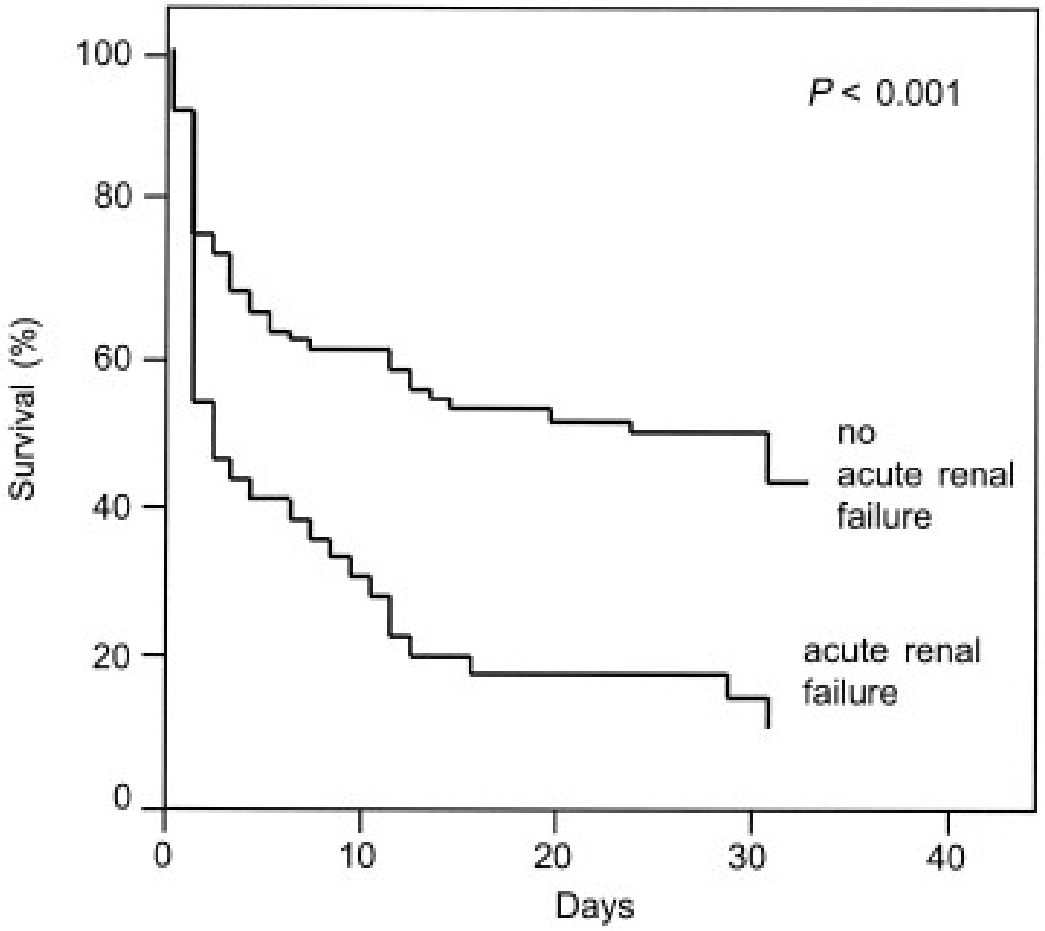
- Short-term mortality in patients with CS may be attributed to end organ damage.
- Acute kidney injury (AKI) may occur in more than 1/3 of CS patients and in 1/2 of patients treated on an ICU.
- AKI was yet demonstraed to increase the risk of all-cause mortality in CS patients.
- Most studies had low sample size and were restricted to AMI-CS patients

N=118 patients with AMI-CS from 1993 to 2000.



Koreny M, Am J Med. 2

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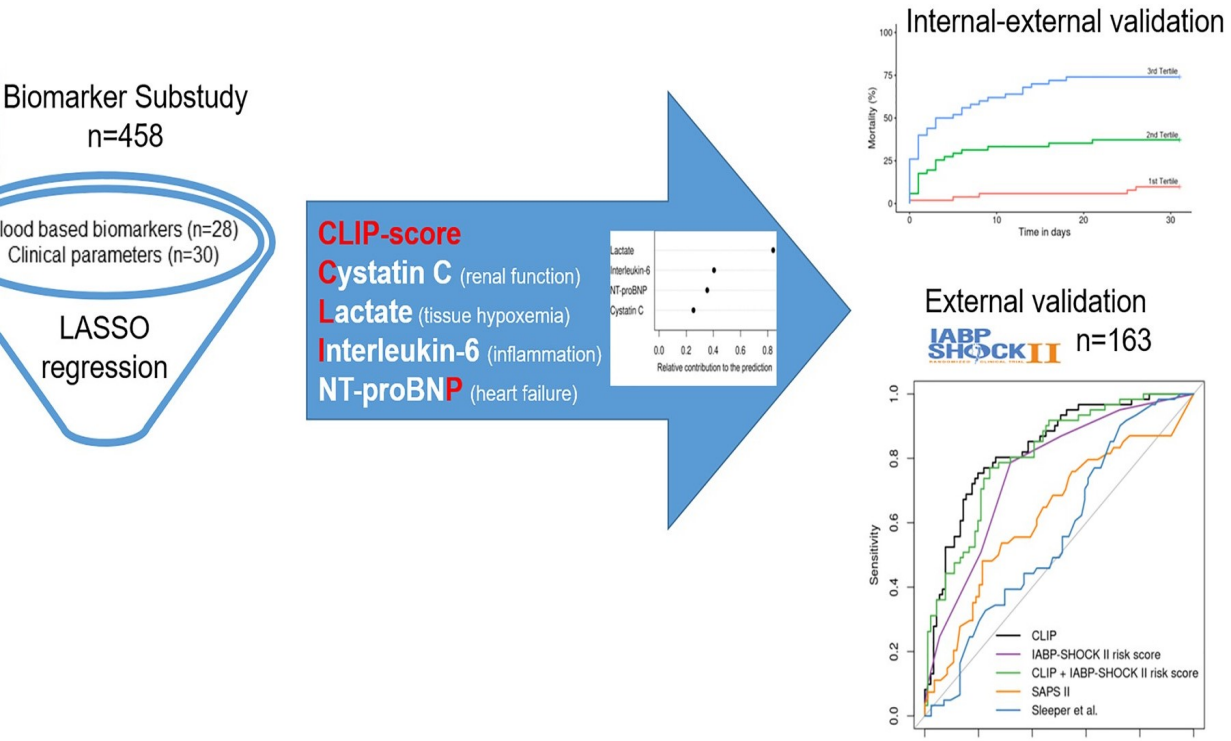
Fast, objective biomarker-based score for cardiogenic shock prognosis estimation



- Furthermore, the inclusion of cystatin c was shown to improve risk prediction in AMI-CS.
- However, cystatin c is unfrequently measured in patients with CS.
- In daily clinical practice, creatinine and eGFR remain the most-frequently biomarkers for the estimation of renal function.

Ceglarek et al. European Heart Journal,

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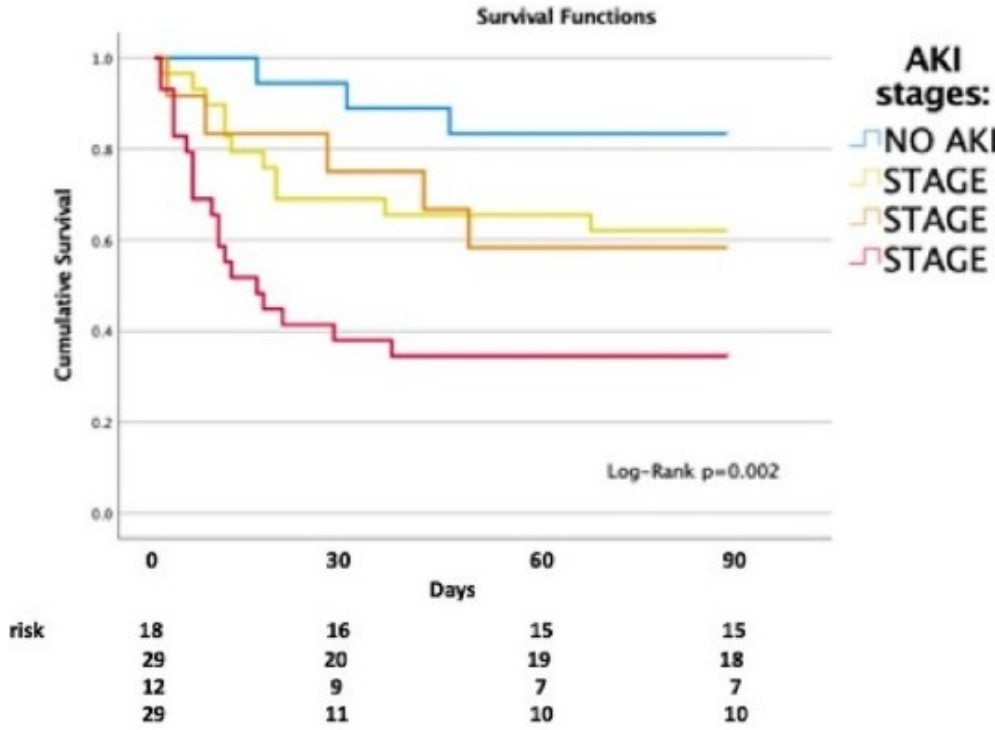
Acute kidney injury in HF-related CS

- Studies investigating the outcomes of AKI in HF-CS were limited to small sample sizes.
- Increased central venous pressure and lactate were predictors of AKI.
- AKI – and more advanced stages of AKI – indicated higher short-term mortality.



Predictors and the effect of AKI need to be verified in studies with higher sample sizes.

Bottiroli Int J Cardio

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rdiogenic Shock Registry Mannheim (CARESMA-registry)

n = 273 consecutive patients with cardiogenic shock of any etiology

admitted to the internistic ICU at University Medical Centre Mannheim (UMM)

From June 2019 to May 2021

Risk stratification was performed according to the presence or absence of acute kidney injury, defined as

- an increase of creatinine > 50% within 48 h referring to baseline creatinine on day 1
- an increase of plasma creatinine > 50% rerferring to pre-admission creatinine
- need for continuous veno-venous haemodiafiltration (CVVHDF)).

primary endpoint: 30-day all-cause mortality

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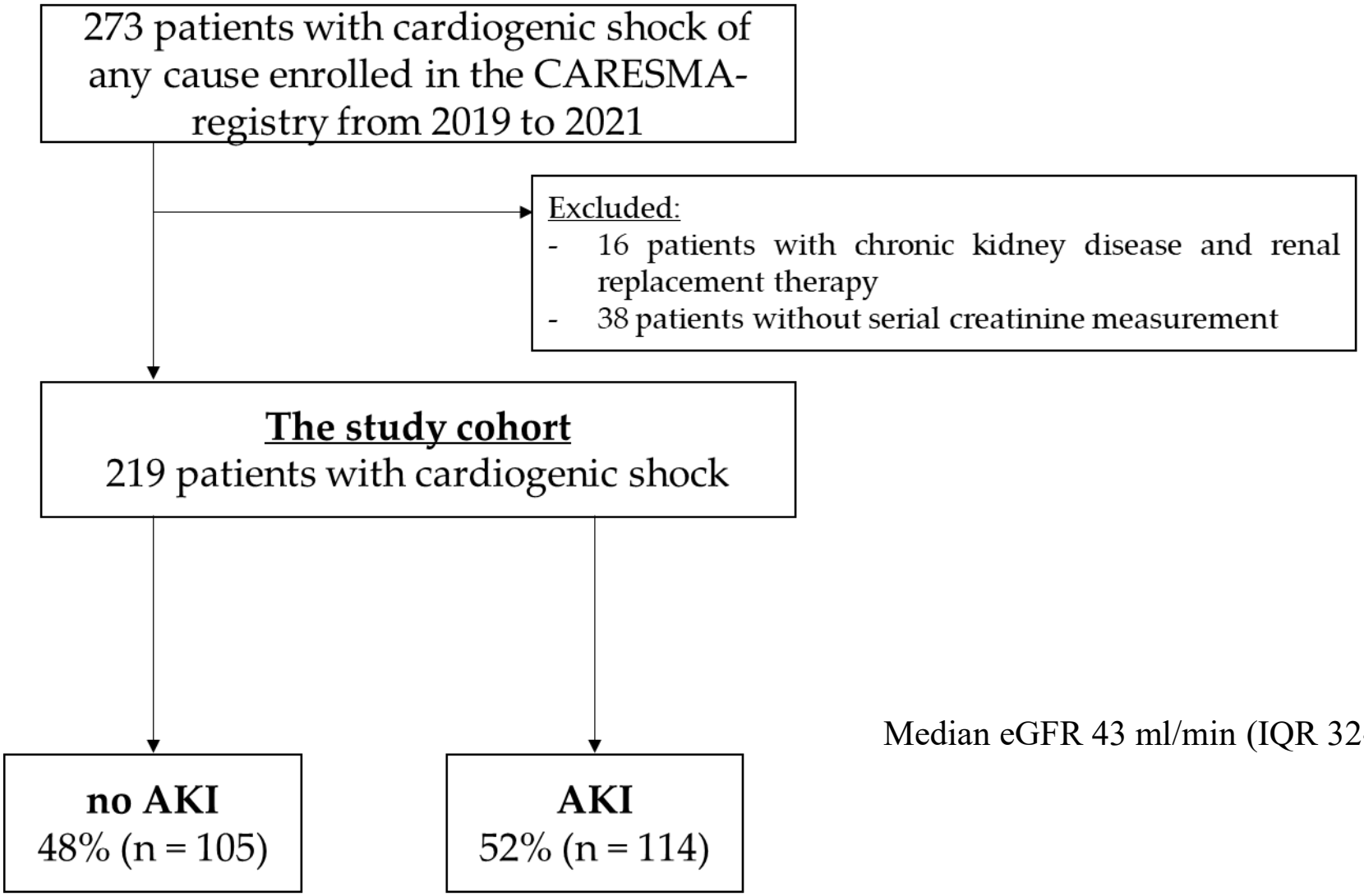

Median eGFR 43 ml/min (IQR 32-60 ml/min)

DGK.

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Table 1. Baseline characteristics.

		No AKI (n=105)		AKI (n=114)	p value
Age, median; (IQR)	74	(64-81)	72	(60-79)	0.263
Male sex, *n* (%)	61	(58.1)	72	(63.2)	0.443
Body mass index, kg/m^2 (median, (IQR))	26.1	(23.9-29.3)	27.0	(24.7-31.0)	**0.039**
Vital signs, (median, (IQR))					
Body temperature (°C)	36.0	(34.8-36.5)	36.0	(35.0-36.8)	0.338
Heart rate (bpm)	84	(69-106)	91	(72-108)	0.489
Systolic blood pressure (mmHg)	115	(94-139)	106	(93-124)	**0.029**
Respiratory rate (breaths/min)	18	(16-22)	20	(17-25)	**0.049**
Cardiovascular risk factors, *n* (%)					
Arterial hypertension	74	(70.5)	87	(76.3)	0.328
Diabetes mellitus	31	(29.5)	53	(46.5)	**0.010**
Hyperlipidemia	48	(45.7)	69	(60.5)	**0.028**
Smoking	37	(35.2)	56	(40.4)	0.436
Prior medical history, *n* (%)					
Coronary artery disease	31	(29.5)	47	(41.2)	
1-vessel disease	3	(2.9)	17	(14.9)	**0.019**
2-vessel disease	4	(3.8)	5	(4.4)	
3-vessel disease	24	(22.9)	25	(21.9)	
Congestive heart failure	28	(26.7)	48	(42.1)	**0.016**
Atrial fibrillation	34	(32.4)	36	(31.6)	0.899
Chronic kidney disease	26	(24.8)	44	(38.6)	**0.028**
Stroke	11	(10.5)	20	(17.5)	0.134
COPD	15	(14.3)	29	(25.4)	**0.040**
Liver cirrhosis	2	(1.9)	7	(6.1)	0.115
Medication on admission, *n* (%)					
ACE-inhibitor	37	(35.2)	41	(36.0)	0.911
ARB	19	(18.1)	17	(14.9)	0.525
Beta-blocker	51	(48.6)	57	(50.0)	0.833
ARNI	5	(4.8)	3	(2.6)	0.401
Aldosterone antagonist	14	(13.3)	20	(17.5)	0.390
Diuretics	37	(35.2)	56	(49.1)	**0.038**
ASA	23	(21.9)	35	(30.7)	0.141
P2Y12-inhibitor	7	(6.7)	11	(9.6)	0.422
Statin	41	(39.0)	59	(51.8)	0.059

ACE, angiotensin-converting-enzyme; ARB, angiotensin receptor blocker; ARNI, angiotensin receptor neprilysin inhibitor; ASA, acetylsalicylic acid; COPD, chronic obstructive pulmonary disease; IQR, interquartile range

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Table 2. Shock-related data, follow-up data and endpoints.

	No AKI (n=105)		AKI (n=114)		p value
Coronary angiography, *n* (%)	77	(73.3)	80	(70.2)	0.604
No evidence of CAD	13	(16.9)	8	(10.0)	
1-vessel disease	9	(11.7)	19	(23.8)	0.125
2-vessel disease	16	(20.8)	11	(13.8)	
3-vessel disease	39	(50.6)	42	(52.5)	
Left main trunk	10	(13.0)	8	(10.0)	0.557
Left anterior descending	46	(59.7)	41	(51.2)	0.285
Right coronary artery	37	(48.1)	36	(45.0)	0.702
Left circumflex	35	(45.5)	41	(51.2)	0.468
PCI, n (%)	48	(62.3)	57	(71.3)	0.236
Number of Stents, (median; (IQR))	1	(0-2)	1	(0-3)	0.349
CABG, n (%)	9	(11.7)	9	(11.3)	0.931
Chronic total occlusion, n (%)	19	(24.7)	19	(23.8)	0.892
Classification of CS, *n* (%)					
Stage A	0	(0.0)	0	(0.0)	
Stage B	3	(2.9)	2	(1.8)	
Stage C	39	(37.1)	43	(37.7)	0.305
Stage D	5	(4.8)	13	(11.4)	
Stage E	58	(55.2)	56	(49.1)	
Transthoracic echocardiography					
LVEF >55%, (*n*, %)	17	(16.2)	9	(7.9)	
LVEF 54-41%, (*n*, %)	10	(9.5)	19	(16.7)	
LVEF 40-30%, (*n*, %)	28	(26.7)	24	(21.1)	**0.029**
LVEF <30%, (*n*, %)	43	(41.0)	60	(52.6)	
LVEF not documented, (*n*, %)	7	(6.7)	2	(1.8)	
VCI, cm (median, (IQR))	1.9	(1.4-2.2)	1.8	(1.6-2.2)	0.489
TAPSE, mm (median, (IQR))	16	(12-20)	14	(11-18)	0.154
Cardiopulmonary resuscitation					
OHCA, *n* (%)	48	(45.7)	38	(33.3)	0.120
IHCA, *n* (%)	10	(9.5)	18	(15.8)	
Shockable rhythm, *n* (%)	68	(64.8)	88	(77.2)	**0.042**
Non-shockable rhythm, *n* (%)	37	(35.2)	26	(22.8)	
ROSC, min (median, IQR)	12	(6-20)	20	(10-35)	**0.002**
Multiple organ support during ICU					
Norepinephrine dose, µg/kg/min (median, (IQR))	0.1	(0.0-0.2)	0.2	(0.1-0.6)	**0.001**
Mechanical circulatory assist device, *n* (%)	5	(4.8)	17	(14.9)	**0.013**
CVVHDF, *n* (%)	0	(0.0)	69	(60.5)	**0.001**
Baseline laboratory values, (median, (IQR))					
pH	7.32	(7.25-7.39)	7.27	(7.19-7.36)	**0.001**
Lactate (mmol/l)	2.6	(1.5-4.1)	4.0	(2.1-7.7)	**0.001**
Sodium (mmol/l)	139	(136-141)	138	(135-141)	0.309
Potassium (mmol/l)	4.1	(3.7-4.6)	4.4	(3.9-5.4)	**0.004**
Creatinine (mg/dl)	1.25	(0.94-1.60)	1.62	(1.34-2.52)	**0.001**
Hemoglobin (g/dl)	12.6	(10.6-14.0)	12.7	(10.3-14.3)	0.959
WBC (10^6/ml)	12.67	(9.85-17.73)	16.40	(11.70-20.20)	**0.010**
Platelets (10^6/ml)	225	(173-271)	222	(174-275)	0.816
Troponin I (µg/l)	0.763	(0.136-5.332)	0.716	(0.159-6.037)	0.725
NT-pro BNP (pg/ml)	3169	(431-15066)	5680	(1094-13486)	0.301
Procalcitonin (ng/ml)	0.28	(0.06-0.42)	0.29	(0.16-1.06)	0.439
CRP (mg/l)	6	(4-28)	15	(4-57)	0.062

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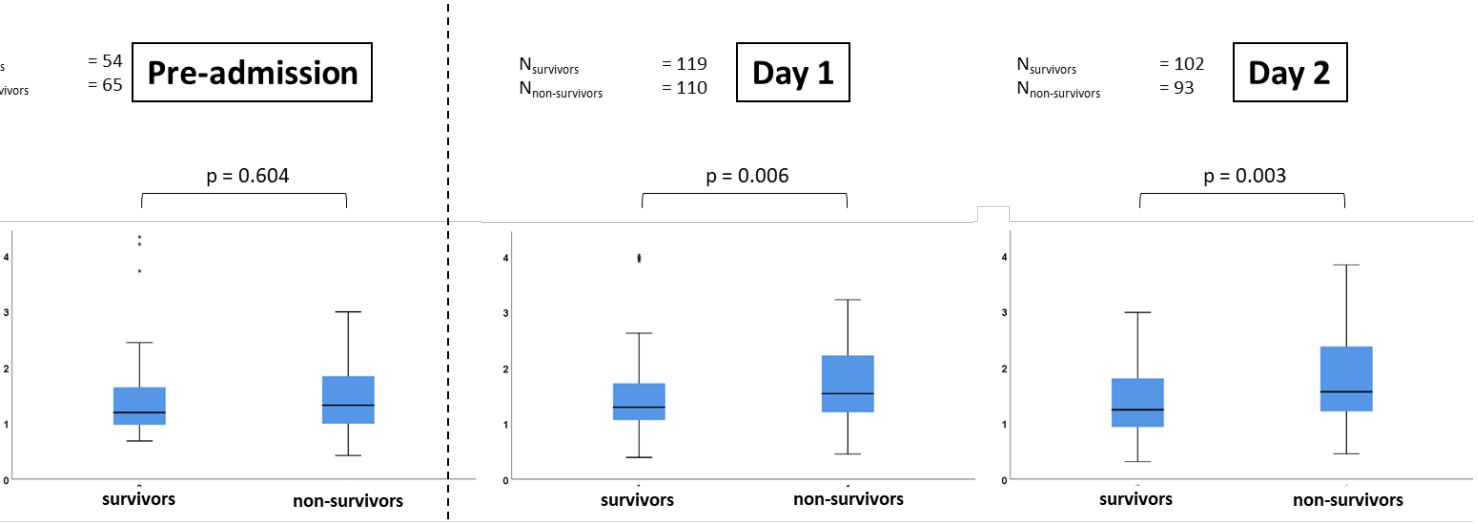

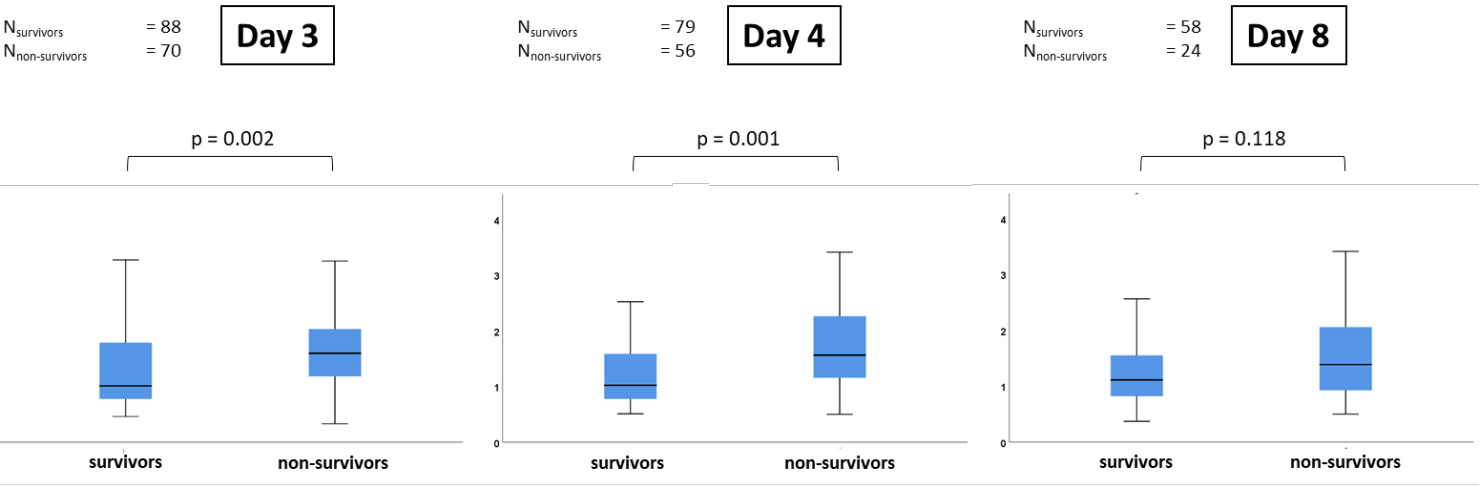

Creatinine levels were consi
higher in CS non-survivors durir
first week of ICU treatment.

In contrast, pre-admission crea
levels did not differ among
survivors and survivors.

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Correlations of creatinine with clinical and laboratory data

Table 3. Correlations of creatinine with laboratory and clinical parameters in all patients on day 1.

	Creatinine	
	r	**p value**
Age	0.229	**0.001**
Body mass index (kg/m^2)	0.163	**0.018**
Heart rate (bpm)	-0.113	0.102
Systolic blood pressure (mmHg)	-0.208	**0.002**
PaO_2/FiO_2 ratio	-0.071	0.336
PaO_2 (mmHg)	-0.112	0.115
Norepinephrine (µg/kg/min)	-0.021	0.759
Hemoglobin (g/dL)	-0.240	**0.001**
WBC (10^6/mL)	0.079	0.252
Platelet count (10^6/mL)	-0.211	**0.002**
INR	0.203	**0.004**
Bilirubin (mg/dL)	-0.014	0.869
cTNI (µg/L)	-0.058	0.428
NT-pro BNP (pg/mL)	0.427	**0.001**
CRP (mg/L)	0.347	**0.001**
Procalcitonin (ng/mL)	0.231	0.060

CRP, C-reactive protein; cTNI, cardiac troponin I; INR, international normalized ratio; NT-pro BNP, aminoterminal pro-B-type natriuretic peptide; WBC, white blood cells.
Level of significance $p<0.05$. Bold type indicates statistical significance.

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Predictors for AKI in all-comers patients with CS.

Table 4. Predictors of acute kidney injury within the entire study cohort.

	OR	95% CI	p value
Age (per 1 year increase)	1.003	0.981-1.027	0.772
Male sex	0.744	0.390-1.422	0.371
Heart rate (bpm) (per 1 bpm increase)	0.994	0.982-1.005	0.275
Lactate (per 1 mmol/l increase)	1.194	1.083-1.316	**0.001**
CRP (per 1 mg/l increase)	1.003	0.997-1.008	0.306
Norepinephrine (per 1 µg/kg/min increase)	1.509	0.882-2.583	0.134
Non-AMI vs. AMI	1.278	0.683-2.391	0.442
Cardiopulmonary resuscitation	0.784	0.470-1.306	0.350

AMI, acute myocardial infarction; CRP, C-reactive protein.
Level of significance $p<0.05$. Bold type indicates statistical significance.

Lactate on admission was the only predictor of AKI in patients with CS.

In contrast, the etiology of CS had no impact on the risk of AKI.

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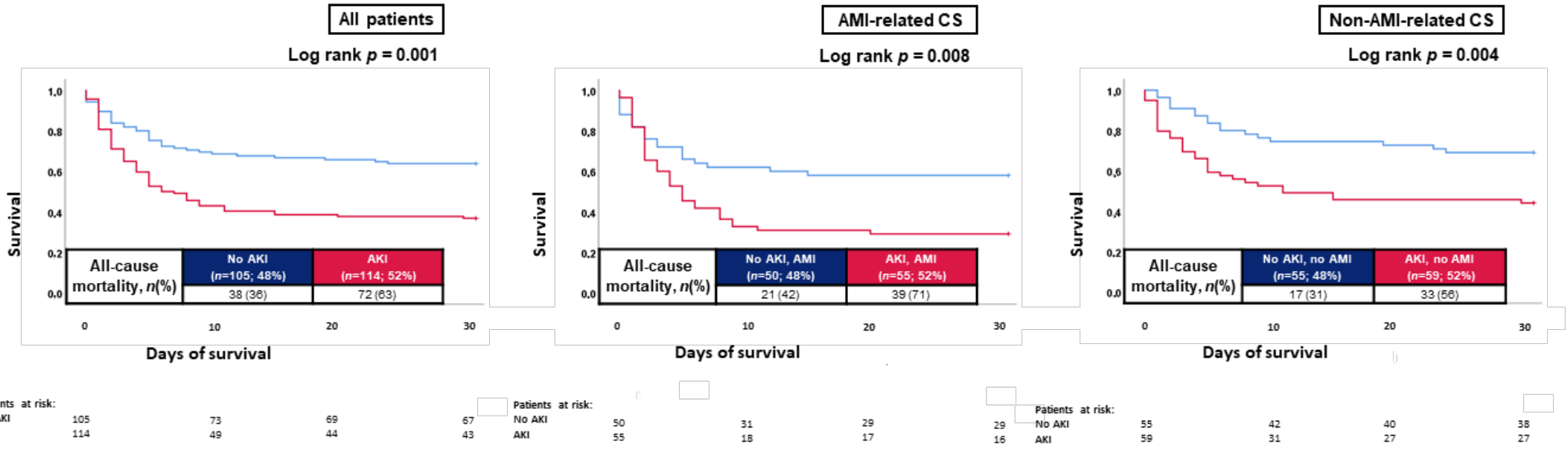

ne presence of AKI was associated with an increased risk of 30-day all-cause mortality (63% . 36%; HR = 2.138; 95% CI 1.441 – 3.171 p = 0.001).

his association was observed irrespective of CS etiology (AMI-related CS: 71% vs. 42%; log nk p = 0.008; HR = 1.979; 95% CI 1.160 – 3.374; p = 0.012; non-AMI-related CS (56% vs. %; log rank p = 0.004; HR = 2.284; 95% CI 1.271 – 4.105; p = 0.006).

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Table 5. Multivariate Cox regression analysis with regard to 30-day all-cause mortality.

Variables	HR	95% CI	p value
Age (per 1 year increase)	1.022	1.005-1.039	**0.010**
Male sex	1.039	0.685-1.575	0.857
Heart rate (per 1 bpm increase)	1.007	0.999-1.015	0.105
Lactate (per 1 mmol/l increase)	1.086	1.033-1.142	**0.001**
CRP (per 1 mg/l increase)	0.998	0.995-1.001	0.190
Norepinephrine (per 1 µg/kg/min increase)	1.209	0.993-1.472	0.058
Non-AMI vs. AMI	1.358	0.896-2.059	0.149
Cardiopulmonary resuscitation	1.169	0.874-1.563	0.293
Acute kidney injury	1.861	1.207-2.869	**0.005**

CRP, C-reactive protein. Level of significance $p<0.05$. Bold type indicates statistical significance.

gher age, lactate and the presence of AKI were demonstrated to be independent edictors of short-term mortality in CS patients.

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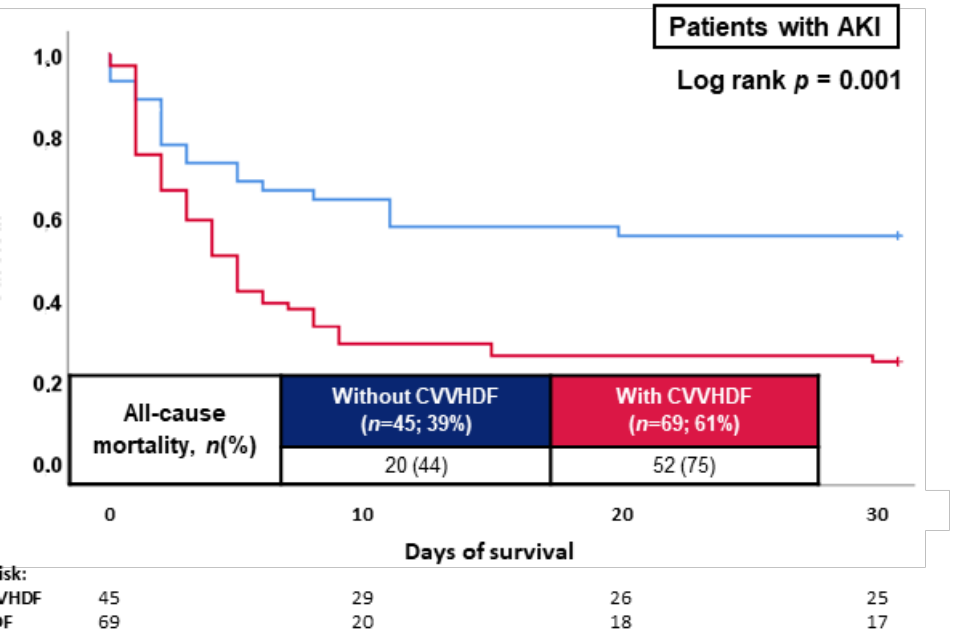

Among patients with AKI, CVVHDF was required in 61%.

CVVHDF was associated with worse prognosis compared to AKI without CVVHDF (75% vs. 44%; log rank p = 0.001; HR = 2.211; 95% CI 1.315 – 3.718; p = 0.003).

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Key messages

Earyl AKI is common in patients with CS and affects more than half of CS patients.

Lactate as a surrogate end organ hypoperfusion was the only predictor for the development of AKI.

AKI is independently associated with adverse outcomes in CS – irrespective of the CS etiology.

Further predictors of short-term mortality were higher age and lactate levels.

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Thank you for your attention!



th a special thank you to the main contributers:

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ahim Akin.