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Acute kidney injury in intensive care patients: Incidence, time course, and risk factors

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Abstract

Background: Acute kidney injury (AKI) is frequent and influences the prognosis of intensive care unit (ICU) patients. The aim of this study was to estimate the incidence, time-course, risk factors, and mortality of AKI among unselected ICU patients.

Methods: All adult ICU patients admitted to the ICU at the University Hospital in Central Norway from 2010 to 2015 with a stay of 24 h or more were included in the study. AKI was defined according to the Kidney Disease: Improving Global Outcomes (KDIGO) guidelines. All patients were followed with respect to reversal of AKI. Risk factors for AKI were analyzed using Cox regression.

Results: Among 2325 ICU patients, 1245 developed AKI during the ICU stay, corresponding to an incidence of 53.5 % (CI, 51.5–55.5). The incidence according to KDIGO AKI stages 1, 2, and 3 was 26.2, 11.7, and 15.7%, respectively. The median duration of AKI was 24 (CI 19–24), 32 (CI 26–39), and 101 (CI 75–164) hours for AKI KDIGO stage 1, 2, and 3, respectively. AKI was transient, persistent, or AKD in 73.4, 16.5, and 10.0% of the patients with a known outcome. AKI reversal was observed in 72.9% of all AKI patients. Independent risk factors for AKI in a multivariate analysis were hypertension, diabetes, heart disease, and higher body weight. Episodes of mean arterial pressure below 73 mmHg were associated with a higher risk of AKI.

Conclusions: In our material, the incidence of AKI was comparable to what has been reported previously. Risk factors for the development of AKI were a MAP below 73, hypertension, diabetes, heart disease, chronic kidney disease, and higher body weight. Most AKI patients regain their kidney function during the ICU stay, particularly in the KDIGO AKI stages 1 and 2.

Editorial Comment

Acute kidney injury in a single-center intensive care cohort was analyzed here, including for the development or resolution over time. The findings confirm previously reported adjusted risks and demonstrate later improved kidney function largely in those with less severe injury to start.

1 | INTRODUCTION

Acute kidney injury (AKI) is frequently observed in critically ill patients. AKI is associated with an increased mortality and length of intensive care unit (ICU) stay.[1] AKI also increases the risk of chronic kidney disease, including long-term dialysis and the possible need for organ transplantation.[2–4] AKI is a challenge for health care globally, as evidence suggests that even minor episodes of AKI can affect long-term renal health.[2–4]

The prevalence of AKI in ICU patients has been assessed in several previous studies.[5,6] However, both patient characteristics and interventions in the ICU have changed in the past decades, and many studies have not applied the currently recommended classification system including lower grade AKI. Therefore, new data reporting the incidence of AKI in critically ill ICU patients with a current recommended classification of AKI is warranted. In addition, few previous studies have described the incidence and course of AKI for the entire duration of the ICU admission.[7]

There are several potential causes for the development of AKI.[1,8] Some patients have known risk factors such as diabetes, hypertension, or lower grade chronic renal failure. The inflammatory response associated with critical illness may induce AKI. Also, extra renal factors such as nephrotoxic drugs, hypoxia, hypovolemia, and arterial hypotension may be part of the pathogenesis of AKI in critically ill patients.[1,5] Data from the FINNAKI study showed the influence of arterial hypotension on the development of AKI in an ICU population with sepsis. The study reported that hypotensive episodes (mean arterial pressure [MAP] <73) were associated with a progression of AKI.[9] It is of interest to observe whether similar findings are present in an unselected ICU population.

The aim of the study was to describe the incidence and time course of AKI using recent data and definitions, to identify risk factors for AKI in an unselected ICU population, and to assess AKI-related outcomes in the ICU.

2 | METHODS

2.1 | Setting and patients

The study was performed in a mixed care 10-bed ICU at the 900-bed St Olavs Hospital, Trondheim University Hospital in Central Norway. The ICU cares for all categories of adult patients except post-surgical cardiac surgery and transplant patients.

All adult ICU patients admitted in the years 2010–2015 with a stay of 24 h or more were included in the study. Organ donors and patients without a valid Norwegian social security number were excluded. In addition, patients with insufficient data for analysis were excluded. ICU readmissions within 48 h were combined with the primary admission and considered as one admission.

2.2 | Data collection

This retrospective study used patient data collected from the local administrative patient database, the electronic data recording system in the ICU (Picis 8.0/8.2, Picis Clinical Solutions, Inc.), and the hospital electronic record system (Doculive EPR, Siemens/Cerner corp. Versions 6.3–6.9). The electronic data recording system stored variables at least every 15 min, automatically collecting clinical variables except urine output, which was measured manually every hour. Serum creatinine concentrations were obtained daily. The severity of the disease was expressed as simplified acute physiology scores (SAPS2).[10]

Baseline characteristics were collected from all data sources. Comorbidities were identified by a text-based search of the admission papers. The patients were followed up with respect to survival for 1 year after the ICU stay.

2.3 | Definitions

AKI was defined by the Kidney Disease: Improving Global Outcomes (KDIGO) criteria.[11] Baseline creatinine serum concentration was defined as the last creatinine value measured 8 to 365 days before admittance to the ICU. If no such value existed, the creatinine serum concentration value was estimated using the MDRD equation, back calculating with an estimated Glomerular filtration rate (GFR) set to 75.[12]

AKI was assessed and staged according to the KDIGO criteria for all available records of hourly urine output and creatinine serum concentration measurements for the entire duration of the ICU admission. Each creatinine measurement was carried forward for 24 h until the next measurement to ensure a creatinine value was available with each hourly record of urine output. For KDIGO staging, baseline creatinine was compared with the highest value obtained during the ICU admission regardless of the length of stay. Recovery of AKI was defined as reversal of the oliguria and/or return of plasma creatinine to the baseline value.[13,14] For patients meeting both the oliguria-based and plasma creatinine change-based definitions of AKI, correction of both plasma creatinine and oliguria were required to define recovery. "Persistent AKI" was defined as renal dysfunction without recovery within 3 days. Rolling windows of 48 and 168 h were used to assess if AKI or AKI-reversal had occurred. To avoid classifying temporary improvements as a reversal, only the last occurrence of reversal during the ICU stay was used in the analysis. AKI duration was defined as the time from the first occurrence of AKI to the time of AKI reversal. AKI was classified as transient, persistent, or acute kidney disease (AKD), when reversal ensued within 48 h, 48–168 h, and more than 168 h, respectively.[15]

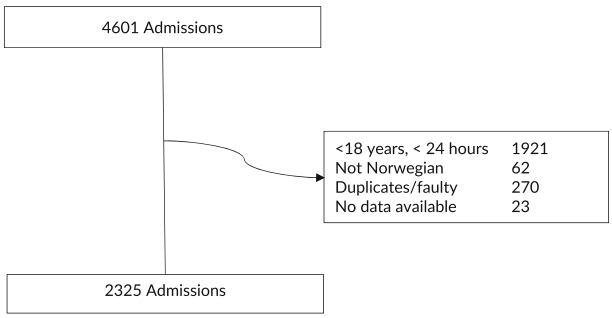

FIGURE 1 Inclusion flowchart

2.4 | Hypotension

To assess the effect of blood pressure (BP) on the risk for AKI, several variables were calculated. First, for all measurements, a time-average of mean arterial pressure (TAMAP) was calculated by dividing the mean arterial pressure area under the curve (AUC) by the duration of measurements. Second, a hypotensive dose was estimated, essentially as described by Poukkanen et al.[9] In brief, to describe hypotensive dose as a time-weighted average of below mean arterial blood pressure thresholds (TWA-MAP<73 mmHg), the AUC below a threshold of MAP of 73 mmHg was calculated and divided by the total observation time until AKI or discharge. The same calculation was done with a threshold of 65 mmHg. Riemann integrals were used to estimate AUC. Third, to eliminate inter-individual differences using patients as their own control, the TAMAP and TWA-MAP<73 mmHg from hour 24–18 before AKI was compared with the last 6 h before AKI.

2.5 | Statistics

Demographic variables are presented as median (IQR, min-max) or mean (SD) as appropriate.

Kaplan–Meier analysis was performed using log-rank tests with reversal as events, and patients were censored at the time of death or discharge. KDIGO AKI score vs ADQI status was analyzed with Fisher's exact test for count data.

We used multiple logistic regression to estimate the crude and adjusted odds ratio (OR) for AKI associated with baseline characteristics believed to be associated with AKI[16] and variables with a potential

TABLE 1 Patient characteristics

	All	No AKI	AKI
Number of patients (*n*)	2325	1080	1245
Age (years)	63.8 (51, 73)	59.8 (43, 70)	66.5 (56, 74)
Male (*n* [%])	1422 (61.2%)	652 (60.4%)	770 (61.8%)
Weight (kg)[a]	80 (68, 90)	76 (66, 77)	80 (70, 83)
Comorbidities (*n* [%])			
Diabetes	342 (14.7%)	119 (11%)	223 (17.9%)
Hypertension	676 (29.1%)	258 (23.9%)	418 (33.6%)
Heart disease	344 (14.8%)	122 (11.3%)	222 (17.8%)
COPD	288 (14.8%)	125 (11.6%)	163 (13.1%)
CKD	84 (3.6%)	12 (1.1%)	72 (5.8%)
Primary reason for admittance to the ICU (*n* [%])			
Respiratory	545 (23.4%)	258 (23.9%)	287 (23.1%)
Circulatory	408 (17.5%)	146 (13.5%)	262 (21.0%)
Trauma	284 (12.2%)	188 (17.4%)	96 (7.7%)
CNS	272 (11.7%)	192 (17.8%)	80 (6.4%)
Infection	266 (11.4%)	91 (8.4%)	175 (14.1%)
Post-operative	262 (11.3%)	120 (11.1%)	142 (11.4%)
Gastrointestinal	204 (8.8%)	65 (6.0%)	139 (11.2%)
Renal	39 (1.7%)	2 (0.2%)	37 (3.0%)
Endocrine	25 (1.1%)	9 (0.8%)	16 (1.3%)
Poisoning	20 (0.9%)	9 (0.8%)	11 (0.9%)
Ventilatory support (*n* [%])	1951 (84.0%)	895 (82.9%)	1056 (83.9%)
Days on ventilator (days)	3.9 (1.5, 8.5)	3.0 (1.17, 6.3)	5.2 (1.9, 10.5)
SAPS2	38 (29, 50)	35 (25, 44)	42 (33, 54)
CRRT (*n*)	142		
RRT (including HD) (*n*)	169		
S-Creatinine at admittance (mmol/l)	84 (60, 134)	67 (53, 84)	135 (90, 215)
Baseline S-creatinine (mmol/l)	84 (67, 92)	81 (67, 91)	85 (68, 93)
ICU length of stay (hours)	91 [44–202]	70 [39–151]	118 [48–251]

Abbreviations: COPD, chronic obstructive pulmonary disease; CKD, chronic kidney disease; CNS, Central nervous system; CRRT, Continuous renal replacement therapy; HD, Hemodialysis (intermittent); RRT, Renal replacement therapy; SAPS2, simplified acute physiology score.

[a]BMI (Body Mass Index) could not be calculated due to missing data on height.

putative effect on renal function. These variables included body weight, SAPS2, hypertension, diabetes, heart disease, chronic obstructive lung disease, and chronic kidney disease (CKD). All associations were adjusted for age and sex.

In the subsample of 1499 patients without clinical evidence of possible AKI at admission, we used Cox regression to estimate hazard ratios (HRs) for a first AKI associated with measures of hypotension at MAP 73 and MAP 65. All HRs were adjusted for age, as well as possible confounding by body weight, diabetes, and cardiovascular disease at admission. The proportional hazards assumption for the hypotension variables was evaluated by the test of Schoenfeld residuals and graphical inspection of log-minus-log plots. To assess the robustness of the results, we conducted sensitivity analyses excluding patients who developed AKI less than 24 h after ICU admission, to reduce potential bias due to already existing AKI at admission. To further reduce possible bias due to interindividual differences in disease severity, we applied a case-crossover design among the 121 patients who developed AKI 48 h or more from the time of admission. Conditional logistic regression was used to estimate ORs for AKI associated with TWA-MAP<73 mmHg and TAMAP. Here, blood pressure readings during 18–24 h before AKI constituted the control period, and blood pressure during the last 6 h before AKI was used as the case period.

The precision of all estimated associations was assessed by 95% confidence intervals (CIs).

The statistical analyses were made with R software v 4.05 (with packages survival, survminer, and rstatix),[17] and STATA 17 (©1985–2021 Stata Corp LLC).

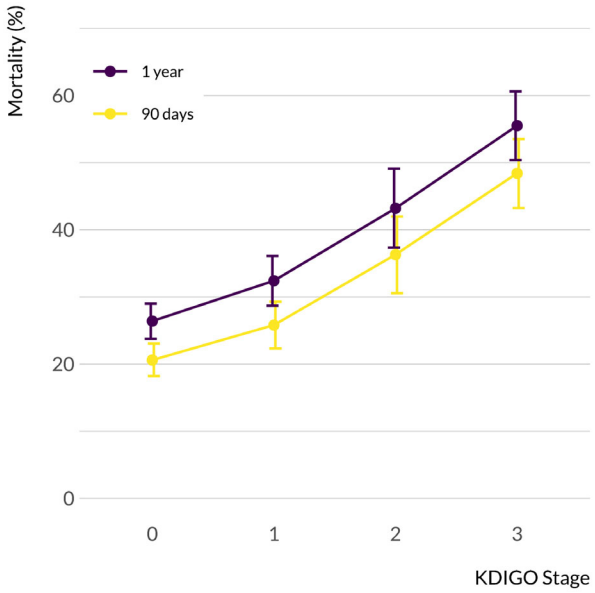

FIGURE 3 Mortality at 90 days and 1 year related to KDIGO stage. (95%CI for within-group comparison)

TABLE 2 Number and proportion of deaths according to AKI status

	All	No AKI	AKI
Death in the ICU (*n* [%])	294 (12.6%)	86 (8%)	208 (16.7%)
Death at 90 days (*n* [%])	655 (28.2%)	223 (20.6%)	432 (34.7%)
Death at 1 year (*n* [%])	802 (34.5%)	285 (26.4%)	517 (41.5%)

FIGURE 2 – Kaplan–Meier plot of AKI duration of AKI KDIGO stage 1, 2, and 3. The plot describes the trajectory of the proportion of patients with a given stage of AKI and still in the ICU.

2.6 | Ethics

The Regional Ethics Committee in central Norway (REK midt) considered the investigation a quality control study according to Norwegian law (Act on medical and health research, §§ 2 and 4), and had no objections against the study. The need for informed consent was waived. The data protection officer of St. Olavs hospital, Trondheim University Hospital approved the study.

3 | RESULTS

In the six-year period, a total of 4601 admissions to the ICU were registered, of which 1921 were patients with a length of stay less than 24 h or less than 18 years of age. Two hundred seventy duplicates or faulty registrations were excluded, 62 of the patients did not have a Norwegian social security number, and 23 did not have any urine output or creatinine data available (Figure 1).

TABLE 3 The odds ratio from multiple logistic regression of AKI related to characteristics at ICU admission in all 2325 patients

Variables	No of patients	No. of AKI cases	Crude OR	Adjusted[a] OR	95% CI
Age					
< 40	349	121	1.00		Reference
40–59	610	297	1.79		1.36–2.35
60–79	1149	677	2.70		2.11–3.47
≥ 80	217	150	4.22		2.93–6.06
Sex					
Male	1422	770	1.00		Reference
Female	903	475	0.94		0.80–1.11
Body weight (kg), quartiles[b]					
1st	681	311	1.00	1.00	Reference
2nd	548	288	1.32	1.33	1.05–1.67
3rd	544	280	1.26	1.26	1.00–1.59
4th	552	366	2.34	2.51	1.97–3.18
SAPS2, quartiles					
5–29	624	238	1.00	1.00	Reference
30–38	540	267	1.59	1.29	1.01–1.65
39–50	610	347	2.14	1.68	1.32–2.14
51–108	551	393	4.03	3.12	2.40–4.04
Hypertension					
No	1649	827	1.00	1.00	Reference
Yes	676	418	1.61	1.30	1.07–1.58
CKD					
No	2241	1173	1.00	1.00	Reference
Yes	84	72	5.46	4.60	2.47–8.56
Diabetes					
No	1983	1022	1.00	1.00	Reference
Yes	342	223	1.76	1.58	1.24–2.01
Heart disease					
No	1981	1023	1.00	1.00	Reference
Yes	344	222	1.70	1.55	1.22–1.98
COPD					
No	2037	1082	1.00	1.00	Reference
Yes	288	163	1.15	0.90	0.69–1.16

Abbreviations: AKI, acute kidney injury; CI, confidence interval; CKD, chronic kidney disease; COPD, chronic obstructive pulmonary disease; HR, hazard ratio; ICU, intensive care unit.

[a]Adjusted for age (<40, 40–59, 60–79, ≥80 years), sex (male, female).

[b]According to the sex-specific distribution (cut-offs 76, 83, and 94 kg in men and 61, 71, and 81 kg in women).

A total of 2325 patients were included in the study, comprising 369,856 h in the ICU. A total of 34,444 creatinine measurements, 362,265 diuresis measurements, and 1,420,430 measurements of blood pressures were analyzed.

The patient's median age was 63.8 years, 1422 (61%) were male, and the most frequent reasons for admission were respiratory failure and circulatory failure. The incidence of potential risk factors for AKI was hypertension (29.1%), diabetes (14.7%), heart disease (14.8%), chronic obstructive pulmonary disease (12.4%), and chronic kidney disease (3.6%). The median SAPS2 value was 38 (Table 1).

The overall incidence of AKI was 53.5% during the ICU stay. The maximum AKI KDIGO stage distribution was as follows: Stage 1; 26.2%, Stage 2; 11.7%, Stage 3; 15.7%. Renal replacement therapy was used in 169 (7.3%) of the patients. (Table 1). AKI was most frequently observed early in the ICU stay, with 90.1% of AKI cases occurring within the first 48 h. AKI was transient, persistent, or AKD in 73.4, 16.5, and 10.0% of the patients with a known outcome, respectively, when staged according to the Acute Disease Quality Initiative (ADQI) criteria.[15] The duration of AKI was estimated to be a median (95%CI) of 24 (19–24), 32 (26–39), and 101 (75–164) hours for AKI KDIGO stage 1, 2, and 3, respectively (Figure 2). (KDIGO 3 vs. 1, $p < .001$, 3 vs. 2, $p < .001$, 1 vs. 2, $p = .054$). The risk for AKD was significantly higher for patients having AKI KDIGO stage 3 compared with those having stages 1 and 2. In patients with known AKI outcomes, transient AKI was observed in all AKI KDIGO stages; 61% in stage 1, 60% in stage 2, and 36% in stage 3. The proportion with AKD was 4%, 6%, and 14% for AKI KDIGO stages 1, 2, and 3, respectively. A total of 338 of the AKI patients either died or were transferred from the ICU before the classification of the AKI outcome could be obtained.

The ICU length of stay was significantly longer for the AKI patients compared to the non-AKI patients; the median was 4.2 days compared to 2.9 days. ($p < .01$). At the time of AKI, the median remaining length of ICU stay was 75.5, 124.0, and 103.5 h for KDIGO AKI stage 1, 2, and 3, respectively ($p < .01$ for AKI KDIGO 1 vs 2 and 3, and $p = .32$ for AKI KDIGO 2 vs 3). The AKI patients also had an increased length of mechanical ventilation compared to patients without AKI (5.2 vs 3.0 days, $p < .001$ [Table 1]). The ICU mortality for the AKI patients was 16.7% compared to non-AKI 8.0%. The 90 days mortality for the AKI patients was 34.7%, for non-AKI 20.6%, and the 1-year mortality for the AKI patients was 41.5%, for non-AKI 26.4% (Table 2 and Figure 3).

Table 3 shows the association between factors measured at admission to the ICU and the occurrence of AKI during the ICU stay. Higher age, body weight, and SAPS2 score, as well as hypertension, diabetes, cardiovascular disease, and CKD were all associated with a higher OR for AKI.

3.1 | Mean arterial blood pressure

Patients with AKI had a lower average TAMAP (median [IQR]) 76.5 (70.5–83.5) vs 81.7(76.1–88.3) mmHg, $p < .001$, and a higher TWA-MAP<73 mmHg (median [IQR] 2.15 [0.51–5.0] vs 1.43 [0.49–2.87], $p < .001$). When splitting patient groups in four, based on their TAMAP after admission, the proportion of patients with AKI appeared to increase linearly if TAMAP was below 74 mmHg (Figure 4).

For every unit increase in TWA-MAP<73 mmHg, the relative risk of AKI increased by 9% (HR 1.09; 95% CI 1.06–1.13) (Table 4). Patients in the highest quartile of TWA-MAP<73 mmHg (3.13–25.73) had a HR of 1.41 (95% CI 1.07–1.84) compared to those in the lowest quarter (<0.52). Correspondingly, those who had 75% or more of their ICU time with a MAP <73 mmHg had a HR for AKI of 2.79 (95% CI 1.96–3.04) compared to those having

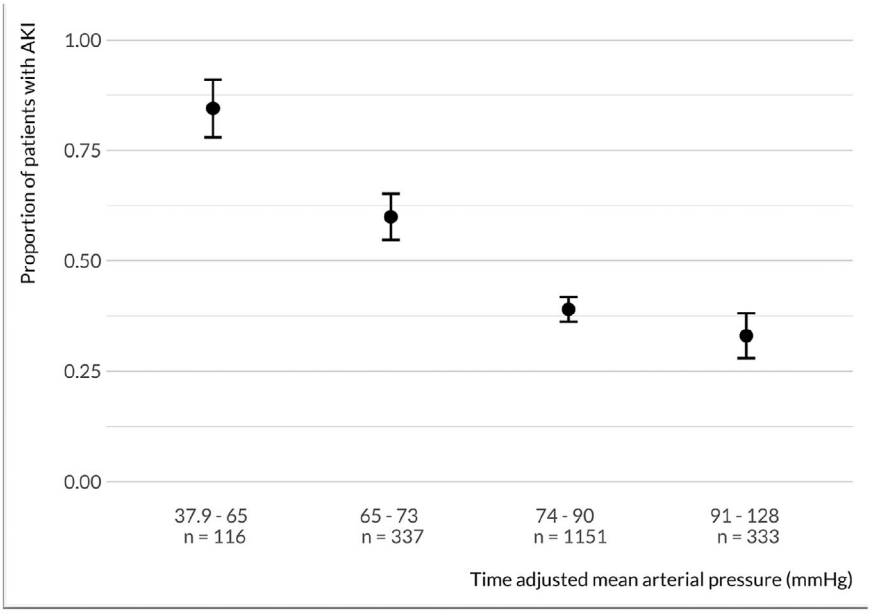

FIGURE 4 Proportions of patients with AKI related to four partitions of time-adjusted mean arterial pressure.

TABLE 4 The hazard ratio for a first AKI during ICU stay associated with measures of time-weighted average mean arterial pressure (TWA-MAP) <73 mmHg among 1499 patients without AKI at admission

Hypotension variables	Number of patients	Number of AKI cases	Crude HR	Adjusted[a] HR	95% CI
TWA-MAP <73, per unit	1499	421	1.09	1.09	1.06–1.13
TWA-MAP <73, quartiles					
< 0.52	375	93	1.00	1.00	(Reference)
0.52–1.52	375	94	0.83	0.74	0.55–0.98
1.53–3.13	375	95	0.78	0.72	0.53–0.96
3.13–25.73	374	139	1.52	1.41	1.07–1.84
Fraction[b] <73					
< 0.25	728	167	1.00	1.00	(Reference)
0.25–0.49	464	131	1.09	1.02	0.81–1.28
0.50–0.74	216	83	1.83	1.83	1.40–2.39
0.75–1.00	91	40	2.69	2.79	1.96–3.04

Abbreviations: AKI, acute kidney injury; CI, confidence interval; HR, hazard ratio; ICU, intensive care unit.
[a]Adjusted for age (years), weight (kg), diabetes (no, yes), cardiovascular disease (no, yes).
[b]Fraction of total time in ICU.

<25% of their ICU time with MAP <73 mmHg. The sensitivity analyses excluding patients who developed AKI during the first 24 h after admission gave largely similar results as the main analyses (Table S1). Further, the case-crossover analyses gave an OR of 2.87 (95% CI, 1.13–7.30) for AKI comparing TWA-MAP <73 mmHg in the highest versus the lowest quarter (Table S2). The analysis of TWA under MAP65 showed largely similar results as for TWA-MAP <73 (Table S3).

4 | DISCUSSION

In this study, we observed that 53.5% of unselected ICU patients developed AKI. AKI occurred early in the ICU stay, with only 10% of AKI having an onset later than 48 h after admission. About half of the patients with AKI had a mild renal failure (KDIGO stage 1). AKI was reversed during the ICU stay in 72.9% of patients, slower in the advanced KDIGO stages. Age-adjusted risk factors for AKI were found to be the presence of hypertension, diabetes, heart disease, chronic kidney disease, and higher body weight. AKI was associated with ICU LOS and mortality.

The incidence of AKI observed in this study is comparable to other observations. In the recent AKI-EPI study, the overall incidence of AKI assessed by KDIGO was 57%,[5] and in the FINNAKI study the corresponding number was found to be 39%.[6] These and our study share that also low-grade kidney injury is included. To include also lower stage AKI is relevant. Hoste et al found an odds ratio above two for mortality relative to non-AKI in the KDIGO group 1 patients,[5] and similar results have been shown also by others as well as in our study.[18,19] Whether these relationships indicate that AKI is a cause for increased mortality or that there is a shared cause for both AKI and increased mortality is unknown. Furthermore, it is not established if ICU patients with lower stage AKI is subject to disturbances in pharmacokinetics or coagulation disorders known to be influenced by renal failure. If so, this further underlines the relevance of identifying all patients with AKI.

In our study, we observed that AKI almost exclusively has an onset early in the ICU trajectory. Thus, the AKI is usually caused by the disease leading to ICU admission. This suggests that late-onset AKI should lead clinicians to consider a new clinical event, such as ventilator-associated pneumonia, catheter-related sepsis, or abdominal complications. This study also illustrates that AKI in the ICU usually is transient, most often within 1–2 days in the KDIGO stages 1 and 2, while considerably longer for KDIGO stage 3 cases, where about one-third of the patients go on to develop AKD. For clinical practice, this would signal that patients with a lower stage AKI could have a standard ICU follow-up, while patients having AKI KDIGO stage 3 should be followed more closely considering their kidney health.

Regarding risk factors for developing AKI, we observed a correlation between a lower blood pressure expressed by TAMAP and the development of AKI, and a correlation between the hypotensive dose, described as TWA-MAP<73, and the incidence of AKI, showing significantly more AKI in those having a higher dose of hypotension. Our results are comparable to a UK study, which found that a perfusion pressure of less than 60 mmHg was associated with a progression of AKI.[20]

In the multiple regression analysis, we found an increased hazard ratio in patients having hypertension and known heart disease, as well as in patients with diabetes. Also, in the patients having CKD there was an increase in the hazard ratio, but as there were few such patients, the significance is uncertain. Interestingly, the hazard ratio increased markedly in the highest sex-specific weight quarter. If this finding also applies to BMI could not be established, due to missing data on height. As expected, age is a risk factor for the development of AKI.

The strengths of this study are the number of patients, that all patients admitted to the unit were considered for inclusion and that most observations were obtained and stored electronically, which increases the resolution of data and minimizes the number of missing values. It is also a strength that relevant variables were obtained as a clinical routine in all patients. The use of an unselected group of ICU patients increases the relevance of the results in a clinical setting.

We recognize some limitations in this study. It is a retrospective, single-centre study, which by nature implies some limitations. The study does not incorporate the use of drugs known to induce renal failure as one of the risk factors. The department policy is generally to avoid the use of aminoglycosides, and due to the bacterial epidemiology in Norway, the use of vancomycin is infrequent. We also recognize that many of the patients were subject to examinations using x-ray contrast media. Finally, the data in the study were collected from 2010 to 2015. We believe that the results are still representative, as the treatment of AKI is mainly unchanged.

5 | CONCLUSION

In this study, we show that more than half of the patients in an unselected ICU population have AKI. Mortality is increased, the onset of AKI was early, and most cases of AKI were reversible. Risk factors for AKI were a time-weighted average of mean blood pressure below 73, pre-existing hypertension, chronic kidney disease, diabetes, heart disease, and higher body weight.

CONFLICTS OF INTEREST

None.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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