Epidemiology and Outcomes in Community-Acquired Versus Hospital-Acquired AKI

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Abstract

Background and objective Compared with AKI in hospitalized patients, little is known about patients sustaining AKI in the community and how this differs from AKI in hospital. This study compared epidemiology, risk factors, and short- and long-term outcomes for patients with community-acquired (CA) and hospital-acquired (HA) AKI.

Design, setting, participants, & measurements A total of 15,976 patients admitted to two district general hospitals between July 11, 2011, and January 15, 2012 were studied. Through use of an electronic database and the AKI Network classification, 686 patients with CA-AKI and 334 patients with HA-AKI were identified. Patients were followed up for 14 months, and data were collated on short-term and long-term renal and patient outcomes.

Results The incidence of CA-AKI among all hospital admissions was 4.3% compared with an incidence of 2.1% of HA-AKI, giving an overall AKI incidence of 6.4%. Patients with CA-AKI were younger than patients with HA-AKI. Risks for developing HA and CA-AKI were similar and included preexisting CKD, cardiac failure, ischemic heart disease, hypertension, diabetes, dementia, and cancer. Patients with CA-AKI were more likely to have stage 3 AKI and had shorter lengths of hospital stay than patients with HA-AKI. Those with CA-AKI had better (multivariate-adjusted) survival than patients with HA-AKI (hazard ratio, 1.8 [95% CI, 1.44–2.13; $P<0.001$] for HA-AKI group). Mortality for the CA-AKI group was 45%; 43.7% of these deaths were acute in-hospital deaths. Mortality for the HA-AKI group was 62.9%, with 68.1% of these deaths being acute in-hospital deaths. Renal referral rates were low across the cohorts (8.3%). Renal outcomes were similar in both CA-AKI and HA-AKI groups, with 39.4% and 33.6% of patients in both groups developing *de novo* CKD or progression of preexisting CKD within 14 months, respectively.

Conclusion Patients with CA-AKI sustain more severe AKI than patients with HA-AKI. Despite having risk factors similar to those of patients with HA-AKI, patients with CA AKI have better short- and long-term outcomes.

Clin J Am Soc Nephrol 9: 1007–1014, 2014. doi: 10.2215/CJN.07920713

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Introduction

AKI is a syndrome of rapid loss of kidney function and oliguria, which is associated with adverse patient outcomes (1–5). AKI is estimated to occur in up to 15% of hospitalized patients and up to 60% of critically ill patients (2,3,6). Despite advances in health care, the incidence of AKI is increasing and may be related to increasingly aggressive medical and surgical therapies in a largely aging population with multiple comorbid conditions (7). Although numerous studies have described the epidemiology, risk factors, and outcomes for patients developing AKI during hospitalization, less attention has focused on AKI that has developed in the community and how this may differ from hospital-acquired (HA) AKI. In this observational study, we compare epidemiology, risk factors, and outcomes of patients admitted to the hospital with community-acquired (CA) AKI in contrast to those who acquired AKI during their inpatient stay.

Materials and Methods

The study was conducted over 6 months in two district general hospitals in the United Kingdom. Data were collected from electronic records of all adult medical and surgical patients admitted to the two district general hospitals that form the Aneurin Bevan Health Board in Southeast Wales. These are Nevill Hall Hospital (500 beds, 8 critical care beds) and Royal Gwent Hospital (774 beds, 16 critical care beds), which combined serve an estimated population of 639,000. This trust has no dedicated onsite renal service and does not provide cardiothoracic, neurosurgery, plastic, or transplantation surgery. Patients requiring these interventions are transferred to the tertiary hospital in Cardiff. The catchment area of this trust includes one large port city with significant representation of ethnic minorities, as well as ex-industrial valley communities, with 38% of residents falling in the most socioeconomically deprived fifth of Wales (8).

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Two nephrologists reviewed electronic records of all admissions to these hospitals. All patients admitted between July 11, 2011, and January 15, 2012, were included in the analysis. Admissions through general practice, accident and emergency and hospital outpatient clinics were included. AKI was defined according to the AKI Network classification, using creatinine criteria for identification of AKI (9).

Any patients admitted to the hospital with AKI (CA-AKI) or developed AKI during the hospital stay (HA-AKI) were identified and included in the analysis. Patients admitted to hospital with AKI apparent on their first serum creatinine (sCr) measured within 48 hours of admission to hospital were denoted as having CA-AKI. Baseline sCr values for patients with CA-AKI were determined through review of all sCr values taken from the patient (from the hospital or the community) during the preceding 12 months. In contrast, patients were identified as having HA-AKI if no AKI was apparent on admission to hospital, but AKI developed during their hospital stay. Baseline sCr for patients with HA-AKI was taken as sCr on admission and was confirmed to be representative of true baseline by review of results from 12 months earlier. When no baseline sCr was available (49 patients), the percentage increase that defines AKI was calculated using the upper limit of normal laboratory reference range for sCr in men and women, respectively. Moreover, patients with unknown baseline values had sCr values charted after AKI resolution, which further enabled approximation of baseline sCr and confirmation of true AKI. This method of baseline sCr identification is recommended in the recent Kidney Disease Improving Global Outcomes AKI guidelines (10).

Patients with preexisting CKD that sustained acute-on-chronic kidney injury were included. CKD was identified from electronic clinical letters or blood tests indicating baseline eGFR<60 ml/min per 1.73 m^2 according to National Institute for Health and Clinical Excellence (NICE) CKD guidelines (11). Clinical data were collated using electronic patient records, including admitting specialty, comorbid conditions, length of hospital stay, intensive care unit (ICU) admission and length of ICU stay, in-hospital renal recovery, and in-hospital mortality. Recovery from AKI was defined as achievement of sCr no longer in keeping with the definition of AKI in comparison to baseline sCr values. Patients discharged with worsening renal function and no acknowledgment of AKI in their hospital discharge letter were defined as a discharge with unrecognized AKI. To be included in the analysis for long-term renal outcomes, patients had to have follow-up data available and be alive at 14 months after discharge. Development of *de novo* CKD was defined according to the CKD NICE guidelines (2008) as kidney damage or an eGFR<60 ml/min per 1.73 m^2 present on at least two occasions for ≥3 months. Progression of preexisting CKD was defined according to United Kingdom CKD guidelines and CKD-NICE guidelines as a decline in eGFR>15% or a decrease in eGFR>5 ml/min per 1.73 m^2 over 12 months (11,12). All patients were followed up for 14 months after discharge.

Statistical analysis was carried out using SPSS software, version 20 (SPSS, Inc., Chicago, IL). A *t* test and one-way ANOVA were used for analysis of normally distributed data. Categorical data were compared using a Pearson chi-squared test. Data that were not normally distributed were analyzed using a Mann–Whitney *U* test, Wilcoxon signed-rank test, and Kruskal–Wallis test. Continuous variables were described using mean±SD or median with interquartile range (IQR). Multivariate Cox proportional hazard modeling was used to determine the significance of comorbid conditions, age, ICU admission, length of hospital stay, AKI severity, and presence of HA-AKI or CA-AKI on survival. Binary regression analysis was used to assess overall mortality in both groups, adjusted for the aforementioned variables. Multivariate linear regression analysis was used to assess rehospitalization rates. Kaplan–Meier survival analysis was performed to assess 14-month mortality outcomes. *P* values<0.05 were considered to represent statistically significant differences.

Results

AKI Characteristics and Patient Demographic Characteristics

AKI Incidence. During the 6 months of the study, there were 15,976 adult admissions to the two district general hospitals. Of these, 1020 patients with AKI were identified, giving an overall AKI incidence of 6.4%. A total of 686 (67.3%) had developed AKI in the community and were admitted to hospital with AKI (CA-AKI). The overall incidence of CA-AKI was 4.3% of all hospital admissions. The remaining 334 (32.8%) AKI episodes were hospital acquired (HA-AKI); 86.2% of all CA-AKI and 85.3% of all HA-AKI were admitted under medical rather than surgical specialties.

AKI Characteristics. Twenty-five percent of AKI episodes were severe (AKI stage 3); most patients (45%) had stage 1 AKI and 30% stage 2. Demographic differences between patients with HA-AKI and CA-AKI are shown in Table 1. Those with CA-AKI were approximately 2 years younger than patients with HA-AKI (74.4 years versus 76.8 years; *P*=0.01). Patients with CA-AKI were more likely to have severe AKI (27% stage 3 in the CA-AKI group versus 20.7% in the HA-AKI group; *P*=0.03). Despite this, patients with CA-AKI were less likely to be admitted to the ICU during their hospital stay. Incidence in men and women among the CA-AKI and HA-AKI groups were similar. Preexisting CKD was seen in 31.9% of patients with AKI, with similar proportions across the CA-AKI and HA-AKI groups (33.2% versus 28.4%, respectively; *P*=0.12). Comparison of prevalence of various comorbid conditions in patients with CA-AKI and HA-AKI revealed approximately equal proportions of such diagnoses as diabetes; lung, liver, and heart disease; and dementia. The only significant difference was a higher prevalence of connective tissue disease in patients with HA-AKI.

In-Hospital Outcomes

Acute Mortality and Renal Outcomes. Short-term outcomes in patients with CA-AKI and HA-AKI are shown in Figure 1. Despite sustaining more severe AKI, more patients with CA-AKI showed recovery of renal function while an inpatient compared with the HA-AKI group (54.8% versus 45.5%; *P*=0.01). Patients with CA-AKI also

Table 1. Demographic data: community- versus hospital-acquired AKI

Variable	CA-AKI (*n*=686)	HA-AKI (*n*=334)	*P* Value
Mean age±SD (yr)	74.4±15.4	76.8±13	0.01
Men, % (*n*)	50.1 (342)	48.2 (161)	0.56
Preexisting CKD, % (*n*)	33.2 (228)	28.4 (95)	0.12
Mean baseline creatinine (preadmission) ±SD (μmol/L)[a]	112±50.8	107.6±33.5	0.68
Medical admission, % (*n*)	86.2 (591)	85.3 (285)	0.72
Surgical admission, % (*n*)	13.8 (95)	14.7 (49)	
ICU admission, % (*n*)	4.7 (32)	9.9 (33)	0.002
Median length of hospital stay (IQR) (d)	7 (3–16)	15 (7–26)	<0.001
AKI severity, % (*n*)			
Stage 1	42.4 (291)	51.8 (173)	0.01
Stage 2	30.6 (210)	27.5 (92)	0.34
Stage 3	27 (185)	20.7 (69)	0.03
Comorbid conditions, % (*n*)			
Congestive heart failure	15.4 (103)	19.8 (65)	0.72
Lung disease	15.9 (107)	21.3 (70)	0.34
Liver disease	3.4 (23)	4.0 (13)	0.66
Ischemic heart disease	20.4 (137)	26.5 (87)	0.28
Peripheral vascular disease	3.4 (23)	4.6 (15)	0.37
Cancer (within 5 yr)	14.5 (97)	12.2 (40)	0.34
Hypertension	31.8 (213)	31.4 (103)	0.95
Dementia	8.2 (55)	5.2 (17)	0.13
Diabetes	24.5 (165)	24 (79)	0.89
Cerebrovascular disease	10.7 (72)	14 (46)	0.95
Connective tissue disease	3.1 (21)	6.1 (20)	0.03

Continuous, normally distributed data analyzed using *t* test. Non-normally distributed variables analyzed using Mann–Whitney U test. Analysis of spread of categorical variables calculated using Pearson chi-squared test or Fisher exact test where appropriate. Sixteen patients from CA-AKI group and 8 patients from HA-AKI group had no comorbidity data available. CA-AKI, community-acquired AKI; HA-AKI, hospital-acquired AKI; ICU, intensive care unit; IQR, interquartile range.
[a]Baseline creatinine assessed as detailed in text. Forty-nine patients had no baseline creatinine available (41 in CA-AKI group and 8 in HA-AKI group).

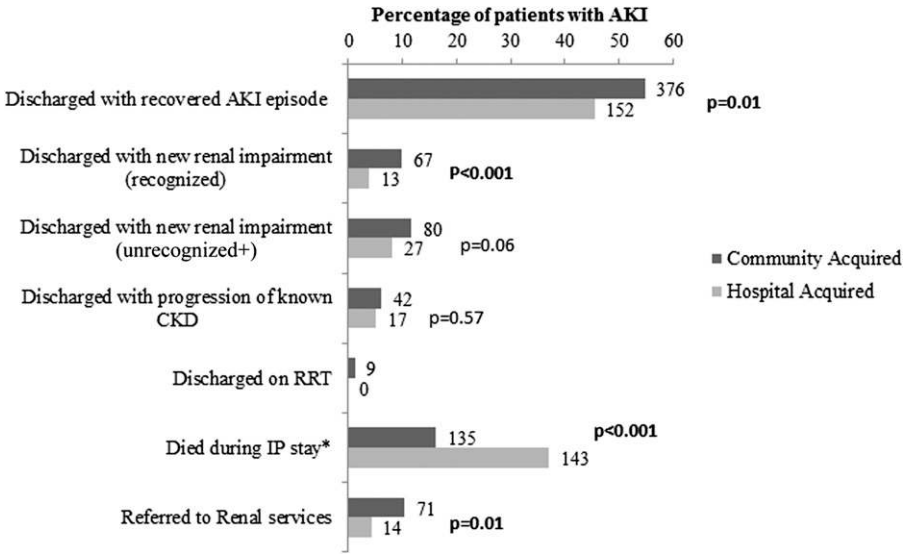

Figure 1. | In-hospital outcomes: community- versus hospital-acquired AKI. The graph demonstrates the percentage of AKI episodes that showed recovery/death/progression of CKD, that were unrecognized, or that occurred in patients discharged on RRT following AKI. The numbers at the top of the individual bars reflect the actual numbers of AKI episodes within this group. *P* values were calculated using Pearson chi-squared test/Fisher exact test where appropriate. [+]Unrecognized renal impairment, defined as a discharge from the hospital with worsening renal impairment without acknowledgment in discharge letter or follow-up planned. *Figures for inpatient (IP) deaths include those who died with AKI and those who had recovered from an AKI episode but died subsequently.

had lower inpatient mortality (19.6% versus 42.8% in HA-AKI group; $P<0.001$). They were also more likely to be labeled as having *de novo* CKD at the point of discharge compared with patients with HA-AKI (9.8% versus 3.9%; $P=0.001$). There were no significant differences between the numbers of patients requiring in-patient RRT (3.5% in the CA-AKI group and 1.8% in the HA-AKI group). Almost 11% ($n=107$) of patients were discharged with a rising creatinine and no acknowledgment of AKI in their hospital discharge letter. The percentages of patients with unrecognized AKI at discharge did not significantly differ between the CA-AKI and HA-AKI cohorts.

Length of Hospital Stay. Patients with CA-AKI had a shorter length of hospital stay, with a mean difference of 7.8 days (95% confidence intervals [95% CI], 5.1 to 10.6 days; $P<0.001$). Patients with CA-AKI had a median stay of 7 days (IQR, 3–16) compared with patients with HA-AKI (median, 15 days; IQR, 7–26 days).

Nephrology Referral. Only 85 of 1020 patients were referred to nephrology (8.3%), and only 82 (8.1%) had renal follow-up after discharge. Patients with CA-AKI were more likely to be referred (10.3% of the CA-AKI group versus 4.2% of the HA-AKI group; $P=0.001$).

Rehospitalization Rates

There were 492 rehospitalization events within 6 months after discharge. Rehospitalization rates did not differ between the CA-AKI and HA-AKI cohorts.

Long-Term Mortality Outcomes

Overall Mortality. All patients were followed up for 14 months after discharge. Within 14 months of the AKI episodes, 507 patients (49.7%) died. This comprised 287 (28.1%) in-hospital deaths and 220 (21.6%) deaths after hospital discharge. Patients with HA-AKI had greater mortality than those with CA-AKI (odds ratio, 2.26; 95% CI, 1.67 to 3.04; $P<0.001$, binary regression analysis). Figure 2 shows Kaplan–Meier survival statistics for both groups. At 14 months after discharge, 55.0% of those with CA-AKI and 36.9% with HA-AKI survived ($P<0.001$). Table 2 demonstrates the independent contribution of listed covariates on 14-month mortality. After adjustment for other significant independent predictors of death, the hazard ratio for mortality in the HA-AKI group versus the CA-AKI group was 1.75 (95% CI, 1.44 to 2.13; $P<0.001$).

Mortality According to AKI Severity. AKI stage 3 was an independent predictor for 14-month mortality, as shown in Table 2 (hazard ratio, 1.3; 95% CI, 1.01 to 1.55; $P=0.04$). However, a significant proportion of patients with stage 1 CA-AKI and HA-AKI had heightened mortality. In particular, >50% of patients with stage 1 HA-AKI died within 14 months (Figure 3).

Long-Term Renal Outcomes

A total of 383 (56%) patients with CA-AKI and 128 (38%) patients with HA-AKI were alive at 14 months with sufficient data available for analysis. Mean sCr at discharge was significantly higher in patients with CA-AKI (mean±SD, 180.4±140.1 μmol/L) than in those with HA-AKI (134.7±51.9 μmol/L) ($P=0.04$). At 14 months after discharge, 39.4% of the CA-AKI group and 33.6% of the HA-AKI group had *de novo* CKD or progression of preexisting CKD. However, there were no significant differences in percentage of patients with *de novo* CKD or with progression of preexisting known CKD at 14 months after discharge. Furthermore, the mean change in eGFR did not significantly differ between patients with CA-AKI and those with HA-AKI (Table 3).

Discussion

Incidence and associated mortality risks of AKI in critically ill patients are well documented (2,3,13,14). Increases in creatinine in non–critically ill hospitalized patients are also common and carry heightened mortality (1,5,15–19). This has been attributed to the older age and increased number of comorbid conditions present in hospitalized patients with AKI. In contrast, studies describing incidence, risk factors, and outcomes of patients who sustain AKI in the community are limited, and the few studies performed have restricted patient numbers. In this study, we identified 686 patients who sustained AKI in the community and were subsequently admitted to the hospital. We compared this cohort with 334 patients who sustained AKI during a hospital stay. The incidence of CA-AKI was higher in the present study than in other studies (9,20–23). However, this probably remains an underestimate of the true amount of CA-AKI that exists because numerous patients with CA-AKI will neither have blood tests performed nor be admitted to the hospital. This ascertainment bias has been described in other CA-AKI studies (24).

This study highlights that risk factors for CA-AKI and HA-AKI are similar, with CA-AKI also being more common in male elderly patients with preexisting CKD, diabetes, heart disease, hypertension, dementia, and cancer. This highlights the demographic characteristics of people in the community who may benefit from more frequent blood tests in the event of an acute illness or medication change.

Even mild AKI is no longer considered to be benign, but rather an independent predictor of mortality (18,19,25). Surprisingly, our data demonstrated that more severe AKI was not necessarily associated with worse outcomes. Patients with CA-AKI had more severe AKI, shorter hospitalizations, yet better long-term survival than patients with HA-AKI. In fact, 14-month mortality was >30% in patients with stage 1 CA-AKI and >50% in patients with stage 1 HA-AKI. Superior survival in CA-AKI was surprising because these patients had comorbid conditions similar to those of patients with HA-AKI. The reasons for these differences remain unclear. Interestingly, a study performed in Scotland demonstrated that inadequacies in recognition and management of hospital-based AKI were particularly high in patients with mild AKI (23). Thus, more severe CA-AKI may have been more appropriately managed, leading to better outcomes. Perhaps further contributing to differences in management of CA-AKI and HA-AKI is that on admission to the hospital in the United Kingdom, patients are generally assessed by senior medical/surgical consultants within 6 to 24 hours. However, after this, senior medical review occurs only two to three times per week, with most patient care otherwise undertaken by junior staff. This may also underlie

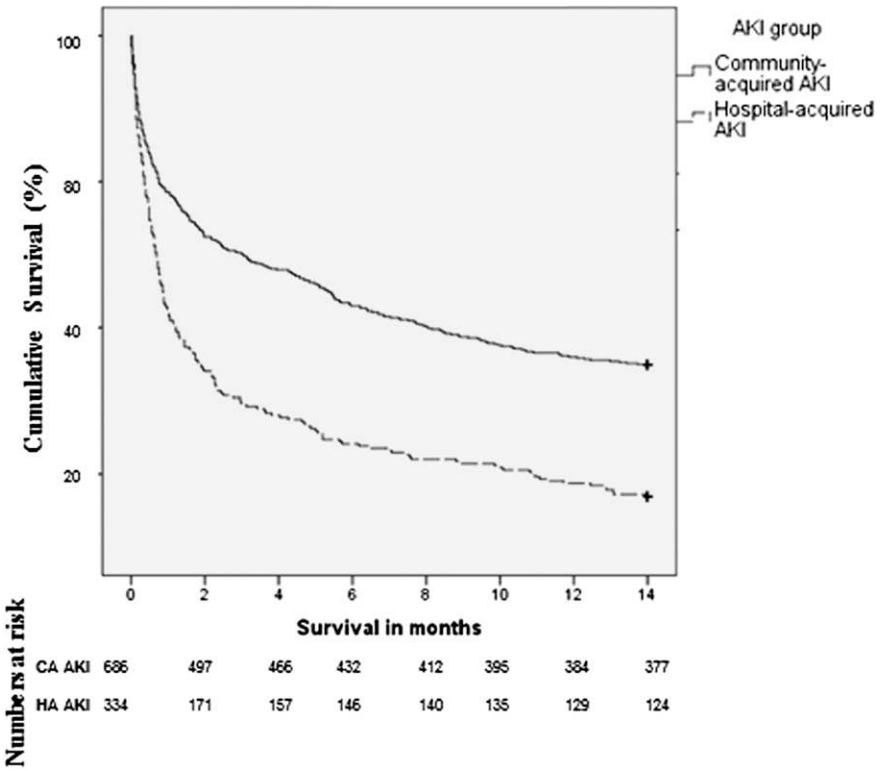

Figure 2. | Kaplan–Meier survival statistics comparing community-acquired AKI (CA-AKI) and hospital-acquired AKI (HA-AKI) groups. $P<0.001$ for all comparisons. Numbers of patients at risk at each time point shown below the graph.

Table 2. Cox regression survival analysis for all-cause mortality at 14 months

Variable	Hazard Ratio (95% CI)	*P* Value
Congestive heart failure	0.80 (0.63 to 1.01)	0.06
Lung disease	0.81 (0.64 to 1.03)	0.84
Liver disease	1.08 (0.70 to 1.67)	0.72
Ischemic heart disease	1.12 (0.95 to 1.50)	0.13
Peripheral vascular disease	0.87 (0.53 to 1.42)	0.57
Cancer (within 5 yr)	0.97 (0.77 to 1.24)	0.82
Hypertension	0.80 (0.65 to 0.99)	0.04
Dementia	1.07 (0.76 to 1.52)	0.67
Diabetes	0.85 (0.67 to 1.07)	0.16
Cerebrovascular disease	0.99 (0.74 to 1.32)	0.93
Connective tissue disease	0.69 (0.42 to 1.12)	0.13
Age	1.00 (1.00 to 1.01)	0.32
Preexisting CKD	0.92 (0.76 to 1.13)	0.42
Admission to ICU	1.68 (1.18 to 2.39)	0.01
Length of stay	0.98 (0.98 to 0.99)	<0.001
AKI stage 3 compared with AKI stages 1 and 2	1.25 (1.01 to 1.55)	0.04
HA-AKI compared with CA-AKI	1.75 (1.44 to 2.13)	<0.001

Cox regression analysis was used to determine the influence of age (hazard per increasing year), length of stay (hazard per inpatient day), AKI severity (stage 3 compared with stages 1 and 2), and comorbidity on risk of death within 14 months after discharge. Listed comorbid conditions obtained from electronic records coding and defined as binary variables (present or absent). The hazard ratio of mortality in HA-AKI versus CA-AKI is shown at the bottom of the table (hazard ratio, 1.75; 95% CI, 1.44 to 2.13). 95% CI, 95% confidence interval; ICU, intensive care unit; HA-AKI, hospital-acquired AKI; CA-AKI, community-acquired AKI.

differences in early appropriate recognition and management of CA-AKI and HA-AKI, which may ultimately influence differences in outcomes. Although we could identify patients with unrecognized AKI at the point of discharge from hospital, we were unable to assess whether AKI was missed or mismanaged during inpatient stay. It

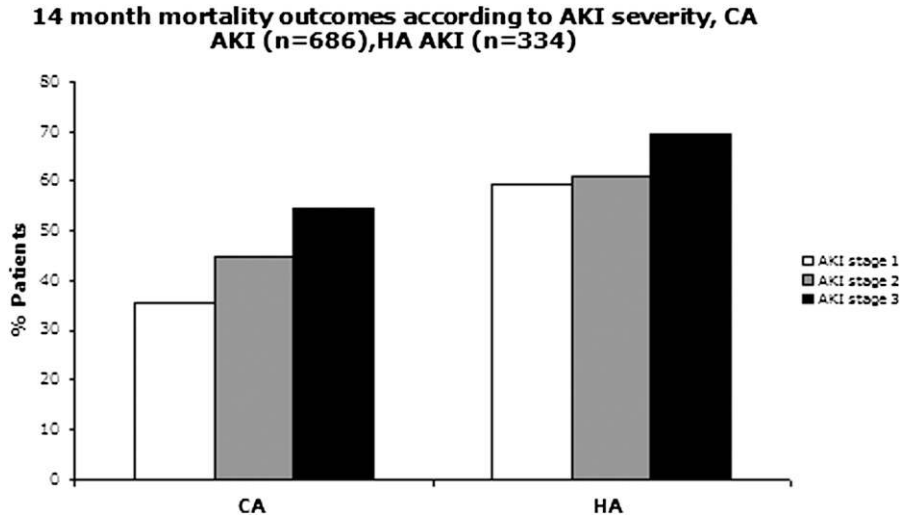

Figure 3. | **Long-term mortality outcomes according to AKI stage.** The graph demonstrates percentages of patients in each AKI stage who died within 14 months. *P*=0.001 for comparisons between death in AKI stage 1 (Pearson chi-squared test). AKI stages 2 and 3 mortality comparisons between community-acquired (CA) AKI and hospital-acquired (HA) AKI groups not significant.

Table 3. Long-term renal outcomes

Patients with AKI Alive at 14 mo after Discharge	CA-AKI (*n*=686)	HA-AKI (*n*=334)	*P* Value
Alive at 14 mo, *n* (%)	383 (56.0)	128 (38.0)	<0.001[a]
CKD at 14 mo, *n* (%)	151 (39.4)	43 (33.6)	0.24[a]
De novo CKD, *n*	94 (62.3)	26 (60.5)	0.86[b]
Progression of CKD, *n*	57 (37.7)	17 (39.5)	0.86[b]
CKD stage at 14 mo, *n* (%)			
CKD 2	4 (2.6)	0 (0)	
CKD 3	86 (57)	28 (65.1)	
CKD 4	44 (29.1)	13 (30.2)	
CKD 5	17 (11.3)	2 (4.7)	0.26[b]
RRT required at 14 mo, *n*	6 (1.6)	2 (4.7)	1.0[b]
Baseline preadmission Cr (μmol/L)	111.2±50.8	107.6±33.5	0.68[c]
Baseline preadmission eGFR (ml/min per 1.73 m^2)	64.5±23.3	63.7±24.4	0.83[c]
Cr on discharge (μmol/L)	180.4±140.1	134.7±51.9	0.04[c]
14-mo eGFR (ml/min per 1.73 m^2)	34.5±14.3	34.9±11.5	0.87[c]
Change in eGFR over 14 mo (ml/min per 1.73 m^2)	−30	−28.8	0.72[d]

Patients who remained alive 14 months following the AKI episode were analyzed for development of *de novo* CKD or progression of preexisting CKD, according to whether they had CA-AKI or HA-AKI. Values expressed with a plus/minus sign are the mean±SD. CA-AKI, community-acquired AKI; HA-AKI, hospital-acquired AKI; Cr, creatinine.
[a]*P* value calculated using Pearson chi-squared test.
[b]*P* value calculated using Fisher exact test.
[c]*P* value calculated using ANOVA.
[d]*P* value calculated using *t* test.

is, however, evident that patients with CA-AKI were more likely to be referred to nephrology, thus further suggesting that they were more likely to be recognized and appropriately managed. Important differences in the precipitants/etiology of AKI may also be a factor in dictating short- and long-term outcomes with previous work that suggested vasculitis, GN, and obstructive uropathy may be more prevalent in CA-AKI, while prerenal failure and acute tubular necrosis are more common in patients with HA-AKI (26). These data could not be reliably obtained in our analysis, thus highlighting an important limitation. However, it is clear that the incidence and mortality of CA-AKI are significant and clinically underappreciated.

A literature review identified only five previous studies on CA-AKI. All of these were performed in patients with CA-AKI admitted to the hospital. Two of these studies, based in Brazil and China, had less than a third of the patient numbers presented here (27,28). The causes of AKI, as well as the prevalence of various comorbid conditions in these studies, probably differ from those present in our

patients; hence, the results may not directly apply to Western populations. Similarly, a study performed in African Americans in 2000 identified only 100 patients with CA-AKI; these findings may not apply to our population because of the increased prevalence of diabetes and ESRD in this specific group (29). The remaining two studies performed in 2013 in Scotland and Louisville, Kentucky, although having fewer patient numbers, demonstrated results similar to those of this study (22,23). A recent meta-analysis of world AKI incidence found an inverse correlation of AKI-associated mortality and percentage of country gross domestic product spent on health expenditure. This represents an additional important factor in interpreting the applicability of the currently available literature (24).

AKI is an important contributor to CKD. Previous studies have highlighted increased risks of *de novo* CKD following episodes of AKI with incomplete recovery (3,15,16,22,25). In this study, one third of patients developed *de novo* CKD or progression of preexisting CKD. Although the peak creatinine at the time of discharge was significantly higher in patients with CA-AKI, the percentages of patients who developed CKD did not significantly differ between the CA-AKI and HA-AKI groups. Over 60% of patients with AKI developed *de novo* CKD. Moreover, half of patients with CA-AKI and >70% of those with HA-AKI who developed CKD had initially sustained AKI stage 1. These rates are higher than those reported in other large AKI population-based studies (30). Possible explanations for this include a more elderly cohort with greater comorbidity in a relatively low-socioeconomic-status area of the United Kingdom. Nevertheless, these findings highlight that even mild AKI strongly affects long-term renal outcomes. Again, the reasons are not clear but may be attributed to reduced recognition and poor management of less severe AKI, as previously described (23). A potential source of bias in our findings may arise from the competing hazard of death in this AKI cohort, a statistical fallacy not accounted for by Kaplan–Meier survival analysis. Of concern, only 8.3% of patients with AKI were referred to nephrology; thus, it is likely that the majority were not assessed for risks of developing CKD and did not have appropriate nephrology follow-up. Thus, hospital teams need to appreciate the long-term risks of CKD, even in mild AKI, and nephrologists need to help implement strategies that lead to appropriate referral in order to prevent CKD progression.

Future work in this area needs to determine whether early recognition and intervention affects the long-term outcomes of patients with AKI. Furthermore, levels of nephrologist involvement in early AKI management need to be evaluated, and educational and training tools for hospital teams involved in AKI management implemented. Moreover, studies will need to determine the importance of subsequent follow-up of these patients in terms of improving renal and patient outcomes.

Our study has limitations. There was no information on cause of AKI. It would be useful to identify whether certain causes are more likely to be associated with poorer long-term survival. The causes of death in our cohort could not be reliably obtained. Information was not obtained on differences in patient management that may have influenced ultimate outcomes. Furthermore, generalizability of the study findings may be hampered by the absence of cardiothoracic and transplantation surgery at the district general hospitals studied, possibly contributing to the lower incidence of HA-AKI in comparison with that reported in other studies (1–3,6). Despite these limitations, this is the largest study to date comparing CA-AKI and HA-AKI, which provides a better understanding of the epidemiology, risk factors, disease course, and outcomes of CA-AKI.

In conclusion, patients with HA-AKI are marginally older than those with CA-AKI but otherwise demonstrate similar demographic and risk factors. Despite this, patients with CA-AKI have better short- and long-term outcomes, the reasons for which are unknown. It is also clear that the independent risks of death and of developing CKD are high, even in minor AKI, and preventive and management strategies for this condition need to be identified, implemented, and evaluated to limit the risks to susceptible individuals.

Disclosures

None.

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Received: July 26, 2013 **Accepted:** January 28, 2014

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Published online ahead of print. Publication date available at www.cjasn.org.