Acute kidney injury: outcomes and quality of care

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Summary

Background: Deficiencies in management have been highlighted as contributory factors in the death of many patients with acute kidney injury (AKI). However, there is little evidence addressing the quality of care provided to patients with milder AKI.

Aim: The aim of this study is to evaluate the quality of care provided to a non-select cohort of patients with AKI and evaluate discrepancies in causation, recognition and management.

Design: Retrospective inception cohort study.

Methods: Demographic data were collected for all 1577 patients admitted to a University Teaching Hospital during a 1-month period. Baseline, admission and peak creatinine were correlated with mortality and length of hospital admission. AKI was classified according to Kidney Disease Improving Global Outcomes criteria. A retrospective case note review of all patients with AKI was carried out to evaluate quality of documentation and clinical management of AKI. Multivariate analysis was undertaken to determine risk factors for AKI.

Results: Incidence of AKI on admission was 4.6%. A further 10.3% developed AKI while in hospital. All cause mortality was 4-fold higher among patients with AKI compared with those without (19 vs. 3.8%; P<0.001). Mortality was significantly higher in those patients who developed AKI while an in-patient compared with those with AKI on admission (27.3 vs. 11.8%; P<0.001). Diabetes, clinician perception of frailty, age and treatment with angiotensin-converting enzyme inhibitor prior to admission were found to be independent risk factors for AKI. AKI was unrecognized in 23.5% of patients, two-thirds of whom were discharged without resolution of renal function. Significant weaknesses in management were poorly kept fluid balance charts (48.2%), failure to withhold nephrotoxic drugs (38.8%) and failure to act upon abnormal biochemistry (41%) in a timely fashion.

Conclusions: AKI is common in hospitalized patients and associated with a significant increase in hospital stay and mortality. AKI is often found in conjunction with other organ failure and in many cases is not preventable. Nevertheless clinicians need to be more vigilant of small creatinine rises to permit early intervention particularly among elderly and frail patients.

Introduction

The incidence of severe acute kidney injury (AKI) requiring renal replacement therapy (RRT) is well documented in the literature. UK data indicate an increase in incidence from 172 per million population (pmp) per year 20 years ago1 to 630 pmp per year in more recent series.2 Many of the population studies are limited by lack of a standardized definition and underreporting of mild AKI. The recent
Kidney Disease Improving Global Outcomes (KDIGO) consensus conference on AKI\(^3\) has attempted to harmonize existing Acute Dialysis Quality Initiative RIFLE (Risk, Injury, Failure, Loss, ESRD) criteria\(^4\) and AKI Network (AKIN)\(^5\) staging systems into a common classification system. It is hoped that this will provide clarity and homogeneity of reporting in future studies.

Severe AKI is well recognized as an independent risk factor for mortality.\(^6\),\(^7\) However, it is less clearly understood whether mild, self-limiting AKI bears any relation to the severe renal dysfunction that is associated with morbidity and mortality. Current education and training strategies focus on the importance of early recognition and management in AKI. A better understanding of the disease course, risk factors and response to interventions would allow optimal focus of resources.

There have been few studies of quality of care in patients who develop AKI. The recent UK National Confidential Enquiry into Patient Outcomes and Death (NCEPOD)\(^8\) highlighted potential deficiencies in the care of \(\sim\)50\% of patients with AKI who died in the peri-operative period. No studies have specifically addressed the quality of care in patients who develop mild AKI in isolation.

The primary aim of this study is therefore to evaluate the quality of care provided to a non-select cohort of patients with AKI and evaluate discrepancies in causation, recognition and management. The processes leading to the development of in-hospital AKI will be examined and the impact of AKI on morbidity and mortality was outlined.

**Materials and methods**

**Study population**

The Western Infirmary is a 650-bedded university teaching hospital in Glasgow which, in addition to admitting for general medicine and surgery, is a tertiary referral centre for renal, vascular surgery and urology. Over 95\% of the hospital’s workload is unscheduled. All patients admitted during a 1-month period (1 April 2011–1 May 2011) had baseline demographics (age, sex, admitting speciality, diagnosis) recorded and were screened for biochemical evidence of AKI using electronic laboratory reporting systems. AKI was classified according to the KDIGO criteria\(^3\) (Figure 1) as recommended by the UK Renal Association\(^9\) as the significance of small changes in creatinine is recognized. Patients with established renal failure on long-term dialysis \((n=112)\) and patients referred from another in-patient facility directly to our renal unit \((n=82)\) were excluded.

A retrospective case note review was undertaken for all patients with AKI and for a cohort of age- and sex-matched controls \((n=245)\) who did not develop AKI, by four experienced clinicians with an interest in AKI (E.A., L.G., L.K. and D.K.). Additional information was collected regarding the co-morbidities, medicine therapy, time of admission, waiting time to first clinician assessment after presentation to the Emergency Department and level of experience of admitting doctor. A presumed ‘cause’ for the AKI was determined in accordance with beginning and ending supportive therapy (B.E.S.T.) kidney criteria\(^10\) and potential contributing factors were examined \{hypotension, hypovolaemia, intravenous contrast agents, nephrotoxic drugs \[primarily non-steroidal anti-inflammatory drug and angiotensin-converting enzyme (ACE) inhibitors\], sepsis\}. An evaluation of the quality of care was undertaken with delays in recognition or management of AKI and adequacy of intervention/specialist referral appraised by the reviewers based on medical and nursing documentation in case notes, observation charts and fluid balance charts. Outcomes, including mortality, length of hospital stay and need for RRT, were also recorded. All case notes and electronic records were viewed by two independent reviewers and cases of discrepancy were resolved by a third reviewer.

Data were recorded anonymously into a database created in Microsoft Excel. A sample of 50 patients was independently double entered for validation and cross-reference purposes.

**Definitions**

AKI was classified according to KDIGO\(^3\) criteria (Figure 1). If available, urine output was included for classification of AKI. Urine output data were

<table>
<thead>
<tr>
<th>Stage</th>
<th>Serum creatinine (Scr) criteria</th>
<th>Urine output criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Increase ≥26μmol/l within 48 hours OR Increase ≥ 1.5 to 1.9 x reference Scr</td>
<td>&lt;0.5ml/kg/hour for &gt;6 consecutive hours</td>
</tr>
<tr>
<td>2</td>
<td>Increase ≥ 2 to 2.9 x reference Scr</td>
<td>&lt;0.5ml/kg/hour for &gt;12 hours</td>
</tr>
<tr>
<td>3</td>
<td>Increase ≥3 x reference Scr OR increase ≥354μmol/l OR commenced on renal replacement therapy (RRT) irrespective of the stage</td>
<td>&lt;0.3ml/kg/hr for &gt;24 hours or anuria for 12 hours</td>
</tr>
</tbody>
</table>

Figure 1. KDIGO classification of AKI.
only available in 54.7% of patients. Both admission and maximum KDIGO scores based on either serum creatinine or urine output criteria (where available) were recorded.

A baseline serum creatinine level, from the 3 months prior to hospital admission, was available for 1293 (88%) of patients. When baseline creatinine was unknown, it was estimated by the Modification of Diet in Renal Disease (MDRD) equation, assuming a glomerular filtration ratio of 75 ml/min/1.73 m², as recently validated in AKI by Bagshaw et al. Hypotension was defined as a systolic blood pressure <90 mmHg. Frailty was considered to be ‘clinician perception of frailty’, i.e. the admitting clinician documented in the case notes a statement indicating that they believed the patient to be frail.

Statistical analysis

All data were analysed with statistical software SPSS for Windows, version 16 (SPSS Inc., Chicago, IL, USA). Descriptive statistics for demographics and clinical variables are presented as the mean (interquartile range) unless otherwise stated. Continuous variables with normal distribution were compared using a Student’s t-test, while categorical variables and continuous variables with skewed distribution were assessed by chi-squared and Mann–Whitney U-tests, respectively. Multivariate analysis was undertaken using the Cox proportional hazards model with factors found to be significant on univariate analysis (P<0.05) and deemed to be clinically relevant included as potential co-variates. A Kaplan–Meier method was used for comparison of survival between the no-AKI and AKI groups. A P-value < 0.05 was regarded as statistically significant.

Ethics approval

Approval for this retrospective review of practice was obtained from the local Clinical Effectiveness Department.

Results

A total of 1577 patients were admitted over the 1-month period. In all, 243 patients (15.5%) had AKI at some point during their hospital admission. A total of 73 patients (4.6%) had AKI at the time of admission (community acquired). A further 170 patients (10.7%) developed AKI during their hospital admission (hospital acquired).

The mean length of time from admission to maximum serum creatinine was 3.04 ± 0.243 (range: 0–19) days and mean time taken from maximum serum creatinine to resolution (to within 10% of baseline) was 3.19 ± 0.25 (range: 2–20) days. Figure 2 illustrates the progressive evolution of AKI.

A total of 143 patients (9%) had a maximum AKI Stage 1; 57 patients had AKI Stage 2 (3.6%) and 43 patients (2.7%) had AKI Stage 3.

Estimated baseline creatinine values

Baseline creatinine was estimated in 284 patients (18%) using the method described earlier; 96% of them saw a return to within 20% of estimated baseline. Within the group where baseline creatinine was estimated, 244 (85.9%) of patients had no AKI, 37 (13%) had maximum AKI Stage 1 and 3 patients (1.1%) had AKI Stage 2.

Outcomes

In hospital, mortality was over 4-fold higher in patients with AKI compared with those without (19 vs. 3.8%, P<0.001, Figure 3a). Similarly, length of hospital stay was twice as long for patients with AKI compared with those without (11.5 ± 1.6 vs. 4.9 ± 1.2 days, P<0.001). Interestingly, patients with hospital-acquired AKI had double the mortality of community-acquired AKI (27.3 vs. 11.8%, P<0.001) and in patients with ‘early AKI’ (within 7 days of admission) compared with ‘late AKI’ (≥7 days of admission) (Figure 3b). Mortality was higher in patients with more severe AKI (AKI 1: 6.1%; AKI 2: 22.6%; AKI 3: 35.9%) (Figure 3c).

Six patients (2.5%) required RRT (three temporarily, three permanent). A decision to limit treatment by not dialysing or filtering was made in a further three (1.2%) patients; 16.9% of patients with AKI developed multi-organ failure (more than two systems), of whom only 61% were managed within a critical care area [Level 2 (high dependency) or 3 (intensive care unit) care]. A treatment limiting/palliation decision was made in a further 36% of cases. Two patients (0.8%) had multi-organ failure which went unrecognized for >24 h; 2.2% of patients with recognized multi-organ failure who may have benefited from Level 2 or 3 care were managed with a Level 1 (ward level care) environment due to bed shortages within critical care facilities.

Data on medium-term renal function were obtained for 99.8% of patients with AKI (8–12 weeks post-discharge). A total of 118 patients (61.1%) had normal renal function; 27.1% had CKD 3; 2.5% had CKD 4 and 1.6% had CKD 5.

A total of 61 patients were discharged prior to resolution of renal function to within 10% of baseline. The AKI was either unrecognized or no plan for follow-up monitoring was made in 54 (88.5%) of these patients. A total of 25 patients (10.3%) with
unrecognized AKI had an estimated glomerular filtration rate (eGFR) >60. Repeat serum biochemistry within 12 weeks of discharge was obtained for all, but three patients discharged with biochemical evidence of AKI. Five patients (8.6%) with unrecognized AKI had new CKD 3 8–12 weeks following discharge. A total of 38 patients (19.2%) of all patients with AKI had new CKD 3 (Table 1).

Risk factors for AKI
In this study, 74.1% of patients with AKI were admitted to a medical speciality. Figure 4 illustrates the proportion of patients with AKI by admitting speciality. Table 2 outlines univariate analysis comparing demographic details of patients with and without AKI. Additionally, details regarding presentation and initial management are compared between patients with AKI and the age- and sex-matched control cohort. Patients in the AKI group were older (76.2 ± 4 vs. 56.7 ± 4.3 years, \(P < 0.001\)) and had more co-morbidities. The mean wait for first assessment in the emergency department for patients with AKI was 3.14 ± 0.165 h compared with 3.07 ± 0.25 h in the sample of age-/sex-matched patients who did not develop AKI \(P = 0.83\). In our study, 16.3% of patients with AKI and 13.9% of patients without AKI waited >4 h for first assessment by a doctor \(P = 0.78\); 45.7% of patients with AKI were admitted out with normal working hours and 32.1% were first seen by a doctor with <2 years experience. Ninety-six per cent of the patients were seen by a consultant within 24 h of admission. There was no significant difference in any of these factors between patients who developed AKI and those who did not (Table 2).

Multivariate analysis (Table 3) was undertaken to include all patients with a highly significant difference on univariate analysis \(P < 0.001\). This demonstrated diabetes mellitus, clinician perception of ‘frailty’, age, hypovolaemia on admission and treatment with ACE inhibitor prior to admission to be independent risk factors of AKI.

Causation and precipitating factors for AKI
The principal factor associated with AKI was considered according to BEST Kidney criteria: \(^{10}\) 6.6% post-op; 39.9% sepsis; 27.2% cardiogenic and 18.9% hypovolaemia. Moreover, 7.4% of patients had inadequate documentation to inform a decision; 3.2% of patients had an obstructed urinary system. This was associated with sepsis in every case of AKI. Eighty-four per cent of AKI was deemed to be predictable by the clinicians reviewing the case notes, 15.2% avoidable and 22.7% iatrogenic.
Additionally, potential precipitants of renal dysfunction were identified: contrast (10.7%), drugs (18.5%) (primarily gentamycin and frusemide), hypotension (30.5%) and infection (44.9%).

A total of 16 (6.6%) patients with AKI developed it in the post-operative period. Three-quarters of these patients had emergency surgery. Hypotension in the peri-operative period was deemed to be contributive to the AKI in 13 (81.2%) of patients, with surgical complications affecting 31.2%, infection 18.7% and inadequacies in post-operative management in 18.7%. Two patients suffered peri-operative acute coronary syndromes. Notably, there was no evidence of inadequate pre-operative management or timing of the surgery for these patients.

**Recognition and management of AKI**

A delay in the recognition of AKI >12 h from the time of biochemical derangement occurred in 18.5% of patients (range: 12–96 h), with a further 23.5% of patients having completely unrecognized...
Table 2: Univariate analysis of risk factors for AKI

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>AKI</th>
<th>No AKI</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>76.2 ± 4</td>
<td>59.6 ± 2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Sex (male)</td>
<td>123 (50.6%)</td>
<td>806 (51%)</td>
<td>NS</td>
</tr>
<tr>
<td>Emergency admission</td>
<td>238 (97.9%)</td>
<td>1282 (81.3%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Boarder (i.e. admitted to a ward dedicated to a different specialty)</td>
<td>8 (7.4%)</td>
<td>80 (6%)</td>
<td>0.08</td>
</tr>
<tr>
<td>Admission to correct speciality</td>
<td>222 (91.4%)</td>
<td>1467 (9.3%)</td>
<td>NS</td>
</tr>
<tr>
<td>Mean wait for first assessment (h)</td>
<td>3.14 ± 0.16</td>
<td>3.07 ± 0.25</td>
<td>NS</td>
</tr>
<tr>
<td>Percentage waiting for first assessment &gt;4 h</td>
<td>16.3%</td>
<td>13.9%</td>
<td>NS</td>
</tr>
<tr>
<td>Presentation out with normal working hours (9 am–5 pm)</td>
<td>45.7%</td>
<td>47.2%</td>
<td>NS</td>
</tr>
<tr>
<td>Percentage seen by admitting doctors</td>
<td>32.1%</td>
<td>34.9%</td>
<td>NS</td>
</tr>
<tr>
<td>Consultant review within 24 h</td>
<td>96%</td>
<td>98%</td>
<td>NS</td>
</tr>
<tr>
<td>Co-morbidities</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CKD</td>
<td>57 (23.5%)</td>
<td>220 (13.9%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hypertension</td>
<td>108 (44.4%)</td>
<td>598 (37.9%)</td>
<td>0.01</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>111 (45.7%)</td>
<td>636 (40.3%)</td>
<td>0.01</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>45 (18.5%)</td>
<td>95 (6%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Peripheral vascular disease</td>
<td>33 (13.6%)</td>
<td>156 (9.8%)</td>
<td>0.01</td>
</tr>
<tr>
<td>COPD</td>
<td>33 (13.6%)</td>
<td>167 (10.5%)</td>
<td>NS</td>
</tr>
<tr>
<td>Frailty</td>
<td>101 (41.6%)</td>
<td>147 (11%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ACE I</td>
<td>84 (34.6%)</td>
<td>240 (15.2%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>NSAID</td>
<td>9 (3.7%)</td>
<td>36 (2.2%)</td>
<td>NS</td>
</tr>
<tr>
<td>Diuretic</td>
<td>120 (49.4%)</td>
<td>448 (28.2%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Contributing factors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypovolaemia</td>
<td>75 (30.9%)</td>
<td>196 (12.4%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Hypotension</td>
<td>56 (23%)</td>
<td>142 (9%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Infection</td>
<td>106 (43.6%)</td>
<td>258 (16.4%)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Data are presented as number of patients (percentage of total). ‘Frailty’ was defined as documented evidence of frailty as perceived by the admitting clinician. NSAID, non-steroidal anti-inflammatory drug; COPD, chronic obstructive pulmonary disease.
AKI. In the majority of cases, this unrecognized AKI was mild (Stage 1), transient and self-resolving. Nearly two-thirds of the patients with unrecognized AKI were discharged within 24 h of initial presentation. The mean length of time taken before documentation of admission biochemistry was 3.82 ± 0.17 h (range: 1–15).

Of the 102 patients with delayed or unrecognized AKI, a clear cause of the delay was established in 48 patients (47%): clinician inexperience (10.7%); inadequate clinical review (21.4%); inadequate observations (7.1%); inadequate investigations (14.3%) and infrequent clinical review (3.6%) were deemed contributing factors.

Factors deemed to be important by the NCEPOD report in prevention and management of AKI were evaluated to determine the quality of care provided to patients with AKI. According to documentation within the case notes, significant deficiencies in management were failure to catheterize to monitor hourly urine volumes, poorly completed fluid balance charts, failure to withhold nephrotoxic drugs and failure to notice or act upon abnormal biochemistry in a timely fashion. Only half (51.8%) of patients had adequate documentation of fluid balance; 43.4% of the remainder had a fluid balance chart in their case notes which was inaccurate and 86.4% had an accurately completed observations temperature, pulse, respirations (TPR) chart at least 12 hourly. Routine observations (temperature, pulse, blood pressure) were taken less frequently than once per day in 3.7% of patients with AKI. 33.7% of patients with AKI (and 85.3% of patients with AKI Stage 2 or 3) were catheterized for measurement of hourly urine volumes. A renal ultrasound was deemed appropriate to investigate the cause of AKI in one-quarter of patients (24.3%). In the majority of these cases it was performed in an appropriate and timely fashion (81.4%). Renal ultrasound altered management decisions in 3.8% of patients; 53.1% of patients were on nephrotoxic drugs at the time of their AKI and 61.2% of these patients had documentation of a timely cessation of nephrotoxics (or decision recorded as to clinical justification to continue). In cases where a renal dose adjustment was required based on creatinine clearance, this is documented to have been carried out appropriately in 71.4% of cases. Finally, advice from a nephrologist was felt to have been required in 10.2% of cases of AKI. This was sought in 72% of cases (45.6% telephone advice; 23.3% ward review; 32.1% care taken over by renal team).

**Discussion**

Our results demonstrate the high incidence of AKI among hospital in-patients and highlight the associated mortality (19%) and increased length of hospital stay (6.6 days). We have illustrated the differences in mortality between ‘hospital–acquired’ and ‘community-acquired’ AKI (27.3 vs. 11.8%) and highlighted some important risk factors for AKI (older age, frailty, diabetes and treatment with ACE inhibitor prior to admission). Our retrospective review of case note documentation highlighted several significant inadequacies in the recognition and management of AKI, most notably failure to recognize AKI (particularly mild AKI) in a timely fashion; inadequate recording of fluid balance and urine output and failure to withhold nephrotoxic drugs. AKI went unrecognized in nearly a quarter of patients, two-thirds of whom were discharged home without resolution of renal function. A small but significant proportion of these patients went on to develop CKD (8.6%).

The incidence of AKI in our study (15.5%) is higher than that historically quoted within the literature (5–7%). We postulate that this represents a combination of an older, more co-morbid population in our series and differences in the definition of AKI between studies. Conversely, the mortality rate for patients with AKI in our study is lower than that previously described in the literature. We believe that this is because our study includes all-comers with AKI while much of the existing literature focuses on the highly selective group of patients with critical illness admitted to the intensive care unit. Our non-selected patient group perhaps

<table>
<thead>
<tr>
<th>Table 3</th>
<th>Multivariate analysis of risk factors for AKI</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Hazard</td>
</tr>
<tr>
<td></td>
<td>ratio</td>
</tr>
<tr>
<td>CKD</td>
<td>1.6</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>3.08</td>
</tr>
<tr>
<td>ACE I</td>
<td>2.3</td>
</tr>
<tr>
<td>Diuretic</td>
<td>1.7</td>
</tr>
<tr>
<td>Frailty</td>
<td>3.4</td>
</tr>
<tr>
<td>Hypovolaemia</td>
<td>2.49</td>
</tr>
<tr>
<td>Hypotension (blood pressure &lt;100 systolic)</td>
<td>2.56</td>
</tr>
<tr>
<td>Infection</td>
<td>2.66</td>
</tr>
<tr>
<td>Age</td>
<td>1.27</td>
</tr>
<tr>
<td>Emergency admission</td>
<td>1.2</td>
</tr>
</tbody>
</table>

Data are presented as hazard ratios and 95% confidence intervals. Italics represent all the significant factors with P-value <0.05.
provides a better indication of the true burden of AKI on healthcare services.

The National Confidential Enquiry in Perioperative Deaths recently produced a report evaluating recognition and management of AKI in surgical patients who subsequently died, identifying failures in management in 50% of cases. Again, however, this report is subject to selection bias as only those patients who actually died had their care appraised. Our study provides a unique ‘real life’ insight into the management of AKI across the entire spectrum of illness permitting a representative assessment of the current standards of care afforded to all patients admitted to hospital with AKI. This analysis is essential as a baseline to inform and target future interventions, education/training and guidelines.

**Limitations**

The generalizability of our results is limited by the fact our study is single centre and lacks long-term (>3 months) outcome data. Short follow-up may lead to an underestimation of loss of renal function as patients with AKI commonly lose muscle mass leading to lower creatinine-based outcomes. Additionally, there are relatively small numbers of patients with AKI, particularly AKI 3, making it difficult to draw any robust conclusions from subgroup analysis. The retrospective nature risks observational bias and cofounders, however minimizes any Hawthorne effect that might be generated in a prospective observational study (i.e. behaviour and practice changing as a result of being studied and increased awareness raised by conducting the study). It was essential for the validity of this study (to ensure it was representative) that clinicians were unaware that an audit of practice being carried out. Any potential selection bias was minimized because the identification of patients with AKI was undertaken prospectively from electronic biochemistry records.

Perhaps the major limitation of this study is the subjectivity of categorization of the adequacy of the patients’ management. While attempts were made to limit subjectivity by double or triple reporting to ensure agreement, under- or overreporting of inadequacies in patient care risks measurement and reporting bias. Additionally, due to the retrospective nature of the study, it was only possible to appraise documentation of recognition and management of AKI. It is recognized that there are likely to have been instances where the clinician recognized the AKI but simply failed to document it.

Where possible, we have used a true baseline creatinine, which the majority of patients in our study had in the 3 months preceding hospital admission. However, where this was not possible, an MDRD-calculated creatinine was used. MDRD is a valid means of obtaining a surrogate baseline creatinine for RIFLE classification systems; however AKIN classification relies upon a true baseline creatinine >48 h prior to admission. There is evidence that the mortality between RIFLE classes may vary as much as 7–13.9% depending on the method used to define baseline creatinine. We found no significant difference in our results when the patients with MDRD estimated creatinine were removed. A lack of urine output data for some patients may also have led to underreporting of AKI in some cases.

**Risk factors for AKI**

The association between advancing age, drugs (ACE inhibitors, loop diuretics and gentamycin) and diabetes mellitus is well described in the existing literature. Other authors have also demonstrated an association between pre-existing CKD and AKI which was not borne out on multivariate analysis in this study. The most significant independent risk factor for AKI in this study was frailty, as perceived and documented by the admitting clinician [heart rate (HR) 3.4]. Frailty has previously not been described as a risk factor for AKI; however, it is recognized risk factor for poor cardiovascular, oncological and orthopaedic outcomes. There is an extensive body of geriatrics literature attempting to define frailty and a plethora of prognostic scoring systems exist. These, however, are only validated within an elderly population. Similarly, McDermid et al. conclude that a clinician’s perception of ‘frailty’ is equally valid for predicting intensive care unit outcomes as any other prognostic scoring system.

**The significance of ‘Mild AKI’**

In our study, 23.5% of patients had unrecognized AKI. In the majority of cases, this was mild, Stage 1 AKI which resolved spontaneously. Nevertheless, our findings, in accordance with those of other authors, indicate a significant increase in mortality associated with even Stage 1 AKI. Furthermore, our results would suggest that a small but significant proportion of patients in our study (8.6%) with mild, unrecognized AKI went on to develop CKD 3 months after discharge. The association between AKI and subsequent CKD is well documented with a recent meta-analysis quoting a hazards ratio of 8.8. Several other studies estimate that 8–22% of critically ill patients are discharged from hospital on haemodialysis; however, there is a relative paucity of data on medium to long-term follow-up of these patients. To our knowledge, this
is the first study that highlights the association between mild AKI and CKD.

Based on HES data from 2009 to 2010, AKI costs the NHS in England between £434 and £620 million/year (split equally between RRT, critical care facilities and increased bed days costs). Prevention of 30% of these cases though early recognition and intervention would save the NHS £130–£186 million/year, a third of these cases through early recognition and intervention would save the NHS £130–£186 million/year, highlighting the importance of appropriate management of mild AKI, to limit the inevitable progression of some cases to severe AKI or CKD.

The additional risk of mortality associated with AKI persists for at least a year beyond the insult. The long-term risk of death was greater in those not achieving complete recovery of renal function (HR 4.14; *P* < 0.001). The same study demonstrated that 10% of those patients who were discharged without recovery of renal function had further decline over the subsequent year, highlighting the importance of recognition and management of AKI in an attempt to prevent progression to CKD. While our study does not have long-term outcome data, our medium-term data at 3 months, indicates a worrying trend of patients discharged with unrecognized AKI. In the majority of cases, this was mild and we postulate may have been the result of clinician mis-interpretation of our hospital computerized biochemistry reporting. Our electronic reporting systems (as with many hospitals in the UK) automatically highlight any results that are perceived to be abnormal (in our case any eGFR <60). However, this study illustrates that this value may be inadequate and misleading to clinicians as there are a significant number of patients with AKI who had an eGFR >60. We postulate that a lack of awareness of caveats in the electronic biochemistry reporting system may have contributed to a number of patients with AKI being discharged without adequate follow-up.

**Recommendations**

A national audit of clinical practice for all patients with AKI is necessary prior to the development of clinical guidelines to ensure that key areas for intervention are appropriately targeted. Larger patient numbers are required to permit meaningful analysis of subgroups, e.g. AKI 1. The long-term prognostic impact of mild AKI requires to be evaluated to determine whether early recognition and intervention prevents progression to CKD. Thereafter, an interventional study with ‘care bundles’ much as those employed by the Surviving Sepsis Campaign will facilitate pragmatic, evidence-based management strategies.

**Conclusion**

AKI is common in hospitalized patients and associated with a significant increase in hospital stay and mortality. ‘Hospital-acquired AKI’ is associated with significantly higher mortality than ‘community-acquired AKI’. It is often found in conjunction with other organ failure and in many cases is not preventable. Nevertheless we have identified risk factors for AKI and demonstrated that in many cases current documentation of the recognition and management of AKI is inadequate. Particularly concerning is failure to identify and follow-up mild AKI given the association with CKD. Clinicians need to be more vigilant of small creatinine rises to permit early intervention especially among elderly and frail patients.

**Conflict of interest:** None declared.

**References**


