A prospective study of incidence and outcome of acute kidney injury among hospitalised patients in Malaysia (My-AKI)

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ABSTRACT

Introduction: The incidence of acute kidney injury (AKI) among hospitalised patients has not been well studied in Malaysia.

Materials and Methods: We conducted a prospective, multicentre study in seven hospitals in West Malaysia. All the adults admitted in March 2017 fulfilling Kidney Disease Improving Global Outcomes (KDIGO) criteria for AKI were included.

Results: Of the 34,204 patients screened, 2,457 developed AKI (7.18%), 13.1% of which occurred in intensive care unit (ICU). There were 60.2% males with a mean age of 57.8 (±17.5) years. The most common comorbidities were hypertension (55.0%), diabetes (46.6%), ischaemic heart disease (15.1%) and chronic kidney disease (12.0%). The commonest causes of AKI were sepsis (41.7%), pre-renal (24.2%) and cardiorenal syndrome (10.8%). Nephrotoxin exposure was reported in 31%. At diagnosis, the proportion of AKI stages 1, 2 and 3 were 79.1%, 9.7%, 11.2%, respectively. Referral to nephrologists was reported in 16.5%. Dialysis was required in 176 (7.2%) patients and 55.6% were performed in the ICU. Acidosis (46.2%), uraemia (31.6%) and electrolyte disturbance (11.1%) were the commonest indications. Continuous renal replacement therapy (CRRT) was required in 14%. The average length of hospital stay was 9.5 days. In-hospital mortality was 16.4%. Among survivors, full and partial renal recovery was seen in 74.7% and 16.4% respectively while 8.9% failed to recover. After a mean follow-up of 13.7 months, 593 (30.2%) of survivors died and 38 (1.9%) initiated chronic dialysis. Mortality was highest among those with malignancies (Hazard Ratio, HR 2.14), chronic liver disease (HR 2.13), neurological disease (HR 1.56) and cardiovascular disease (HR 1.17).

Conclusion: AKI is common in hospitalised patients and is with associated high mortality during and after hospitalisation.

KEYWORDS:
Acute kidney injury, prospective study, Malaysia

INTRODUCTION

Acute kidney injury (AKI) is a condition with rapid reduction in renal function. It is independently associated with morbidity, mortality, increased length of hospital stays, progression of chronic kidney disease (CKD), increased need for renal replacement therapy (RRT) and increased healthcare costs. The incidence varies from 5 to 20% of hospitalised patients in developed countries.

In Malaysia, the incidence of AKI among hospitalised patients has not been well studied. Single centre studies over the past few decades have shown increasing incidence of AKI among hospitalised patients.[1-3] The Malaysian Registry of Intensive Care in 2016 reported that 15.7% of the patients in intensive care unit (ICU) developed AKI within the first 24 hours of their admission.[4]

In the past there has been a lack of standardisation in defining AKI. Recently, consensus has been reached on the universal definition and staging of AKI after harmonising the previous definitions and staging systems to allow early detection and management of AKI.[5,6] This will also enable future research on the incidence, aetiologies, risk factors, outcomes and efficacy of therapeutic interventions for AKI globally.

All hospitalised patients are at risk of AKI either through their presenting illness, complications or iatrogenic causes. The rising incidence of AKI in developed countries is largely driven by sepsis, dehydration or volume depletion, trauma and exposure to nephrotoxic drugs. It may also be related to aggressive medical and surgical therapies on an ageing population with multiple comorbidities. These patients are mostly admitted under the care of non-nephrology healthcare professionals, who may not be familiar with the early detection of AKI. Prevention and optimum care of AKI

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in these patients may not be provided in a timely manner.[7] In 2009, the National Confidential Enquiry into Patient Outcome and Death (NCEPOD) in the United Kingdom described systemic deficiencies in the care of patients who died from AKI and only 50% of them had received "good" care. Deficiencies included failure in AKI prevention, recognition, therapy and timely access to specialist services.[8] Published series on AKI suggested that up to 30% of cases may be preventable, with a further significant percentage potentially remediable through simple interventions such as volume repletion, discontinuing and/or avoiding certain potentially nephrotoxic agents and earlier recognition of conditions causing rapid progression of AKI.[9-11] It is therefore, important to raise awareness of AKI and its prevention among healthcare professionals.

AKI in developing countries is commonly due to gastroenteritis or infections-related diseases such as malaria, leptospirosis and dengue. These are largely preventable and occur in young and healthy individuals.[12] Patients with AKI have a poorer prognosis with mortality ranging from 10 to 80% depending on the patient population.[13-15] A total of 5 to11% of ICU patients will require renal replacement therapy (RRT) and long-term survival is poor.[16-19] AKI is a risk factor for the development of chronic kidney disease (CKD), worsening of pre-existing CKD and may lead to end stage kidney disease (ESKD).[20-22]

The purpose of this study is to determine the incidence and evaluate the causes, risk factors and outcomes of AKI among hospitalised adult patients in Malaysia.

MATERIALS AND METHODS

This is a prospective, observational study designed to determine the incidence and outcomes of AKI in hospitalised patients (18 years and above) in seven hospitals in western states of Peninsular Malaysia.

Approval and waiver of informed consent was obtained from the Ethics Committee as no intervention was planned and confidentiality of patient information was maintained (NMRR-16-2033-32927). This study was supported by a research grant from the Ministry of Health Malaysia.

Patients were identified in March 2017. Daily laboratory data of serum creatinine of patients admitted from 12:01 am 1st March 2017 until 12:00 midnight 31st March 2017 was obtained from the pathology department of each hospital. A screening list was generated using STATA programme version 10 to identify potential patients fulfilling the criteria for AKI.[23] All the new admissions during this study period were screened for AKI using the Kidney Disease Improving Global Outcomes (KDIGO) 2012 definition of AKI.[5] Urine output was excluded as a criterion for recruitment because it was not possible to obtain accurate records of urine output for patients in the general wards. Patients with AKI who were readmitted during the study period were excluded.

Nephrologists were not involved in the management of the patients unless a referral was made. Patients alerted by the screening process were reviewed within 48 hours. Clinical and laboratory data from the patients' medical notes were reviewed and possible causes of AKI were identified.

Inclusion criteria for the study included hospitalised adults (18 years and older) who fulfilled the criteria for AKI during the study period. Patients with ESKD on dialysis and renal transplant patients or patients with increasing creatinine due to advancing CKD (as determined by the investigators) were excluded.

AKI was defined as an increase in serum creatinine of 0.3mg/dl (26.5µmol/l) within 48 hours or an increase in serum creatinine to ≥1.5 times baseline, which is known or presumed to have occurred within the prior 7 days.[5]

Baseline creatinine was defined as creatinine level at or within 7 days before the hospital admission, or the lowest creatinine (excluding the post dialysis creatinine if dialysis was initiated) during the index hospitalisation for those whose baseline creatinine were unknown.[24-26]

Serum creatinine concentration was measured by Jaffe method at six sites and kinetic enzymatic method at one site. The creatinine assay calibration was traceable to isotope dilution mass spectroscopy method.

Renal recovery was determined at hospital discharge. Full renal recovery was defined as return of serum creatinine to or below baseline or within 20% of baseline creatinine. Partial renal recovery was defined as serum creatinine which remained 20% above the baseline, but below 50% and not dialysis dependent or if previously required dialysis and is now dialysis independent. Failure to recover renal function was defined as serum creatinine remaining 50% above the baseline or dialysis dependence.[27] The total number of hospital admissions of adults (18 years and older) was compiled from the admissions office of each hospital.

The electronic records of the National Registration Department (NRD) were searched for deaths up till 31st December 2018. A search of the National Renal Registry (NRR) was also made to identify those who started on chronic dialysis up till 31st December 2018. Both NRD and NRR datasets included only Malaysian citizens.

Statistical analyses were performed using STATA version 10. Normally distributed continuous variables were described using mean and standard deviation, or median with interquartile range (IQR) for variables that were not normally distributed. Categorical data were described using proportions and percentages. Normally distributed data were analysed with t-test and one-way ANOVA. Categorical data were compared using a Pearson Chi-squared test. Data that were not normally distributed were analysed using Mann-Whitney U-test, Wilcoxon signed rank test, and Kruskal-Wallis test.

Univariate and multivariate survival analysis were performed using the likelihood ratio test of the stratified Cox proportional hazard model to determine the significance of covariates for the Malaysian cohort. Covariates included in the univariate analysis were age, gender, race, comorbid

conditions (hypertension, diabetes mellitus, chronic lung disease, endocrine disease, malignancy, chronic liver disease, arthropathy, gastrointestinal disease, psychiatric disease, haematological disease, connective tissue disease, HIV infection), CKD, cardiovascular disease, neurological disease, primary aetiology (sepsis, prerenal, cardiorenal syndrome, obstructive uropathy, drug induced, hypertensive emergency, ischaemic acute tubular necrosis, hepatorenal syndrome, intratubular obstruction, glomerulonephritis, dengue, malaria, leptospirosis, contrast induced), history of exposure to nephrotoxic drugs, referral to nephrology, admission to ICU and dialysis dependence. Factors which were found to be significant were included in the multivariate analysis. P-value of <0.05 was considered statistically significant.

RESULTS

A total of 34,204 adult admissions were screened from seven hospitals, of which 2,457 developed AKI (incidence rate 7.18%) (Figure 1).

There were 1,480 (60.2%) males and the mean age on admission was 57.8 (±17.5) years. The ethnic distribution reflects the general population but there were 109 foreigners (4.4%). The most common comorbidities were hypertension (55.0%), diabetes mellitus (46.6%), ischaemic heart disease (15.1%), chronic lung disease (12.2%) and CKD (12.0%) (Table I).

A sole aetiology was found in 1 902 (77.4%) while 22.5% had multiple causes for AKI. The common causes of AKI were sepsis (41.7%), prerenal (24.2%) and cardiorenal syndrome (10.8%).

The common causes of sepsis included pneumonia, diabetic foot ulcers, cellulitis, intra-abdominal sepsis and pyelonephritis/urinary tract infection. Prerenal causes included haemorrhage, acute gastroenteritis and dehydration. Cardiorenal syndrome was caused by cardiogenic shock, congestive heart failure, valvular heart disease and other cardiac diseases.

There were 175 cases of dengue fever, 20 with leptospirosis, one snake bite and malaria each, 72 with gastroenteritis and 17 pregnancy-related. Dengue contributed to 7.1% of AKI. The causes of pregnancy related AKI included preeclampsia, abruptio placenta, septic abortion, ruptured ectopic pregnancy and hyperemesis gravidarum.

At the diagnosis, 79.1% were at KDIGO stage 1, 9.7% were at stage 2 and 11.2% were at stage 3.

Exposure to nephrotoxins was reported in 760 (31%) of 2,457 patients and this included angiotensin converting enzyme inhibitors and angiotensin receptor blockers (432), diuretics (323), non-steroidal anti-inflammatory drugs (78), traditional medications (58), contrast media (51), aminoglycosides (9), other antibiotics (9), illicit drugs (10), antivirals (8) and antifungals (2). However, only 46 (1.9%) cases were deemed drug-associated AKI and 16 (0.7%) were related to contrast media.

The nephrology departments received 405 (16.5%) referrals for further clinical management. Of these referrals, 176 (43.4%) patients were started on RRT (Table II). A total of 404 (16.4%) AKI patients were admitted to ICU during their hospitalisation. Of these, 322 (13.1%) developed AKI while in ICU.

Patients needing dialysis made up 7.2% of the total AKI population and the majority (55.6%) were performed in the ICU. Acidosis (46.2%), uraemia (31.6%) and electrolyte disturbance (11.1%) were the commonest indications for RRT (Table III).

At the start of dialysis, the mean blood urea was 28.1 (±12.1)mmol/l, serum creatinine was 548.5 (±368.3) µmol/l and potassium were 4.63 (±1.0)mmol/l. Of the 171 with full data 24 (14.0%) required continuous renal replacement therapy (CRRT) and 147 (86.0%) haemodialysis (HD) or slow low efficiency dialysis (SLED). A total of 20 patients on CRRT were on inotropic support and another two had intracranial bleed. Of the 24 patients on CRRT, 20 were on continuous veno-venous hemofiltration (CVVH) and two each were on continuous veno-venous haemodialysis (CVVHD) and hemodiafiltration (CVVHDF). The patients on CRRT made up 25.3% of those dialysed in ICU.

In-hospital mortality was 16.4% (404 deaths). At discharge, 74.7% of survivors had full renal recovery, 16.4% partial and 8.9% failed to recover. The mean serum creatinine on discharge was 119.5 (±89.8) µmol/l. The average length of stay was 9.49 (±11.4) days. Five patients still required haemodialysis on discharge (0.2% of all patients).

The outcome of 1,964 Malaysians from this cohort was traced from the NRD and NRR as of 31st December 2018 (89 foreigners were excluded and considered lost to follow-up at discharge). Mean follow-up of the total cohort was 13.74 (±9.8) months, ranging from one day to 22 months and the median follow-up was 21.5 months. A total of 593 patients died after discharge from hospital (30.2%). Mortality was highest in the first 3 months of admission. Survival of the cohort at three months and one year was 71% and 63% respectively. A total of 38 patients (1.9% of those on follow-up) subsequently initiated chronic dialysis (34 HD, 4 peritoneal dialysis) (Table IV, Figure 2).

In the multivariate analysis, advanced age and having certain comorbidities increased the mortality risk. Mortality was higher in those with chronic liver disease (Hazard Ratio, HR 2.13, 95% Confidence Interval, 95%CI 1.63, 2.76), malignancy (HR 2.14, 95%CI 1.79, 2.55), neurological disease (HR 1.56, 95%CI 1.33, 1.84) and cardiovascular disease (HR 1.17, 95%CI 1.02, 1.35). In contrast, diabetes mellitus and CKD had no significant effect on mortality.

The risk was higher among foreigners (HR 3.02, 95%CI 1.91, 4.79). Hepatorenal syndrome (HR 1.94, 95%CI 1.24, 3.04), cardiorenal syndrome (HR 1.35, 95%CI 1.11, 1.65) and sepsis-associated AKI (HR 1.25, 95%CI 1.10, 1.43) were poor predictors for survival. Dengue fever (HR 0.13, 95%CI 0.05, 0.32) and ischaemic acute tubular necrosis (HR 0.35, 95%CI 0.17, 0.70) were associated with better outcomes.

Table I: Demography of patients with Acute Kidney Injury (AKI) (N=2 457)

Parameters	No. (%)
Age (Mean±SD)	57.8 (±17.5)
Gender	
Male	1480 (60.2)
Female	977 (39.8)
Race	
Malay	1283 (52.2)
Chinese	579 (23.6)
Indian	469 (19.1)
Foreigner	109 (4.4)
Others	16 (0.7)
Comorbidity	
Yes	2002 (81.5)
No	455 (18.5)
General comorbidities	
Hypertension	1352 (55.0)
Diabetes mellitus	1144 (46.6)
Chronic lung disease	299 (12.2)
Endocrine disease	244 (9.9)
Malignancy	194 (7.9)
Chronic liver disease	90 (3.7)
Arthropathy	59 (2.4)
Gastrointestinal disease	51 (2.1)
Psychiatric disease	32 (1.3)
Haematological disease	24 (1.0)
Connective tissue disease	22 (0.9)
HIV infection	18 (0.7)
Renal comorbidities	
Chronic kidney disease	294 (12.0)
Prostate disease	74 (3.0)
Urolithiasis	48 (2.0)
Urological malignancy	8 (0.3)
Obstructive uropathy	6 (0.2)
Pyelonephritis	1 (0.0)
Neurogenic bladder	1 (0.0)
Others	2 (0.1)
Cardiovascular comorbidities	
Ischemic heart disease	371 (15.1)
Congestive heart failure	133 (5.4)
Cardiac arrhythmias	62 (2.5)
Congenital heart disease	22 (0.9)
Peripheral vascular disease	20 (0.8)
Valvular heart disease	13 (0.5)
Aortic aneurysm	2 (0.1)
Others	7 (0.3)
Neurological comorbidities	
Stroke/Transient ischaemic attack	197 (8.0)
Chronic neurological diseases	54 (2.2)
Others	3 (0.1)
Miscellaneous comorbidities	41 (1.7)
Primary aetiology of AKI	
Sepsis associated AKI	1023 (41.7)
Prerenal	595 (24.2)
Cardiorenal syndrome	266 (10.8)
Dengue	175 (7.1)
Obstructive uropathy	96 (3.9)
Drug associated AKI	46 (1.9)
Hypertensive emergency	45 (1.8)
Ischaemic acute tubular necrosis	42 (1.7)
Hepatorenal syndrome	31 (1.3)
Leptospirosis	20 (0.8)
Intratubular obstruction	19 (0.8)
Glomerulonephritis	19 (0.8)
Contrast induced AKI	16 (0.7)
Malaria	1 (0.1)
Others	60 (2.4)
KDIGO stage AT AKI diagnosis	
1	1944 (79.1)
2	237 (9.7)
3	276 (11.2)
Mean baseline serum creatinine (µmol/l)	104.2 (±70.8)

KDIGO- Kidney Disease Improving Global Outcomes

Table II: In-patient course of acute kidney injury

Parameters	Number (%)
Referred to nephrology	
Yes	405 (16.5)
No	2,052 (83.5)
History of ICU admission	
Yes	404 (16.4)
No	2,053 (83.6)
AKI diagnosed in ICU	
Yes	322 (13.1)
No	2,135 (86.9)
Started on dialysis	
Yes	176 (7.2)
No	2,281 (92.8)

ICU-Intensive Care Unit

Table III: Renal replacement therapy results, N = 171, 5 missing

Parameters	No (%)
Where was RRT first started	
ICU	95 (55.6)
Non-ICU	76 (44.4)
Indication for first RRT	
Acidosis	79 (46.2)
Uraemia	54 (31.6)
Electrolyte disturbance	19 (11.1)
Overload	9 (5.3)
Oliguria	9 (5.3)
Paraquat poisoning	1 (0.5)
First RRT mode	
IHD/SLED	147 (86)
CRRT	24 (14)
PD	0 (0)
Mean±SD	
Blood urea at initiation of RRT (mmol/l)	28.1 (±12.1)
Serum creatinine (µmol/l)	548.5 (±368.3)
Bicarbonate (N = 166)	15.4 (±5.0)
Potassium (mmol/l) (N = 170)	4.63 (±1.0)
Lactate mmol/l (N = 69)	4.4 (±4.0)

ICU-Intensive Care Unit; CRRT - Continuous renal replacement therapy; IHD - Intermittent haemodialysis; SLED - Slow low efficiency dialysis; PD - Peritoneal dialysis

Table IV: Matching with National Renal Registry for Malaysians who developed end stage kidney disease (N=1 964) excluding 89 foreigners

Parameter	Number	%
Matched	38	1.9
Not matched	1 926	98.1
Modality		
Haemodialysis	34	89.5
Peritoneal dialysis	4	10.5
Outcome		
Alive	32	84.2
Dead	6	15.8

Date of study: March 2017
Date of matching: 31 December 2018

Table V: Univariate and multivariate analysis of risk factors associated with mortality for patients with Acute Kidney Injury from admission till 31st Dec 2018 (N = 2 457)

Parameters	5	Univariate			Multivariate		
	Number	HR	95% CI	*p* value	HR	95% CI	*p* value
Risk factors							
Age		1.03	1.02-1.03	<0.001	1.02	1.02-1.03	<0.001
Ethnicity							
Malay	1 283	1			1		
Chinese	579	1.36	1.19-1.56	<0.001	1.07	0.93-1.23	0.355
Indian	469	0.87	0.74-1.03	0.097	0.88	0.74-1.03	0.114
Foreigner	109	1.84	1.17-2.89	0.008	3.02	1.91-4.79	<0.001
Others	16	1.04	0.56-1.95	0.890	0.88	0.47-1.65	0.696
Diabetes mellitus							
No		1			1		
Yes	1144	1.22	1.09-1.36	0.001	1.09	0.96-1.25	0.181
Hypertension							
No		1			1		
Yes	1 352	1.22	1.09-1.37	0.001	0.84	0.74-0.97	0.018
Chronic liver disease							
No		1			1		
Yes	90	2.00	1.57-2.54	<0.001	2.13	1.63-2.76	<0.001
Malignancy							
No		1			1		
Yes	194	2.18	1.84-2.58	<0.001	2.14	1.79-2.55	<0.001
Chronic kidney disease							
No		1			1		
Yes	434	1.34	1.15-1.57	<0.001	1.02	0.86-1.20	0.818
Cardiovascular disease							
No		1			1		
Yes	630	1.38	1.21-1.57	<0.001	1.17	1.02-1.35	0.028
Neurological disease							
No		1			1		
Yes	254	1.73	1.48-2.04	<0.001	1.56	1.33-1.84	<0.001
Aetiology: Ischaemic ATN							
No		1			1		
Yes	42	0.36	0.18-0.72	0.004	0.35	0.17-0.70	0.003
Aetiology: Cardio-renal syndrome							
No		1			1		
Yes	266	1.28	1.08-1.52	0.004	1.35	1.11-1.65	0.003
Aetiology: Sepsis associated AKI							
No		1			1		
Yes	1023	1.34	1.19-1.50	<0.001	1.25	1.10-1.43	0.001
Aetiology: Hepatorenal syndrome							
No		1			1		
Yes	31	2.08	1.40-3.09	0.001	1.94	1.24-3.04	0.004
Aetiology: Dengue							
No		1			1		
Yes	175	0.05	0.02-0.13	<0.001	0.13	0.05-0.32	<0.001
Management risk profiling							
History of ICU admission							
No		1			1		
Yes	404	1.65	1.433-1.89	<0.001	1.67	1.43-1.95	<0.001
Nephrology referral							
No		1			1		
Yes	405	2.03	1.78-2.32	<0.001	1.48	1.23-1.79	<0.001
Requirement for RRT							
No		1			1		
Yes	176	2.67	2.25-3.16	<0.001	1.45	1.14-1.85	0.003

HR-Hazard Ratio; 95%CI-95% Confidence Intervals; ATN-Acute tubular necrosis, Hazard ratio (HR) for death among hospitalised patients with AKI.

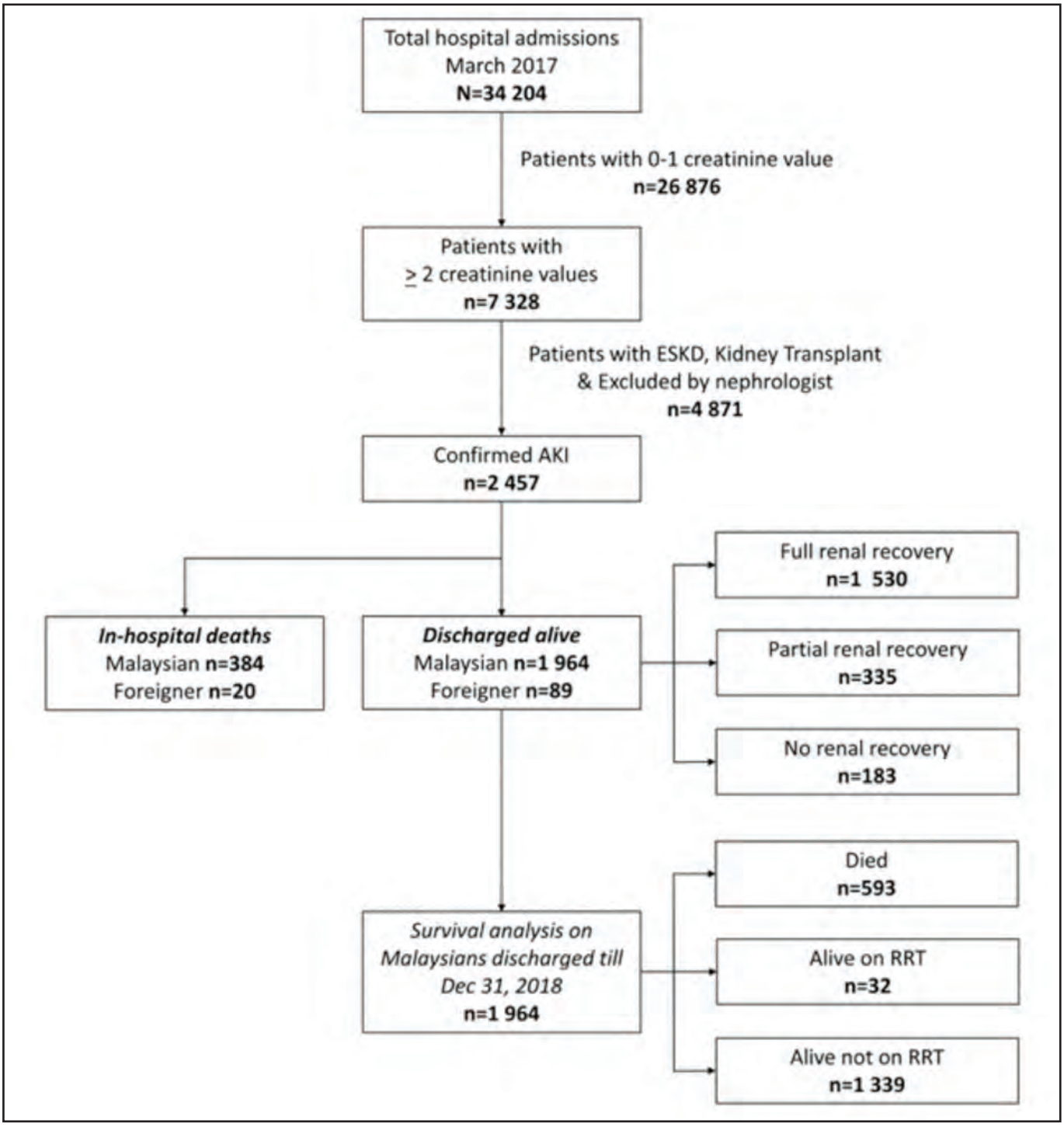


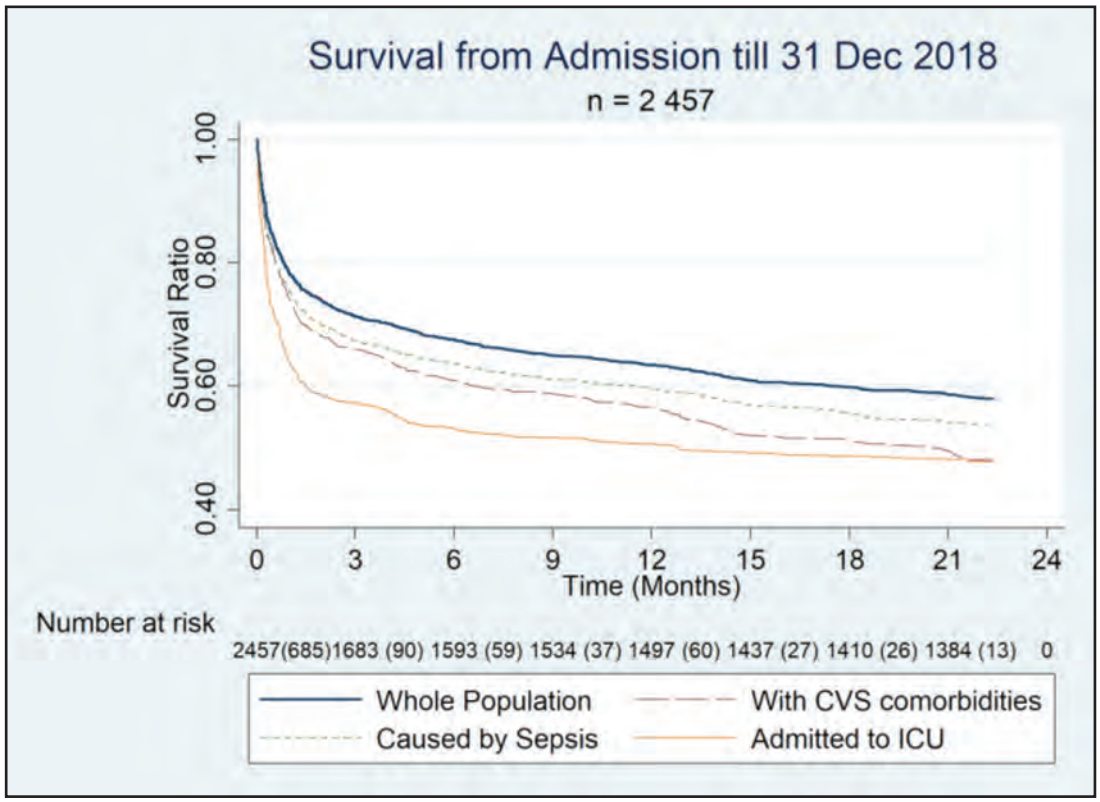
Fig. 1: Flow diagram.

Nephrology referral (HR 1.48, 95%CI 1.23, 1.79), ICU admission (HR 1.67, 95%CI 1.43, 1.95) and dialysis requirement (HR 1.45, 95%CI 1.14, 1.85]) also predicted death (Table V, Figure 2).

DISCUSSION

This is the first prospective, multicentre study in Malaysia using the KDIGO AKI 2012 criteria to define the incidence, aetiologies and outcomes of AKI among hospitalised patients. We included an extended follow-up duration up to 22 months.

The baseline creatinine was determined as the lowest creatinine during the hospitalisation as serum creatinine within seven days before hospitalisation was commonly not available. This methodology was adopted from the KDIGO AKI and various other groups.[26,28,29]



Hospital mortality: 404 out of 2 457 = 16.4%

Population	Number of subjects	Number of deaths (Rate)	In-hospital mortality (Rate)	Survival				
				30 days	60 days	90 days	1 year	1.5 year
Whole population	2 457	997 (40.6%)	404 (16.4%)	0.78	0.74	0.71	0.63	0.60
With CVS Comorbidities	535	277 (51.8%)	100 (36.1%)	0.74	0.68	0.66	0.55	0.52
Caused by sepsis	1 023	461 (45.1%)	209 (45.3%)	0.75	0.70	0.67	0.59	0.56
Admitted to ICU	404	204 (50.5%)	136 (66.7%)	0.64	0.59	0.57	0.50	0.49

CVS-cardiovascular, ICU-intensive care unit

Fig. 2: Survival curve for all AKI patients from admission till 31st December 2018 (N = 2 457).

All of the participating centres are public hospitals funded by the Ministry of Health Malaysia and are equipped with intensive care units. Four centres are tertiary referral hospitals, and the remaining three centres are district hospitals.

The incidence of hospitalised AKI (7.2%) was comparable to Yang L et al.,28 but lower than other studies in China (11.6%), Japan (11.6%) and United States of America (18.3%).[29-31] The discrepancies might be due to differences in methodology and population case-mix. The mean age of our cohort (57.8 years) was similar to study by Yang L et al., but relatively younger compared to other reports.

Four out of five patients (81.5%) who developed AKI had at least one comorbidity. There was a high prevalence of diabetes in our study cohort, consistent with the rising prevalence of 17.5% among the general Malaysian population.[32]

Malaysia is categorised among upper middle-income countries by the World Bank.[33] The rate of AKI incidence attributed to gastroenteritis was 3%. Gastroenteritis caused 19% of AKI in a series from Malawi[34] and 23% of AKI with febrile illness from a recent study in India.[35] Only a small proportion of AKI was associated with tropical diseases and pregnancy. Leptospirosis, malaria and snake bite causing AKI was also rare. The initiative from the International Society of Nephrology aspires to eliminate preventable deaths from AKI by 2025.[36] All the patients who required dialysis were referred to nephrologists to facilitate transition between different modalities of dialysis such as CRRT, SLED and intermittent HD. Peritoneal dialysis was not utilised to treat AKI patients in any of the participating centres.

The patients who were admitted to the ICU and acquired AKI were usually more ill hence they have higher mortality. Most of those who received CRRT were on inotropic support.

The overall in-hospital mortality was 16.4%, mostly in relation to the cause of AKI and severity of illness. There was also an increased death rate after being discharged home (30.2%) and 1.9% progressed to end-stage kidney failure during an average follow-up of 13.7 months. A single centre study from Singapore reported a mortality rate of 9.4% among AKI patients within six months of post-discharge from hospital for AKI.[37] Another study from Canada showed a mortality of 28.0% at one year.[38] Therefore, it is important that patients with a history of AKI are followed up long term. Foreigners (n=89, 3.6%) were excluded from follow-up analysis as they were not registered with NRD and NRR.

Besides this, dengue fever is a cause for concern as it is endemic in Malaysia. Dengue as a cause of AKI is unique to the region. Our study showed that dengue patients who developed AKI had five times higher mortality (1.1%) compared to the national reported dengue mortality of 0.21%.[39] However, from multivariate analysis, dengue had better outcome compared to other causes of AKI. Factors that contribute to better prognosis of dengue include nationwide active surveillance, adherence to standard protocol and clinical practice guidelines as well as dengue outcome being a key performance indicator for administrators in the Ministry of Health.[40]

The main treatment is assessment of fluid status and timely fluid resuscitation. The condition tends to improve when the proper amount of fluid has been replaced, although in a small percentage, complications can arise, with deterioration to shock syndrome and the patient goes into ICU.

This multicentre study revealed the common causes, population at risk of developing AKI and the predictors of mortality. The incidence of AKI in hospitals and mortality may be reduced by early detection and prompt management of risk factors. Renal replacement therapy escalates the costs and mortality.

The strength of the study includes the large population studied, inclusion of multiple sites and the use of the KDIGO 2012 criteria for AKI.

However, there are limitations to the study. The statistical power of this study might improve with longer duration of follow-up and involvement of more sites especially from East Malaysia. Case-mix from this region might differ from West Malaysia. Apart from this, urine output criteria for diagnosis of AKI were not included, and hospitalised patients who only had a single or no serum creatinine test were excluded. This might underestimate the actual number of AKI.

CONCLUSION

Acute Kidney Injury (AKI) is common in hospitalised patients. It is associated with high in-patient mortality and increased mortality even after discharge. There is a risk of end stage kidney disease (ESKD), and patients need continued surveillance. It is recommended to have a guideline for AKI in Malaysia and to raise awareness so that there is earlier diagnosis and treatment of this serious condition.

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DISCLOSURES

None

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