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Original Article

Outcomes of acute kidney injury in children at Muhammad Husin Hospital, Palembang

Hertanti Indah Lestari, Dahler Bahrun, Eka Intan Fitriana

Abstract

Background Acute kidney injury (AKI) is a common problem in hospitalized pediatric patients, with effects on morbidity and mortality.

Objectives To assess for the incidence and common etiologies of AKI, as well as to review factors that affect patient outcomes at Muhammad Husin Hospital, Palembang.

Methods We reviewed data from our nephrology registry from January 2010 to June 2013. Independent variables were age, stage and etiology of AKI, requirement of renal replacement therapy (RRT), and PICU admission. The dependent variable was patient outcomes, categorized as survived or died. Association between clinical data and outcomes were analyzed by Chi-square test.

Results The incidence of AKI was 28.3%. Using the pediatric risk, injury, failure, loss, end stage renal disease (pRIFLE) criteria, 65 (36.7%) patients were in the risk stage, 56 (31.6%) in the injury stage, and 56 (31.6%) in the failure stage. Twelve (6.8%) patients required RRT and 29 (16.4%) patients were admitted to the PICU. The mortality rate from AKI was 20.9%. The common etiologies of AKI were acute glomerulonephritis (55 subjects; 31.1%), multiple organ dysfunction (24 subjects; 13.6%), dehydration (23 subjects; 13.0%), hypoalbuminemia (20 subjects; 11.3%), heart failure (11 subjects; 6.2%) and nephrotoxic agents (12 subjects; 6.8%). The mortality rate was significantly higher in children of younger age (<5 years) (P=0.0001), in the failure stage of AKI (P=0.014), with non-renal origin of illness (P=0.0001) and those with an indication for PICU admission (P=0.0001).

Conclusion AKI is found in one-third of nephrology patients. The most common etiology of AKI is acute glomerulonephritis. One-fifth of patients with AKI do not survive. Recognition of risk factors and detection of AKI in early stages might improve patient outcomes. **[Paediatr Indones. 2014;54:266-72.]**

Keywords: *acute kidney injury, pRIFLE*

Acute kidney injury (AKI), formerly known as acute renal failure (ARF), is a common problem in hospitalized pediatric patients, leading to increased patient morbidity and mortality.

The precise incidence of AKI in children is unknown, but tends to be high in hospitalized children.[1] Variations in epidemiological data are due to differences in definitions and classifications used among studies.[1] There are many definitions of AKI in adults and children, and many methods to diagnose AKI, however, serum creatinine level is the preferred test. Since the *Acute Dialysis Quality Initiative Group* (ADQI) proposed the RIFLE criteria in 2004, the classification has gained wide popularity in nephrology research. In 2007, the *Acute Kidney Injury Network* (AKIN) modified the classifications for pediatric patients and termed them the pRIFLE criteria.[2-7]

Depending on the etiology, there are three types of AKI: pre-renal injury (pre-renal AKI), intrinsic renal disease and obstructive uropathy (post-renal AKI).[8,9] The etiology of AKI also varies in different

From the Nephrology Division, Department of Child Health, Sriwijaya University Medical School/Dr. Muhammad Husin Hospital, Palembang, Indonesia.

Reprint requests to: Hertianti Indah Lestari. Department of Child Health, Sriwijaya University Medical School, Jalan Jendral Sudirman km 3.5 Palembang, Indonesia. Tel. +62-8153821666. E-mail: hertantilestari@yahoo.com.

populations due to underlying disease epidemiology. The aims of this study were to assess for the incidence and common etiologies of AKI, as well as to review factors that affect outcomes at Muhammad Husin Hospital, Palembang, South of Sumatera.

Methods

This retrospective study was conducted at Muhammad Husin Hospital in Palembang, a regional referral center in the southern region of Sumatera. We reviewed data from our nephrology registry from January 2010 to June 2013. The available data were patient's identity, physical and laboratory examinations at the time of admission including plasma creatinine level, underlying diseases, requirement for renal replacement therapy (RRT), PICU admission, and patient outcomes.

Diagnosis and classification of AKI were determined according to the pRIFLE criteria: risk of renal dysfunction (risk), injury to the kidney (injury), failure or loss of kidney function and end-stage renal disease (ESRD). The AKI stages were defined as follows: risk for estimated creatinine clearance (eCCl) decrease of 25%, injury for eCCl decrease of 50%, failure for eCCl decrease of 75% or <35 mL/min/1.73m^2, loss for persistent failure > 4 weeks, and ESRD for persistent failure > 3 months.[2] Etiology/risk factors of AKI were determined by the presence of underlying diseases and classified into pre-renal, renal (intrinsic) and post-renal factors.[8,9] Patient outcomes were categorized as survived and died within less than 4 weeks. Associations between clinical data and outcomes were analyzed by Chi-square test.

Results

There were 626 patients in our nephrology registry during the study period. The incidences of AKI were 26% in 2010, 22.6% in 2011, 33.3% in 2012, and 18.1% in the 1st semester of 2013, with an overall

Table 1. Characteristics of patients with AKI by year (n=177)

Characteristics	All		2010		2011		2012		1st semester 2013	
	n	%	n	%	n	%	n	%	n	%
Gender										
Male	110	62.1	27	58.7	23	57.5	36	61.0	24	75.0
Female	67	37.9	19	41.3	17	42.5	23	39.0	8	25.0
Age										
Neonates	4	2.3	2	4.3	0	0.0	0	0.0	2	6.2
1 – 6 mo	13	7.3	6	13.0	2	5.0	5	8.5	0	0.0
>6 – 12 mo	8	4.5	3	6.5	2	5.0	1	1.7	2	6.2
>1 – 5 y	34	19.2	9	19.6	7	17.5	13	22.0	5	15.6
>5 – 10 y	63	35.6	15	32.6	16	40.0	22	37.3	10	31.2
> 10 y	55	31.1	11	23.9	13	32.5	18	30.5	13	40.6
pRIFLE criteria										
Risk	56	31.6	18	39.1	14	35.0	17	28.8	7	21.9
Injury	56	31.6	18	39.1	12	30.0	20	33.9	6	18.8
Failure	65	36.7	10	21.7	14	35.0	22	37.3	19	59.4
Etiology										
Pre-renal	78	44.1	20	43.5	14	35.0	31	52.5	13	40.6
Renal	89	50.3	25	54.3	24	60.0	22	37.3	18	56.2
Post-renal	10	5.6	1	2.2	2	5.0	6	10.2	1	3.1
Management										
Supportive	165	93.2	45	97.8	36	90.0	56	94.9	28	87.5
RRT	12	6.8	1	2.2	4	10.0	3	5.1	4	12.5
Intensive care										
Non-PICU	148	83.6	41	89.1	38	95.0	45	76.3	24	75.0
PICU	29	16.4	5	10.9	2	5.0	14	23.7	8	25.0
Outcomes										
Survived	140	79.1	33	71.7	35	87.5	48	81.4	24	75.0
Died	37	20.9	13	28.3	5	13.5	11	18.6	8	25.0

RRT=renal replacement therapy

incidence of 28.3%.

Characteristics of patients with AKI are shown in **Table 1**. There were 110 (62.1%) males and 67 (37.9%) females, a 2:1 ratio. Ages ranged from 7 days to 16 years (mean 79 months). Based on pRIFLE criteria, there were 65 (36.7%) patients in the risk stage, 56 (31.6%) in the injury stage, and 56 (31.6%) in the failure stage. According to standard management for acute conditions, 12 (6.8%) patients required RRT (including 8 with hemodialysis and 4 with acute peritoneal dialysis). Twenty-nine (16.4%) patients were admitted to the PICU with various indications. The mortality rate of AKI was in the range of 13.5-28.3%, with an average mortality rate of 20.9% for the three and a half-year study period.

The AKI etiologies in this review were intrinsic renal disease in 89 (50.3%) patients, pre-renal injury in 78 (44.1%) patients, and post-renal AKI in 10 (5.6%) patients. The most common etiology of AKI was acute glomerulonephritis (55 subjects; 31.1%), followed by multiple organ dysfunction (24 subjects; 13.6%), dehydration (23 subjects; 13.0%), hypoalbuminemia (20 subjects; 11.3%), heart failure (11 subjects; 6.2%), and nephrotoxic agents (12 subjects; 6.8%). The relationships among specific AKI etiologies, stages of AKI and patient outcomes are shown in **Table 2**.

The most common etiology of intrinsic renal disease was glomerulonephritis, especially acute post-infectious glomerulonephritis, post-streptococcal glomerulonephritis (APSGN) and some cases of rapid progressive glomerulonephritis (RPGN). Other causes of intrinsic renal diseases were included tumor lysis syndrome (3 cases; 1.7%) and hemolytic uremic syndrome (2 cases; 1,1%).

Dehydration as a part of pre-renal condition was usually caused by diarrhea, Hirschprung's disease, or tubulopathy with polyuria. Multiple organ dysfunction (MOD) was associated with hemodynamic instability that impaired renal perfusion. Hypoalbuminemia was commonly found in patients with nephrotic syndrome, sepsis, or burn trauma. In addition, plasma leakage in dengue shock syndrome (DSS) was found in 2 (1.1%) cases.

In our review, there were a few cases of post-renal AKI (obstructive uropathy), such as congenital anomaly of the kidney and urinary tract (CAKUT), stones in the kidney or urinary tract, vesico-uretheral reflux (VUR), jengkolic intoxication (jengkolic acid crystallization), and mechanical obstruction of the urinary tract caused by an intra-abdominal mass.

Glomerulonephritis was the most common underlying disease of AKI in this review, but it had a relatively low mortality rate. The mortality rate was relatively higher in the pre-renal group, especially in cases with MOD.

Table 2. Relationships between diagnosis etiology, degree of AKI and outcomes

Diagnosis	n	%	Risk (n=56)		Injury (n=56)		Failure (n=65)	
			Survived	Died	Survived	Died	Survived	Died
Pre-renal conditions (n=78)								
Multiple organ dysfunction	24	13.6	1	3	3	6	4	7
Dehydration	23	13.0	8	0	6	0	5	4
Hypoalbuminemia	20	11.3	4	0	6	1	7	2
Heart failure	11	6.2	3	1	4	1	1	1
Plasma leakage (DSS)	2	1.1	0	0	2	0	0	0
Renal (intrinsic) (n=89)								
Glomerulonephritis	55	31.1	22	0	12	2	16	3
Urinary tract infection	13	7.3	5	0	2	2	4	0
Nephrotoxic agents	12	6.8	5	0	4	0	3	0
Tumor lysis syndrome	3	1.7	0	0	3	0	0	0
Hemolytic uremic syndrome	2	1.1	0	1	0	0	1	0
Post-renal conditions (n=10)								
Nephrolithiasis/ urolithiasis	3	1.7	0	0	0	0	3	0
Intra-abdominal mass	3	1.7	0	0	0	0	0	3
Vesico-uretheral reflux	2	1.1	2	0	0	0	0	0
Congenital anomaly	2	1.1	0	0	1	0	1	0
Jengkolic acid crystals	2	1.1	1	0	1	0	0	0

Table 3. Association of AKI outcomes with various factors (N=177)

Variables	N	Died (n=37)		Survived (n=140)		P value	OR (95% CI)
		N	%	N	%		
Gender							
Male	110	20	18.2	90	81.8	>0.05	
Female	67	17	25.4	50	74.6		
Age							
< 5 years	59	22	37.3	37	62.7	0.0001	4.083 (1.917 to 8.697)
≥ 5 years	118	15	12.7	103	87.3		
Mean age (SD), months	177	47.7 (51.74)		86.9 (51.29)		0.0001	
Stage of AKI							
Failure	65	20	30.8	45	69.2	0.014	2.484 (1.188 to 5.192)
Non-failure	112	17	15.2	95	84.8		
Etiology of AKI							
Intrinsic (renal)	89	9	10.1	80	89.9	0.0001	0.241 (0.106 to 0.549)
Non-renal origin (pre- & post-renal)	88	28	31.8	60	68.2		
Need of intensive care unit							
PICU	29	16	55.2	13	44.8	0.0001	7.443 (3.133 to 17.683)
Non-PICU	148	21	14.2	127	85.8		
Management							
RRT	12	5	41.7	7	58.3	0.067	2.969 (0.885 to 9.963)
Supportive	165	32	19.4	133	80.6		

RRT=renal replacement therapy

Bivariate analysis between clinical variables and outcomes is shown in **Table 3**. The mean age of those who died [47.7 (SD 51.74) months] was significantly younger than those who survived [mean 86.9 (SD 51.29) months]; (P=0.0001). There was not a significant relationship between gender and outcomes.

Significant relationships were also observed between AKI stage, AKI etiology, PICU admission, and outcomes. Twice as many patients in the AKI failure stage died than in the non-failure group (P=0.014). This observation suggests that the pRIFLE criteria used in this study was suitable to determine prognosis. Patients with intrinsic renal diseases had lower mortality rate than patients with non-renal origins (pre- or post-renal) (P=0.0001). Also, patients admitted to the PICU had a higher mortality rate than non-PICU patients (P=0.0001). However, more patients who underwent RRT died than non-RRT patients, but this relationship was not statistically significant (P=0.067).

Outcomes of AKI patients in the acute phase were 140 survived and 37 died. Of the surviving group, 10 had prolonged diseases consisting of 4 patients with loss of renal function (>4 weeks) and 6 patients with chronic kidney disease (CKD) (>3 months). At the time of this sudy report, 3 patients in the loss stage died, 1 patient in the loss stage was in follow up, 3 patients with CKD V had ongoing continuous ambulatory peritoneal dialysis (CAPD), 1 patient with CKD III was in follow up, and 2 patients were lost to follow up.

Discussion

There was a trend of increasing numbers of nephrology patients in our study period, especially in 2012 and the 1st semester of 2013. Proportions between gender, age and etiology of patients were similar each year. Acute kidney injury remains to comprise a large proportion of our nephrology patients each year, with a range of 22.6%-33.3% (mean 28.3%). The prevalence rate of AKI was 3.9/1,000 admissions in the US and ranged from 0.5-9.9/1,000 admissions in Thailand.[10,11]

Acute kidney injury is characterized by a reversible increase in the blood concentration of creatinine and nitrogenous waste products, as well as the inability of the kidney to appropriately regulate fluid and electrolyte homeostasis. There are many causes of AKI and many diseases can affect the kidney

in a variety of ways.

Based on the etiology, there are three types of AKI: pre-renal injury (pre-renal AKI), intrinsic renal diseases, and obstructive uropathy (post-renal AKI).[8,9] Some causes occur more commonly in certain age groups, for example, renal vein thrombosis occurs more commonly in neonates, whereas rapid progressive glomerulonephritis (RPGN) generally occurs in older children and adolescents.[8]

In pre-renal AKI, the kidney is intrinsically normal and renal function promptly returns to normal with the restoration of adequate renal perfusion. In renal AKI, the kidney has sustained intrinsic injury which requires repair and recovery before renal function returns to normal.[8,9,12] Some studies reported that recent AKI conditions were more commonly due to secondary AKI (non-renal origin), caused by systemic illnesses or adverse effects of their treatment.[11,13]

Intrinsic renal disease may be caused by hypoxic/ischemic AKI due to prolonged pre-renal injury, nephrotoxic agents (mostly aminoglycoside antibiotics, intravascular contrast media, chemotherapeutic agents, acetaminophen or other less common medications), uric acid nephropathy, tumor lysis syndrome, acute interstitial nephritis, rapidly progressive glomerulonephritis, as well as vascular insults (cortical necrosis, hemolytic uremic syndrome/HUS).[8,9] Measurements to differentiate pre-renal injury from intrinsic AKI include urine osmolality, urine sodium concentration, and fractional excretion of sodium. However those measurements were not practical especially in acute condition. In this study, pre-renal injury as an AKI etiology based on the clinical diagnosis which has been well understood can impair renal perfusion, such as dehydration, septic shock or heart failure.

For the full 3 ½ years of this study, the most common AKI etiology was intrinsic renal diseases in 89 (50.3%) patients. Only in 2012 was pre-renal injury more common. The most common conditions underlying intrinsic renal disease were acute glomerulonephritis (especially APSGN), urinary tract infections, and nephrotoxic agents (especially chemotherapeutic agents), while less common conditions were HUS and tumor lysis syndrome. Some studies also reported glomerular diseases to be the most common etiology,[10,14,15] but others reported sepsis to be the most common.[11,16,17]

Nephrotoxic agents are suspected to be the etiology of intrinsic AKI if no other mechanisms can be identified and patients are at risk due to nephrotoxic treatments, such as cytotoxic agents or intravenous antibiotics (aminoglycosides). In this review, nephrotoxic agents involved included chemotherapeutic agents and amikacin. Hemolytic uremic syndrome is a syndrome consisting of hemolytic anemia, thrombocytopenia, and renal failure. There were two cases of HUS, one with bloody diarrhea (HUS+) and one with another infection (HUS-). In some regions of the world HUS is an important cause of AKI in children.[18]

Several studies in developing countries reported that the majority of causes of AKI in children are secondary to volume-responsive mechanisms, including acute diarrheal losses and sepsis.[19-21] In our study, pre-renal injury was found in 78 (44.1%) patients (due to MOD and dehydration caused by gastrointestinal losses, hypoalbuminemia in nephrotic syndrome, and heart failure caused by underlying cardiac diseases). The most common causes of MOD were sepsis (especially in PICU patients) and dehydration (gastrointestinal losses). In the US, the most common factor associated with AKI is shock (OR 2.15).[3]

Pre-renal injury as the AKI etiology occurs when blood flow to the kidney is reduced due to true intravascular volume contraction (hemorrhage, dehydration due to gastrointestinal losses, renal salt-wasting or central/nephrogenic diabetes insipidus, diseases with third space losses, such as sepsis or nephrotic syndrome or capillary leak syndrome) or decreased effective blood volume (congestive heart failure, cardiac tamponade, or hepatorenal syndrome). Pre-renal injury is reversible once the blood volume and hemodynamic conditions have been restored to normal. However, prolonged injury can cause intrinsic AKI due to hypoxic/ischemic acute tubular necrosis.[8,12]

Integrated care of patients with other disciplines was usually associated with multiple organ involvement (non-renal origin or secondary AKI) and less common intrinsic renal diseases. Such intrinsic renal diseases included patients exposed to nephrotoxic agents as well as those with atypical HUS (due to infections) and tumor lysis syndrome.

Management of AKI includes non-dialytic

or supportive therapy as well as dialytic therapy. Supportive therapies include fluid and electrolyte management, medications, and avoid any drug or procedure that harmful to kidney. Dialytic therapy (RRT) includes hemodialysis or peritoneal dialysis, which have indications such as hyperkalemia, hyperphosphatemia, especially with hypocalcaemia, severe metabolic acidosis, or fluid overload, and symptoms of uremia.[9] In this study, RRT was performed in overall mean 6.8% (Table 1), less than reported in Lithuania and Thailand, with 28% and 17.3% respectively.[11,14] The most common indications of RRT were uremic toxin (ureum level >200mg/dL) and severe metabolic acidosis (HCO_3 level <12 mmol/L). The overall mortality of patients with RRT in acute conditions was 5/12 (41.7%). This result was similar to that of Pundziene *et al.*[14] who reported a mortality of RRT in range of 12.5-75%. However, the US mortality rate was lower (27.1%).[10]

In this review RRT was not a significant indicator of outcome, but patients who underwent RRT had higher mortality than non-RRT patients, with OR 2.969 (95% CI 0.885 to 9.963). Renal replacement therapy (RRT) as an emergency procedure had to be done when indicated, because more than half of patients undergoing RRT (7/12) survived in acute conditions.

Required PICU admission in the four time periods ranged from 5-25% (overall mean 16.4%). This result was lower than that of Pundziene *et al.*, who reported that 69.3% needed the PICU due to serious conditions.[14] This difference might be due to the majority of underlying diseases, sepsis, in their study compared to acute glomerulonephritis being more common in our study. The most common indications for PICU admission in our patients were hemodynamic instability and respiratory failure caused by MOD. Patients admitted to the PICU were at higher risk of death than non-PICU patients with OR 7.443 (95% CI 3.133 to 17.683).

The mortality rate of AKI in the four time periods ranged between 13.5-28.3% (overall mean 20.9%). Other studies reported different mortality rates, lower in the US (15.3%),[7] higher in Thailand (41.5%),[11] and relatively similar in Lithuania (24%).[14] In our study, mortality was significantly higher in patients who were of younger age (< 5 years), had a non-renal origin, in the AKI failure stage of pRIFLE, and/or admitted to the PICU. In comparison, a US report found that mortality was higher in younger age (neonates), ICU patients and dialytic patients.[10]

Among those who survived, full recovery after AKI was documented in 130 patients (73.5%). This relatively high complete recovery from AKI may be due to having acute glomerulonephritis as the most common etiology, a condition known to have good prognoses. Other studies similarly reported full recovery of their patients in a range of 60-83.7%.[11,14] Four of our patients continued to have loss of renal function (>4 weeks) and 6 patients remained with chronic kidney diseases.

In conclusion, AKI is found in one-third of nephrology patients. The most common AKI etiology in our population is acute glomerulonephritis. One-fifth of patients with AKI die. Outcomes of patients was significantly influenced by age, stage of AKI, etiology of AKI, and PICU admission. Based on these findings, patients with AKI who are at a younger age, in the failure stage, have a non-renal origin, or have an indication for PICU admission, should be monitored carefully. Recognizing risk factors and detecting AKI in early stages might improve outcomes.

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