Kidney is essential in the urinary system and functions in maintaining electrolyte and acid-base balance as well as removing excess organic molecules and potentially metabolic toxic end products from the blood. An abrupt decline in renal function is defined as acute kidney injury (AKI). AKI includes multiple abnormalities such as elevated serum creatinine (SCr) and blood urea nitrogen (BUN), oliguria or anuria, electrolyte imbalance, acidosis, and difficulties with fluid management[1].

To well recognize AKI, a standardized definition based on the changes in SCr and urine output, called Kidney Disease Improving Global Outcomes (KDIGO) criteria, was proposed in 2012[2, 3]. The diverse etiologies of AKI can be categorized into pre-renal, intrinsic renal or post-renal causes[4, 5]. Pre-renal AKI refers to a decline in perfusion to the kidney which is due to hypovolemia, ischemia, peripheral vascular dilatation or renal vascular contraction[6], intrinsic renal AKI is the consequence of renal parenchyma defects, and post-renal AKI is caused by obstruction of the urinary tract such as urolithiasis and congenital urinary tract malformations[7].

Epidemiological studies of pediatric AKI patients have been performed in multiple countries and regions[8-12]. AKI is a common risk factor for hospitalized children, especially for critically ill children, and its incidence ranges from 0.3% to 82%[13]. Studies showed that AKI had multifactorial etiologies, instead of primary renal diseases[14, 15]. Although these advances in epidemiology of AKI in children have been achieved, the epidemiological report in China remains inadequately described.

Recently, a multicenter study investigated the morbidity, etiology and outcome of AKI in Chinese children who were hospitalized in 2008[16], however, there is still lack of a more recent report on the overall incidence, risk factors, outcome and prognostic factors of AKI. Hence, in this study, we firstly reported the morbidity of AKI among inpatients in our pediatric hospital from 2003 to 2013 and then retrospectively investigated 205 patients with AKI during this period. The main objectives were to
screen the risk factors associated with AKI stage and prognostic factors for renal recovery, which could be useful in early detection and mortality prevention in children with established AKI.

1 MATERIALS AND METHODS

1.1 Patients and Study Design

Firstly, the annual morbidity of AKI among child inpatients in our pediatric hospital was investigated from 2003 to 2013. All AKI patients aged ≤18 years were enrolled in this study, except those who met one of the following exclusion criteria: patients who had undergone only one SCr measurement; patients who had a length of hospital stay less than one week; patients with chronic renal insufficiency; patients who had incomplete clinical data; or patients who were included in other clinical studies. The demographic data, clinical data (such as clinical symptom, stage of AKI, classification of AKI, and length of hospital stay), and outcomes of the enrolled AKI patients were then collected. Also, laboratory tests including routine blood test, blood gas analysis, routine urine test at admission, as well as serum creatinine (SCR) and blood urine nitrogen (BUN) at admission and discharge were reviewed.

This retrospective study was approved by the ethics committee of our hospital.

1.2 Definition, Staging and Classification Criteria of AKI

In this study, we firstly searched patients who were diagnosed and recorded as acute renal insufficiency, acute kidney injury, acute renal failure, renal insufficiency, kidney injury or renal failure in the electronic medical records system of our hospital, and then defined AKI according to the KDIGO criteria\textsuperscript{[2, 14]}. In detail, children were considered to have AKI, if a SCr increase of more than 0.3 mg/dL within 48 h or 1.5 fold of the baseline level, an estimated glomerular filtration rate ≤90 mL/min\textsuperscript{-1}.73 m\textsuperscript{2}, or a urine output of <0.5 mL·kg\textsuperscript{-1}·h\textsuperscript{-1} for 6 h or above was detected. AKI was staged according to KDIGO criteria as follows: stage 1, SCr increase of more than 0.3 mg/dL in 48 h or 1.5–1.9 fold of the baseline level, or urine output decrease to ≤0.5 mL·kg\textsuperscript{-1}·h\textsuperscript{-1} which lasts for 6–12 h; stage II, SCr increase of 2.0–2.9 times, or urine output decrease to ≤0.5 mL·kg\textsuperscript{-1}·h\textsuperscript{-1} which lasts for 12 h; and stage III, SCr increase >4.0 mg/dL or ≥3.0 fold of the baseline level, estimated creatinine clearance <35 mL·min\textsuperscript{-1}.1.73 m\textsuperscript{2}, or urine output decrease to <0.5 mL·kg\textsuperscript{-1}·h\textsuperscript{-1} for 24 h or 0.3 mL·kg\textsuperscript{-1}·h\textsuperscript{-1} for 12 h. Besides, AKI etiologies were analyzed and divided to pre-renal, intrinsic renal or post-renal causes\textsuperscript{[17]}

1.3 Laboratory Tests

Routine blood test, blood gas analysis, routine urine test were performed at admission. Hematological parameters, including white blood cells (WBCs), red blood cells (RBCs), hemoglobin (HGB), platelets (PLTs) and hematocrit (HCT) were determined by Sysmex KX-21 fully automated Hematology Analyzer (East Asia Co., Japan). Parameters in routine urine test including urine blood (BLD), urine protein (PRO) and leukocytes (LEUs) were tested with Sysmex UF-50 automated urinalysis analyzer (East Asia Co., Japan). The data of blood pH, partial pressure of carbon dioxide (\(\text{PaCO}_2\)), partial pressure of oxygen (\(\text{PaO}_2\)), oxygen saturation (\(\text{SO}_2\)), base excess (BE) and bicarbonate (\(\text{HCO}_3^-\)) were collected by ABL800 FLEX arterial blood gas analyzer (Radiometer Medical ApS, Bronshøj, Denmark). Levels of SCR and BUN were measured using an automatic biochemistry analyzer (Modular DPP System; Roche Modular DPP, Hitachi Ltd., Japan).

1.4 Treatments and Outcomes of AKI

AKI patients were treated by conservative therapy, surgical therapy and/or renal replacement therapy. Treatment method was determined by two experienced doctors according to the causes and severity of AKI. The outcomes were classified as renal recovery and exacerbation. Renal recovery included complete recovery which was defined as a SCr decrease to its baseline level and partial recovery. No renal recovery after treatments, discharge without receiving any treatment and death were classed as exacerbation.

1.5 Statistical Analysis

Quantitative data were expressed as \(x \pm s\) if they met normal distribution or median and range if not. Categorical data were shown as frequencies and percentages. Differences in morbidity between boy and girl inpatients were analyzed by using chi-square test. Binomial logistic regression was conducted to screen risk factors which were significantly associated with AKI stage III and analyze the prognostic factors for renal recovery in child inpatients with AKI. When more than one significant variable were obtained by the univariate analysis, multivariate logistic regression was performed to identify independent factors using a forward conditional strategy. All statistical analyses in this study were performed using SPSS version 19.0 (SPSS Inc., USA). \(P<0.05\) was set as the cut-off for statistical significance. Odd ratio (OR) and 95% confidence interval (CI) were also revealed for logistic regression analysis.

2 RESULTS

2.1 Morbidity of AKI

A total of 65,237 children were admitted to our pediatric hospital from 2003 to 2013. The annual morbidities of AKI in this study cohort are shown in table 1. A decline trend on annual morbidity had been observed since 2012. Except 2011 (\(P=0.004\)), no significant differences in annual morbidity were found between boy and girl inpatients, as well as the overall morbidity during the period of 2003–2013 (\(P=0.05\)).