Causes of Acute Kidney Injury in Egyptian Children

Thesis

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من أنوار الوحي العظيم

قال تعالى: {الْحَمْدُ لِلَّهِ الَّذِي هَدَانَا لِهَذَا وَمَا كُنَّا لِنَهْتَدِيَ لَوْلَا أَنْ هَدَانَا اللَّهُ} [الأعراف: ٤٣]

وقال جل جلاله: {سُبْحَانَكَ لَا عِلْمَ لَنَا إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ الْعَلِيمُ الْحَكِيمُ} [البقرة: ٣٢]

وقال سبحانه: {رَبِّ زِدْنِي عِلْمًا} [طه: ١١٤]

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LIST OF ABBREVIATION

AKI: Acute Kidney Injury.

ANA: Anti Nuclear Antibody**.**

C3: Complement 3

C4: Complement 4

CRF: Chronic Renal Failure**.**

ESRD: End Stage Renal Disease.

FSGS: Focal Segmental GlomuruloSclerosis.

HSP: Henoch-Schonlein Purpura.

HTN: Hypertension.

HUS: Hemolytic Uremic Syndrome.

NS: Nephrotic Syndrome.

PKD: Polycystic Kidney disease.

PSGN: Post Streptococcal Glumerulonephritis.

RIFLE: Risk – Injury – Failure – Loss – End stage.

RPGN: Rapidly Progressive Glumerulonephritis.

RTA: Renal tubular Acidosis.

CKD: Chronic Kidney Disease.

SLE: Systemic Lupus Erythematosus.

SRNS: Steroid Resistant Nephrotic Syndrome.

UTI: Urinary Tract Infection.

VUR: VesicoUreteric Reflux.

INTRODUCTION

Acute kidney injury (AKI) is a term proposed to reflect the entire spectrum of acute renal failure (ARF), a complex disorder that occurs in a wide variety of settings with clinical manifestations ranging from minimal elevation in serum creatinine to anuric renal failure ***(Mehta et al., 2007)***.

The epidemiologic importance of AKI as a public health problem is underscored by evidence showing that even a small reduction in the renal function of hospitalized adult and pediatric patients (equivalent to 0.3 mg/dl increase in serum creatinine) is a risk factor for morbidity and mortality ***(Price et al., 2008)***.

The etiology of AKI has changed over the last 10–20 years from primary renal disease (e.g., hemolytic uremic syndrome, glomerulonephritis) to the renal complications of systemic illness or its treatment (e.g., postoperative cardiac surgery and oncologic diseases). ***(Akcan-Arikan et al., 2007)***. Some causes of AKI, such as rapidly progressive glomerulonephritis (RPGN), may present as AKI but rapidly evolve into chronic kidney disease (CKD). Several renal diseases, such as the hemolytic–uremic syndrome (HUS), Henoch–Schönlein purpura, and obstructive uropathy with associated renal dysplasia, may present as AKI with improvement of renal function to normal or near-normal levels, but the child's renal function may slowly deteriorate, leading to CKD several months to years later ***(Andreoli, 2009)***.

As long as AKI remains underreported, it will not be addressed properly. In developing countries, the most common causes of AKI are frequently associated with volume-responsive "prerenal", infectious, or

toxic mechanisms; thus, inexpensive, simple interventions such as education on oral rehydration, or management of infection may result in a dramatic reduction in the incidence and severity of AKI ***(Cerdá et al., 2008)***.

OJECTIVES

The aim of this study was to survey the causes of AKI in pediatric nephrology center of the Cairo University Hospital (Abo-Elriesh) to come up with understanding of the etiology as well as a staging of the patients in this center.

REVIEW OF LITERATURE

Chapter One

ACUTE KIDNEY INJURY

DEFINITION

Historically, a substantial rise in SCr and a drop in urine output have been used to determine if a child has AKI. Prior to the 2004, over 30 definitions of AKI existed in the literature which made comparison between studies very difficult. In 2004, the ADKI group proposed the RIFLE (Risk, Injury, Failure, Loss and End-Stage) classification definition of AKI ***(Bellomo et al., 2004).***

The first 3 categories (Risk, Injury and Failure) staged the degree of AKI based on whether the amplitude of SCr rise (or decrease in estimated GFR) and/or a drop in urine output. The last two categories (Loss and End-stage) defined temporary or permanent loss of kidney function after AKI. In 2007, a similar definition was proposed for pediatric patients and has been used to describe several cohorts ***(Akcan-Arikan et al., 2007 & Zappitelli et al., 2008).*** The RIFLE definition was updated in 2007 by the Acute Kidney Injury Network ***(Mehta et al., 2008)***, by many of the same experts who proposed RIFLE.

The AKIN definition is similar to the first 3 stages of the RIFLE classification with a couple of changes. Recently, the Kidney Disease Improving Global Outcomes (KDIGO) has brought together international experts from many different specialties to produce a definition and staging system which will harmonize these recent definitions. It is expected that this definition will be accepted globally in 2011.

The adjustments in these definitions over time should be considered gradual evidence-based improvements. These SCr-based classification definitions of AKI have provided great insights; however, when using these definitions, it is important to acknowledge the shortcoming and limitations of using SCr-based AKI definitions ***(Askenazi, 2011)***.

The use of changes in function markers such as SCr to define AKI is not ideal as SCr concentrations may not change until 25–50% of the kidney function has already been lost and thus it may take days after an injury before a significant rise in SCr is seen ***(Brion, 1986).*** At lower GFR, SCr will overestimate renal function due to tubular secretion of creatinine ***(Brion, 1986).*** SCr varies by muscle mass, hydration status, sex, age and gender and method of measurement ***(Lolekha et al., 2001 & Rajs & Mayer, 1992).*** Once a patient receives dialysis, SCr can no longer be used to assess kidney function since SCr is easily dialyzed. In neonates, SCr in the first few days of life reflects mother's; thereafter, the SCr improve at different rates depending on the degree of gestational age ***(Gallini et al., 2000).***

In addition, SCr based definitions fails to delineate the severity, timing and cause of the injury. Despite the above concerns about these AKI definitions, these new SCr-based classification definitions of AKI have allowed for valuable comparisons among different studies, which have shed new light about the epidemiology of AKI. Using these definitions, we have learned that AKI is an independent predictor of mortality in critically ill neonates ***(Askenazi et al., 2009)***, children ***(Akcan-Arikan et al., 2007 & Zappitelli et al., 2008)***, and adults ***(Ricci et***