Acute kidney injury (AKI) is an important cause of morbidity and mortality in critically ill children. Though most pediatricians treating critically ill children are aware of the potential for renal injury in these patients, traditional markers of renal injury focus primarily on the serum creatinine, an inherently unreliable indicator. Even modest elevations in serum creatinine have been shown to be a risk factor for mortality. In addition, easy-to-perform bedside calculated estimation of creatinine clearance (eCCr) is not recommended in children with rapidly changing renal function. To add to this confusion, there are more than 30 published definitions of AKI. It is important to identify renal injury early since it is associated with high mortality, as high as 60% in critically ill children. Even those patients who survive have a significant risk of persistent renal dysfunction.

In an effort to standardize definitions, AKI has replaced the term acute renal failure and two similar definitions have been described in recent times. These are the pRIFLE (pediatric Risk, Injury, Failure, Loss, and End-stage renal disease) criteria based on a modification of the adult RIFLE criteria developed by the Acute Dialysis Quality Initiative Group and the Acute Kidney Injury Network (AKIN) scoring systems. Both are based upon serum creatinine and urine output.

Since their publication, there are increasing reports of the epidemiology of AKI in children using these classifications. However, most studies have been from developed countries and many are retrospective. Various studies have shown that AKI in developed countries is predominantly due to surgery, malignancies, and nephrotoxic drugs, whereas in developing countries, the causes are more likely to be severe infection, diarrheal dehydration, hemolytic-uremic syndrome, and post-infectious glomerulonephritis. A recent prospective study from northern India showed that using AKIN criteria, the incidence of AKI in hospitalized children was 36% in critically ill and 9% in non-critically ill children. The same study also reported that younger age, shock, sepsis, and the need for mechanical ventilation were independent risk factors for developing AKI, and that patients with AKI had a longer hospital stay and higher mortality. Shock was an independent risk factor for mortality in this study.

In this issue of the journal, Krishnamurthy and colleagues present the results of a prospective observational study of AKI in children admitted to a PICU in southern India. Using AKIN criteria, they showed that the incidence of AKI amongst 215 children admitted to the PICU was 25%. Like other reports from developing countries, the major cause of AKI was infection (63%). Interestingly, causes of AKI in developed countries, such as malignancy, postoperative state, and nephrotoxic drugs, were extremely rare. The children with AKI were seriously ill, as evidenced by the high mean PRISM III score (23.4) and the fact that nearly 80% were mechanically ventilated. These data differ from those reported from North India, where only 48% of children with AKI required ventilation – this
may also be due to differences in the availability of resources and institutional practices in the two medical centers. In the present study, mortality in the group with AKI was high (46%). About 28% of children with AKI underwent dialysis – the mortality rates in the dialyzed and non-dialyzed groups were similar. The need for mechanical ventilation was an independent predictor of mortality. It is heartening to note that though the mortality was high, 79% of survivors had complete recovery of renal function. One drawback of the present study is that urine output, an integral part of the definition of AKI, was measured every 6 h – it is not clear whether this was measured with an indwelling bladder catheter (the most accurate method) or by other techniques.

The authors of this study must be congratulated for carrying out this well-designed prospective analysis using standardized criteria. They have added to the growing body of knowledge of AKI amongst seriously children in developing countries. It is evident from this and other studies that the incidence of AKI in critically ill children is high, and this is associated with high mortality. It is no longer enough to look at serum creatinine alone in this population and using standard definitions of AKI will allow clinicians to identify renal injury early. One drawback with these definitions is that they rely on serum creatinine/eCCr and urine output and are therefore only surrogate markers for renal injury – various biomarkers for renal injury, such as serum cystatin C, interleukin-18, and urinary neutrophil gelatinase-associated lipocalin (NGAL) levels, are under investigation.[5] Until better markers are available, clinical criteria remain the best available options. Since numbers in an individual center are likely to be small, there is an urgent need for larger, multi-center studies looking at the epidemiology of AKI in children using standardized definitions and severity of illness scoring.

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