EDITORIAL

Acute Peritoneal Dialysis in Neonates with Acute Kidney Injury

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Acute kidney injury (AKI) is defined as sudden decrease in glomerular filtration rate leading to fluid and electrolyte imbalance, disturbed acid-base homeostasis and retention of nitrogenous waste products. It affects about 5% of patients admitted to hospitals and 30% of cases in intensive care units [1]. In the recent past, two large cohort studies, Assessment of worldwide acute kidney epidemiology in neonates (AWAKEN) [2] and Assessment of worldwide acute kidney injury, renal angina, and epidemiology (AWARE) [3] have provided an in depth spectrum of AKI in neonates, children and young adults. In neonates, the incidence of AKI was found to be 30%, which varied according to gestational age such as 47.9% in gestation of ≥22 to <29 weeks, 18.3% between ≥29 to <36 weeks and 36.7% in ≥36 weeks [2]. The overall incidence of AKI in children has been reported to be 27%; with severe AKI in 11.6% of children in intensive care settings [3]. As such there are several definitions to define AKI based on rise in the serum creatinine level, decrease in urine output and estimated glomerular filtration rate. However, serum creatinine varies with age, muscle mass, nutritional and hydration status. It has major limitations in newborns because of reflection of maternal creatinine in initial 48-72 h after birth, varying degree of reabsorption from proximal tubules, lower glomerular filtration rates and maturation differences based on gestational age. Modified kidney diseases: Improving global outcomes (KDIGO) criteria can be applied to define AKI in neonates [4]. This classification defines AKI in different stages based on absolute rise in serum creatinine from a previous trough level and decrease in urine output or anuria over time.

Regarding etiologies of AKI, hypovolemia following acute gastroenteritis, sepsis, hemolytic uremic syndrome and malaria are common in older children in developing countries [5], while ischemic/hypoxic and nephrotoxic injury to preterm/term neonates, sepsis and post-cardiac surgery are predominant etiologies in developed countries [6].

The neonates may present with lethargy, fever/ low body temperature, decreased urine output/ anuria, vomiting, hypotension, seizures, and palpable kidneys and bladder, if there is obstructive uropathy. Initial investigations include hemogram, complete blood count, blood culture, C-reactive protein, renal function test, arterial blood gas analysis, urine microscopy and culture study, and ultrasonography kidney, ureter and bladder to detect underlying congenital malformations. Voiding cystourethrogram can be performed earlier (within 24-72 h of life) in patients with suspected lower urinary tract obstruction.

Supportive therapy is in the form of maintenance of fluid and electrolyte balance, antibiotics in modified doses for treatment of sepsis, use of vasopressor agents for hypotension, and ventilatory support, if required. Oliguria and fluid balance are important parameters in critically ill patients. Cases of oliguric AKI have a three-fold increased risk of undergoing renal replacement therapy as compared to non-oliguric AKI. The renal replacement therapies available for neonatal AKI are peritoneal dialysis (PD), hemodialysis and continuous renal replacement therapy. Choice of therapy depends upon the technical expertise, vascular access and availability of machines. The option of PD is often the only modality available in developing countries, which can be instituted at the earliest. The type of catheter can be flexible (Tenckhoff double cuffed straight/swan neck or Cook PD soft catheter) or rigid straight with stylet or improvised PD catheters (pig-tail, angiocath, intercostal drainage tube). However, flexible catheter is preferred because of better inflow, outflow and lesser chances of leakage, peritonitis and perforation.

The article by Okan, *et al.* [7] in this issue of *Indian Pediatrics* reports on the use of acute PD with multifunctional flexible catheter in the treatment of very low birth weight (VLBW) and extremely low birth weight (ELBW) neonates. In this small observational study, the etiologies of AKI were patent ductus arteriosus, necrotizing enterocolitis, sepsis, asphyxia and hydrops

fetalis. About 2.8% of neonates required PD and the mortality was high (81%), which could be because of lower gestation and birthweight. Kara, *et al.* [8] also found high mortality of 77% in their study on acute PD in neonatal AKI. The contributors for mortality in AKI patients are multiorgan failure, sepsis, AKI stage 2 and 3, presence of fluid overload and need for ventilatory support [3,8,9,10]. Fluid balance has been found to be closely associated with outcome and negative fluid balance at post-natal day seven in the hospital setting was associated with a lesser risk of need for mechanical ventilation in near-term/term neonates [9]. In a large cohort of AWAKEN study, significant contributors for mortality were AKI, and longer duration of hospital stay [2]. As such, AKI is an independent risk factor for mortality during hospitalization.

The initiation of PD in VLBW and ELBW is the preferred dialysis modality to treat AKI. Since mortality in neonatal AKI is still very high, early institution of PD should be undertaken as a life-saving procedure.

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