EDITORIAL

NEONATAL RENAL FAILURE

In the last decade, published data on neonatal renal failure has increased and various authors report an incidence of acute renal failure (ARF) in Neonatal Intensive Care Units (NICU) ranging from 1-8%(1-5). This is attributed to the following factors: (*i*) Prolonged survival of seriously ill newborns with improved resuscitation and ventilatory support; (*ii*) Increased awareness of renal failure and importance of monitoring urine output and serial measurements of blood urea and serum creatinine in neonates; (*iii*) Antenatal diagnosis of renal anomalies by ultrasonography, many of which lead to renal failure in infancy; (*iv*) Use of nephrotoxic drugs and increased incidence of Gram negative sepsis; (*v*) Increased survival of premature neonates at a higher risk of renal failure due to physiological immaturity of renal function, sudden adaptation to extrauterine life and exogenous stress factors which break down the homeostasis of body function regulated by the placenta in fetal life.

Published data on neonatal renal failure in India is scanty(6). Hence it is mandatory for all pediatricians treating neonates to document the time of first urination and whether the urine output during the first 48 hours was adequate. In a suspected case, a simple blood urea nitrogen (BUN) and serum creatinine level should be estimated to detect renal failure. This entity in a newborn is characterized by reduction in urine output (>1 ml/kg/h) and increase in BUN (>20 mg/dl and S. creatinine (>1.5 mg/dl) associated with hyperkalemia (>7.5 mEq/L), and acidosis (arterial pH <7.25).

The diagnosis of neonatal renal failure is not straight forward in the first few days because of physiologic variations in the two important criteria for the diagnosis of renal failure, namely, urine output and BUN or serum creatinine values.

Urine Output: Seven per cent of normal neonates fail to void urine in the first 24 hours of life. In preterms the urine output varies between 0.5 to 2 m1/kg/h in the first 24 hours independent of fluid intake. Urine output may increase three fold without significant change in intake from 2-5 days and subsequently from the sixth day onwards, urine output decreases and this depends on fluid intake(7). Hence total anuria or diminishing urinary output less than 0.5-0.8 ml/kg/h using condom catheter in male neonates or urine collection bags in females is imperative for the diagnosis of oliguric renal failure. A small number of nonoliguric renal failure may still be missed.

Blood Urea Nitrogen: This is influenced by protein intake and a misleading rise in BUN levels in catabolic states such as trauma, sepsis, sequestered blood, is known. The higher anabolic state of a healthy neonate suggests that relatively small increments in blood urea may reflect renal impairment. In a study of ARF in a NICU, 13/24 infants had a plasma urea concentration within normal range at the time of diagnosis(2).

Serum creatinine: Cord blood or serum creatinine levels in first 72 hours, reflect maternal creatinine levels (0.7-1.5 mg/dl) which fall in next few days of life to 0.2-0.4 mg/dl. Hence a serial rise in serum creatinine or failure to fall, in first 7-10 days of life if accompanied by hyperkalemia, acidosis, edema or hypertension(7) is a reliable index of renal impairment in newborns.

A high index of suspicion in a clinical setting which may lead to renal failure should, therefore, be maintained for early diagnosis of renal failure in neonates.

Common situations leading to renal failure in neonates are summarized in *Table I.* It is crucial to differentiate prerenal failure or functional oliguria from intrinsic renal failure in neonates. In early stages, prerenal failure can be prevented from progressing to acute tubular necrosis (or in severe cases cortical or medullary-necrosis) by rapid correction and restoration of renal perfusion (*Fig. 1*). Although urinary indices may be helpful, no single test is pathognomonie(1,4,9). Hence fluid challenge and intravenous furosemide has remained the gold standard for the treatment of prerenal failure and diagnosis of intrinsic renal failure irrespective of etiological cause. This procedure can be easily adopted for secondary level care.

TABLE I —*Clinical Settings for Neonatal Renal Failure in India*

(A)	Antenatal diagnosis of congenital anomaly of kidneys and urinary tract
(B)	At birth
(i)	Asphyxia, respiratory distress, shock, hemorrhage, umbilical artery catheterisation
(ii)	Congenital malformations of urinary tract or gastro-intestinal tract, meningomyelocele, *etc.*
	(Clues: Renal masses, distended bladder, ear anomalies, Prune Belly syndrome, *etc.*)
(C)	2-28 days
	Poor urinary stream, late urination, acute gastroenteritis with dehydration, sepsis, jaundice, drugs (nephrotoxins), shock, hemorrhage, DIC, following cardiac or urologic surgery

Oliguria (post renal cause to be ruled out by clinical examination and urethral catheterisation)

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IV 15 ml/kg 1/5 normal saline in 1 hour

↓ No diuresis within ½ - 1 h

IV Furosemide 2-5 mg/kg

↓ No diuresis within ½ - 1 h

IV Dopamine drip 1-5 μg/kg/min

↓ No diuresis within ½ - 1 h

Peritoneal dialysis (established intrinsic renal failure)

Fig. 1. Algorithm for treatment of oliguria in neonates.

Iatrogenic renal failure in neonates can occur due to use of nephro-toxic agents. Antibiotics such as aminoglycosides and cephalosporins; indomethacin (for pharmacological closure of patent ductus arteriosis in preterms); tolazoline (to reduce pulmonary hypertension); captopril (for maternal hypertension) and contrast dyes (for cardiac catheterisation) can precipitate renal failure in a neonate, more so if prematurity, sepsis or dehydration coexist(10).

Management: Awareness, anticipation, measurement of urine output using condom catheter in male and urine bags in female infants and estimation of BUN, serum creatinine, serum electrolytes and

blood gases is the key to the early diagnosis. The equipment and expertise required for peritoneal dialysis which may be required in few cases is simple and is available in most of the centres with tertiary level care. Rarely exchange transfusion, continuous arteriovenous hemofiltration (AVH) or hemodialysis (AVHD) is required in hemodynamically unstable neonates which is available in very few centres in India.

Outcome: Mortality ranges from 14-75%(1,10) and depends on severity of underlying cause, delay in the diagnosis, and treatment and associated non renal factors such as sepsis and multiple congenital malformations. Non oliguric renal failure has a better outcome(1,4,8).

In our prospective study of 936 sick neonates 30 (3.2%), developed ARF (22 oliguric and 8 non oliguric). Congenital anomalies were found in 10 and in 20 various perinatal factors such as birth asphyxia, sepsis, polycythemia, hyperbilirubinemia, respiratory distress syndrome, congenital heart disease and acute gastroenteritis with dehydration contributed to the renal failure. Mortality was high, *i.e.,* 60% with congenital malformations and 70% with acquired causes; 2/3 dying within 24 hours of presentation due to severe electrolyte disturbances(11).

In conclusion, neonatal renal failure should be considered in an anticipatory manner with careful monitoring of urine output and serial measurements of BUN and S. creatinine in all sick neonates. A simple approach using intravenous fluid administration along with furosemide (if no evidence of fluid overload or obstruction to urinary tract) in oligo-anuric newborns will prevent intrinsic renal failure. Timely peritoneal dialysis if fluid challenge fails is life saving. Poor outcome is associated with severe asphyxia, septicemic shock or severe congenital malformations.

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REFERENCES

1. Stapleton FB, Jones DP, Grees RS. Acute renal failure in neonates: Incidence, etiology and outcome. Pediatr Nephrol 1987, 1: 314-320.
2. Modi N. Treatment of renal failure in neonates. Arch Dis Child 1989, 64: 630-631.
3. Meeks ACG, Sims DG. Treatment of acute renal failure in neonates. Arch Dis Child 1988, 63: 1372-1376.
4. Anand SK. Acute renal failure in neonates Pediatr Clin North Am 1982, 29: 791-800.
5. Norman M. Assadi FK. A prospective study of acute renal failure in the newborn infant. Pediatrics 1979, 63: 475-479.
6. Srivastava RN, Moudgil A, Bagga A. Acute renal failure. In: Pediatric and Neonatal Emergencies. Eds Srivastava RN, Man Mohan, Sachdev HPS, Puri RK. New Delhi, Indian Pediatrics 1990, pp 409-417.
7. Bidiwala KS, Loenz JM, Kleinman L. Renal function correlates of postnatal diuresis in preterm infants. Pediatrics 1988, 82: 50-58.
8. Eunice J. Renal disorders and diseases of the newborn. In: Text Book of Neonatology. Ed Vidyasagar D. New Delhi, Interprint, 1987, pp 277-292.
9. Mathew OP, Jones AS, James E, *et al.* Neonatal renal failure: Usefulness of diagnostic indices. Pediatrics 1980, 65: 57-60.
10. Guignard JP. Renal function in newborn infant. Pediatr, Clin North Am 1982, 29: 777-790.
11. Mehta KP, Ali U, Dixit M, Kothari N, Rao S. Renal disorders of newborns in India (Abstract). Pediatr Nephrology 1989, 3 : 4, C 200.