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Acute renal failure in neonates: incidence, etiology and outcome*

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Abstract. Acute renal failure (ARF) occurs in as many as 8% of neonates admitted to neonatal intensive care units. Most often, ARF is recognized because of oliguria (urinary flow rate <1 ml/kg per hour) although nonoliguric neonatal ARF is being detected with increasing frequency. Among urinary indices utilized to differentiate oliguric neonatal ARF from prerenal oliguria, a fractional excretion of sodium greater than 3% or a renal failure index (RFI) greater than 3 are helpful in confirming ARF. Such indices must be viewed with caution in very premature infants who may have a physiologically high sodium excretion rate and in neonates with the nonoliguric form of ARF. The mortality of oliguric neonatal renal failure may be as high as 60% in medical ARF and even higher in neonates with congenital heart disease, or with anomalies of the genitourinary system. In contrast, nonoliguric renal failure in neonates has an excellent prognosis. Long-term abnormalities in glomerular filtration rate and in renal tubular function are common in survivors of neonatal ARF.

Key words: Acute renal failure – Neonate – Oliguria

Introduction

Aggressive neonatal intensive care has markedly increased the survival of high-risk, premature infants. Babies, who would previously have succumbed to pulmonary immaturity, overwhelming sepsis or complex cardiac malformations, are now frequently the source of consultations by pediatric nephrologists for oliguria, hypertension or fluid and electrolyte disturbances. Often, the distinction between prerenal and renal azotemia in neonates is unclear, the etiology of renal failure is obscure, and the most effective and appropriate management interventions in neonates are controversial. As dialysis and ultrafiltration therapies have become technically feasible for newborns, the necessity for early recognition and prognostic indicators for acute renal failure (ARF) in neonates has become essential. This discussion will review the definition, incidence, etiology and outcome of ARF in the 1st month of life.

Definition

In a broad sense, ARF may be defined as an abrupt decrease in the glomerular filtration rate which prevents the kidneys from regulating the composition of the extracellular fluid compartment. However, the neonate presents a unique diagnostic dilemma, as it is not until birth that the kidneys assume a primary regulatory role. Therefore, when impaired renal function is encountered in the early neonatal period, one must consider the possibility that it results from renal dysgenesis or intrauterine obstructive uropathy, in which case azotemia may represent chronic renal failure and the prognosis would be extremely unfavorable. Alternatively, ARF may develop from insults at or following birth in otherwise normal kidneys. This discussion will concentrate on acute renal impairment resulting from extrauterine insults.

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Traditionally, neonatal renal failure has come to clinical attention because of decreased urinary output. Indeed, many excellent reviews of ARF in the neonatal period have focused exclusively on the oliguric form of renal failure [1–5]. A consensus definition of oliguric neonatal ARF is a urine flow rate of less than 1 ml/kg per hour which fails to respond to a fluid challenge and is associated with a serum creatinine greater than 1.5 mg/dl [6–9]. As in older individuals, oliguric ARF must be differentiated from prerenal forms of azotemia and for this reason an intravenous fluid challenge is administered to neonates who have decreased urinary flow rate and increased serum creatinine. Several investigators have examined the usefulness of urinary chemical determinations in distinguishing ARF from decreased renal perfusion (prerenal azotemia). The most useful of such urinary indices are the fractional excretion of sodium (FE_{Na}) and the renal failure index (RFI). The latter is calculated using the following formula:

$$\frac{\text{Urinary sodium concentration (mEq/l)}}{\text{Urine/plasma creatinine concentration ratio}}$$

In general an FE_{Na} of above 2.5%–3.0% or an RFI of more than 3.0 is present in most neonates with oliguric renal failure [6–8]. Unfortunately, these tests are not infallible. Ellis and Arnold [8] found that an FE_{Na} above 2.5% was present in all neonates with oliguric ARF (highly sensitive), but unfortunately these values were also present in infants with prerenal oliguria (poor specificity). Others have also failed to find urinary indices useful in the diagnosis of neonatal azotemia [10]. Arant [11] warned that the high urinary sodium excretion observed in very premature infants may limit the usefulness of these measures of urinary sodium excretion, especially in the diagnosis of renal failure in neonates born before 32 weeks' gestation. Other urinary indicators, such as the urine to plasma ratio of urea, creatinine or osmolarity have not generally been helpful in distinguishing renal from prerenal azotemia. However, Norman and Asadi [6] observed a urine to plasma osmolar ratio of less than 1.0 in neonates with ARF.

For obvious reasons, nonoliguric renal failure has been recognized less frequently than oliguric renal failure in neonates. Two reports have examined nonoliguric renal failure in infants, which is defined as persistent elevation of the serum creatinine above 1.5 mg/dl associated with a normal urinary flow rate [9, 12]. Neither FE_{Na} or the RFI were beneficial in the diagnosis of nonoliguric renal failure in these two studies.

In summary, ARF in neonates is characterized by a persistently elevated serum creatinine of greater than 1.5 mg/dl. When such a patient is encountered (without extracellular fluid volume overload), a diagnostic intravenous fluid challenge of 10 ml/kg of either 0.25% or 0.45% sodium chloride over 1 h is most helpful in excluding a prerenal form of azotemia. In oliguric neonates, the fractional excretion of sodium of below 3% or an RFI of below 3 will generally predict which infants will respond to a fluid challenge with a reduction in the serum creatinine. In neonates with azotemia and signs of excessive extracellular fluid volume, a fluid challenge is contraindicated. Urinary indices to determine the fractional excretion of sodium and the RFI should be obtained.

Incidence of neonatal acute renal failure

The incidence of oliguric ARF in neonatal intensive care units ranges from 1% to 6% [6, 9]. These figures probably underestimate the incidence of renal failure because the nonoliguric form of ARF is seldom recognized. To determine how frequently ARF was recognized in our neonatal intensive care unit, the discharge diagnoses of 2,513 consecutive neonatal admissions were reviewed. When a diagnosis of ARF was listed, the chart was reviewed to determine whether the diagnosis satisfied the criteria of urinary flow rate less than 1 mg/kg per hour (unresponsive to a fluid challenge) accompanied by a serum creatinine greater than 1.5 mg/dl. Oliguric ARF was recognized in 75 infants (3%). No child with nonoliguric ARF was identified. As no systematic program for determining serum creatinines was available during

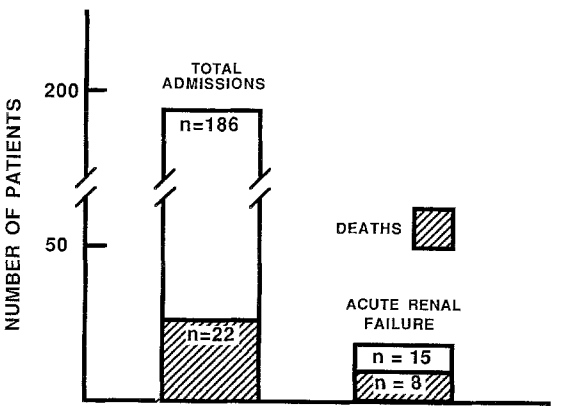

Fig. 1. Prospective study to determine the incidence of acute renal failure in admissions to the Regional Neonatal Intensive Care Unit, University of Tennessee, Memphis, from December 1985 through March 1986. Diagnostic criteria for acute renal failure are defined in the text

the period of retrospective review, we conducted a prospective study in which serum creatinines were repeatedly surveyed in all 186 neonates admitted to our neonatal intensive care unit during the 4-month period from December 1985 to March 1986. Oliguric renal failure (< 1 ml/kg per hour) was identified in 14 infants using the previously stated criteria (Fig. 1). Nonoliguric renal failure (> 1 ml/kg per hour) occurred in 1 neonate. Thus, ARF occurred in 8% of admissions to our neonatal intensive care unit during the 4-month period. These data indicate that a systematic evaluation of serum creatinine concentrations is required to arrive at realistic estimates of the incidence of ARF and that renal failure is quite common in this patient polulation.

Etiologies of acute renal failure in the neonatal period

Common etiologies of ARF in the neonatal period are shown in Table 1. Of the 15 recently identified neonates *(supra vide)* with renal failure, 9 had perinatal asphyxia, 5 were septic and no etiology was identified in 1 child. Other frequently encountered etiologies of ARF are hypoxia [13–15], renal vascular thromboses [16, 17], and cardiac surgery [18, 19]. An underestimated potential etiology of neonatal ARF may be acute uric acid nephropathy [20–22].

We have dialyzed two infants with presumed acute uric acid nephropathy. Both had serum uric acid concentrations above 16 mg/dl (one following cardiac surgery and the other following cardiac catheterization). Ahmadin and Lewy [22] reported three infants in whom renal failure was presumed to be the result of uric acid nephropathy. Hyperuricemia was attributed to perinatal asphyxia in these neonates. Increased production of uric acid occurs following tissue hypoxia [23] and with complete or partial deficiencies in purine enzymes, such as hypoxanthine-guanine phosphoribosyl transferase [20]. In addition, hyperuricemia and increased urinary uric acid excretion have been observed in infants with perinatal asphyxia [24], hemolysis and rhabdomyolysis [25], cyanotic heart disease [26] and following cardiopulmonary bypass surgery [27]. The pathogenesis of acute uric acid nephropathy consists of the precipitation of uric acid or monosodium urate crystals in the lumen of the nephron.

Other factors, myoglobin in rhabdomyolysis, for example, may also contribute to intraluminal obstruction in urate nephropathy. Newborns may be particularly vulnerable to urate nephropathy because of the high fractional excretion of uric acid (4–7 times upper normal values for adults) in both term and preterm infants [28]. Thus an additional urinary burden of uric acid may exceed the solubility of uric acid in the urine. Hyperosmolar radiographic contrast agents [29] increase the urinary excretion of uric acid and might potentially contribute to acute urate nephropathy when uric acid production is increased. Monitoring of serum and urinary uric acid concentrations in infants with ARF may increase the recognition of acute urate nephropathy in this patient population.

Neonates with congenital heart disease represent a patient population at high risk for renal failure during the neonatal period. In the past 4 years, 12 neonates with congenital heart disease have required dialysis in the medical-surgical intensive care unit at LeBonheur Children's Medical Center. Three of our patients developed renal failure following cardiac catheterization. Gruskin et al. [30] reported medullary necrosis and renal tubular vacuolization following cardiac catheterization. Expanded studies by these same investigators revealed that angiographic contrast media reduced renal blood flow in neonatal piglets [30] and in older children [31]. Others have also demonstrated hemorrhagic renal medullary necrosis following administration of radio-contrast media to neonates [32]. Perhaps the more recent, less concentrated, nonionic contrast agents will be less toxic to the neonatal kidney than the currently employed agents [33].

Outcome of acute renal failure in infancy

Three outcome variables of neonatal ARF will be

Table 1. Etiologies of acute renal failure (ARF) in neonates

Renal ischemia	**Nephrotoxins**
Hypotension	Aminoglycosides
Cardiac failure	Indomethacin
Hypoxia/asphyxia	Contrast media
Renal parenchymal	**Obstruction**
Polycystic kidney disease	Intrarenal:
Bilateral cystic dysplasia	Uric acid nephropathy
Hypoplasia/dysplasia	Myoglobinuria
Agenesis	Hemoglobinuria
Pyelonephritis	Extrarenal:
Intrauterine infection	Urethral obstruction
Acute tubular necrosis	Bilateral ureteral obstruction
Renal vascular	**Other**
Renal venous thrombosis	Cardiac surgery
Renal arterial thromboses	Necrotizing enterocolitis
Disseminated intravascular coagulation	

considered: (1) mortality, (2) functional abnormalities, and (3) renal growth.

Mortality

The mortality rate of neonatal ARF ranges from 14% to 73% [4–9, 14, 34] (Table 2). The overall survival rate in previous studies has generally been similar for medical causes and for renal failure associated with congenital renal anomalies. A notable exception is the report by Reimold et al. [4], who found a 75% mortality rate in neonates with renal failure and congenital anomalies. Of the 15 infants recently identified with ARF in our neonatal intensive care unit, 8 ultimately died. Of these, 5 had neonatal asphyxia and 3 overwhelming sepsis (Fig. 1). Two of the deaths were directly attributable to hyperkalemia from renal failure.

Previous survival statistics for ARF in neonates have come from studies in neonatal intensive care units where asphyxia, pulmonary, infectious and vascular etiologies might be expected to account for the majority of patients with renal failure. A different population of infants with ARF is encountered in a medical-surgical pediatric intensive care unit. From 1982 to mid-1986, 16 patients were dialyzed for ARF in the 1st month of life in the medical-surgical intensive care unit at LeBonheur Children's Medical Center. The etiologies of renal failure in these patients are shown in Table 3. Twelve neonates required dialysis for renal failure associated with congenital heart disease. Renal failure followed cardiopulmonary bypass surgery in three infants. The only survivor of these neonates with heart disease was an infant with presumed acute urate nephropathy secondarily associated with cardiopulmonary bypass surgery. Two of three children survived neonatal sepsis and renal failure. The poor survival rate among this population of babies requiring dialysis reflects the severe nature of the underlying etiology of renal failure. Hodson et al. [35] also reported a very poor outcome of neonates and infants dialyzed for renal failure. In their report all five neonates who required dialysis ultimately died. Therefore, the determinant of survival of neonatal ARF often rests with the reversibility of the underlying condition (hypoxia, shock, infection or impaired cardiac output) which is responsible for the renal failure.

In contrast, survival rates in neonates with the nonoliguric form of ARF are excellent. Grylack et al. [12] described seven neonates with nonoliguric renal failure secondary to perinatal asphyxia, respiratory distress syndrome and hypoxia, all of whom survived. Chevalier et al. [9] also described nonoliguric ARF in eight neonates, five of whom were asphyxiated and three had congenital anomalies. There were no deaths in this group of neonates with nonoliguric renal failure. We have recently treated one baby with nonoliguric ARF who also survived.

Table 2. Survival of neonates with oliguric ARF

Reference	Year	No. of patients	Survival (%)	
			Medical ARF	Congenital anomalies
Dauber et al. [14]	1976	7	71%	
Griffen et al. [5]	1976	14	86%	86%
Reimold et al. [4]	1977	36	50%	25%
Anand et al. [34]	1978	14	64%	
Norman and Asadi [6]	1979	20	55%	
Mathew et al. [7]	1980	16	37%	
Ellis and Arnold [8]	1982	28	50%	50%
Chevalier et al. [9]	1984	8	50%	50%

Table 3. Acute peritoneal dialysis in neonates at LeBonheur Children's Medical Center 1982–1986

Etiology of ARF	Total	Survivors
Congenital heart disease	12	1
Post-operative (3)		(1)
Post-catheterization (2)		(0)
Congestive heart failure (7)		(0)
Sepsis	3	2
Necrotizing enterocolitis	1	0
Total	16	3

Positive prognostic factors for survival from neonatal ARF therefore appear to be a reversible insult leading to renal failure, maintenance of urinary flow rate and, perhaps, evidence of renal perfusion in radionuclide scans. Chevalier et al. [9] found evidence of renal perfusion and function with renal scans using ^{99m}Tc-glucoheptinate or ^{131}I-orthohippurate in all eight survivors of nonoliguric renal failure and in three of four survivors of oliguric neonatal ARF. Others, however, have not found radionuclide scans to have prognostic value either for survival or subsequent need for dialysis therapy [36].

Long-term prognosis

Among survivors of neonatal ARF, both decreased glomerular filtration rates and renal tubu-

lar dysfunction have been observed as functional sequelae. Persistently decreased creatinine clearance has been reported in 40% of survivors of the oliguric form of neonatal renal failure caused by asphyxia, vascular thromboses, hypotension and toxins [14, 34, 37, 38]. In contrast, the incidence of chronic renal failure in neonates with genitourinary anomalies and renal failure is much grater. Reimold et al. [4] identified chronic renal failure in 88% of neonates who survived renal failure associated with congenital anomalies. Ellis and Arnold [8] also described subsequent chronic renal failure in two of three neonates with renal failure and obstructive uropathy. Residual impairment in glomerular filtration rate is not limited to children with oliguric renal failure. Chevalier et al. [9] described persistently elevated serum creatinine concentrations in three of eight neonates following non-oliguric renal failure.

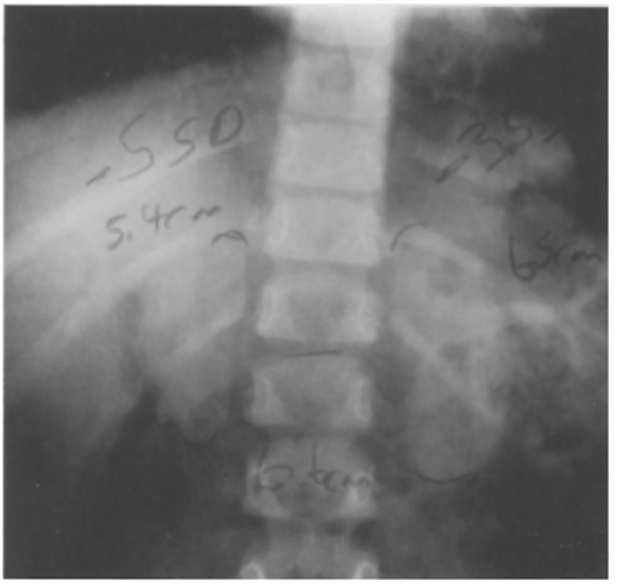

Fig. 2. Bilateral reduction in renal size is seen in this excretory urogram in a 5-year-old girl who had neonatal acute renal failure secondary to asphyxia at birth. The length of the right kidney is 5 SD below the mean of normal and the length of the left kidney is 3,5 SD below the mean of normal. The reduction in renal mass is presumed to be the result of the perinatal renal insult

Renal growth

Renal tubular dysfunction with growth failure and rickets have been described in children who have recovered from neonatal renal failure. Stark and Geiger [13] reported three infants who recovered from renal venous thrombosis during the neonatal period. Each of these infants developed rickets during the 1st year of life. A variety of renal tubular defects was observed with only moderate decreases in the glomerular filtration rate. One child had generalized renal tubular dysfunction, another had impaired abilities to concentrate and acidify the urine, and the third child demonstrated proximal renal tubular bicarbonate wasting. Similar results were found in two of five children who recovered from neonatal renal venous thrombosis [16], and in whom bicarbonate wasting and polyuria were the clinical manifestations of tubulopathy. We are currently treating two children (aged 2 and 5 years) with midly impaired glomerular filtration rate and bicarbonate wasting following ARF associated with neonatal sepsis and perinatal asphyxia. These data indicate that a covert neonatal vascular thrombosis should be considered in children who present in early childhood with renal tubular dysfunction. Whether similar renal tubular dysfunction occurs following nonoliguric renal failure is not known at present.

The developmental status of the neonatal kidney at the time of renal insult may determine the nature of residual functional deficits. Rodriguez-Soriano et al. [38] reported the examination of two children who were suffering from severe dehydration and shock at 13 and 15 months of age. Both children demonstrated cortical necrosis in renal biopsy specimens; however, unlike adult patients with cortical necrosis, the juxtamedullary nephrons in infants' kidneys were severely atrophic and sclerotic with normal nephrons located in the outer cortex. In an adult with cortical necrosis, sclerotic glomeruli in the outer renal cortex and normal parenchyma in the deep cortex were demonstrated. Renal function studies were consistent with the site of parenchymal injury. The two infants were unable to concentrate their urine or to lower their urine pH to less than 5.9 and had a diminished free water clearance for the degree of distal sodium delivery. In contrast, the adult patient with cortical necrosis demonstrated relatively intact deep cortical nephron function by lowering the urinary pH to 4.9, concentrating the urine to 544 mosmol/kg and demonstrating normal free water clearance. The explanation of the predilection for necrosis in the deep cortex of the infants with renal failure may be related to the relatively greater perfusion of the inner cortex in neonates [39]. It may be hypothesized that the lower perfusion of the outer renal cortex in infants may render this area of the kidney more resistant to renal ischemia.

The effect of ARF associated with hypoxia and shock upon renal growth was emphasized by

Anand et al. [34], who reported bilateral renal papillary necrosis in three of five survivors of neonatal ARF, and diffuse or focal cortical atrophy in four of the five patients. Similarly, long-term bilateral or unilateral renal atrophy was observed in children with renal venous thrombosis in the neonatal period [16]. Without an awareness of the neonatal renal insult, the excretory urograms of these children with cortical atrophy might easily be interpreted as representing congenital hypoplastic, dysplastic kidneys. Figure 2 demonstrates bilateral small kidneys in a 5-year-old girl with a creatinine clearance of 51 ml/min per 1.73 m^2. She has a serum bicarbonate concentration of 16 mEq/l and has polyuria and polydipsia. This girl was born at 34 weeks' gestation with severe perinatal asphyxia and hypotension.

Conclusion

The frequency with which pediatric nephrologists encounter ARF in the neonatal period is likely to increase as the survival of very premature infants improves. Clinical indices both to diagnose renal failure and to predict outcome are needed, particularly in very premature infants. Neonates with ARF from congenital heart disease, cardiac surgery or congenital anomalies of the urinary tract appear to have an extremely poor prognosis. Improved therapeutic interventions are needed to increase survival in these and in other groups of neonates with ARF.

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