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Acute Renal Failure in the First Month of Life

MB ABDURRAHMAN*

Summary

Abdurrahman MB. Acute Renal Failure in the First Month of Life. *Nigerian Journal of Paediatrics* **1982; 9: 71.** A review of 19 newborn infants with acute renal failure has revealed the presence of congenital structural anomalies in 63% of the cases. These anomalies included posterior urethral valve, prune belly syndrome, renal dysplasia and hydronephrosis. In the remaining 37% of the cases, renal failure was associated with acute gastroenteritis, septicaemia and meningitis. The overall mortality was 63% while the mortality was 50% among those with anomalies and 85·7% among those without anomalies. It is concluded that a high index of suspicion and simple laboratory tests, including serum electrolytes, urea and creatinine, are particularly useful in the diagnosis of acute renal failure in the newborn. In addition, urine volume, its appearance and composition should be determined routinely in all high-risk infants, especially in asphyxiated babies, as well as in those with severe dehydration and shock.

Introduction

ALTHOUGH impaired renal function has been recognized in the newborn period,[1] [2]only few reports on renal failure in early life have been documented.[3-6] This may be due to the rarity or misdiagnosis of renal failure in early life. The purpose of the present study was to document the causes, course and outcome of acute renal failure, (ARF) in infants, aged between birth and 30 days, admitted into the Department of Paediatrics, Ahmadu Bello University (ABU), Zaria.

Ahmadu Bello University, Zaria

Department of Paediatrics

* Professor

Materials and Methods

In the present study, ARF is defined as serum urea level of >5mmol/l (>30mg/dl) and rising, or serum creatinine level of >90umol/l (>1mg/dl) and rising, persisting for more than 24 hours. Cases with transient impairment of renal function were excluded from the study. Thus, infants with temporarily elevated serum urea following acute gastroenteritis and dehydration were excluded. Oliguria was excluded as a *sine qua non* criterion for the diagnosis since accurate fluid output in the newborn was not often recorded. All infants who fulfilled the above criteria were included in the study which covered the period, January 1, 1978 to December 30, 1980. Investigations carried out routinely included urinalysis, urine culture,

serum urea, creatinine and electrolytes and, in some cases, blood culture, intravenous pyelography (IVP) and voiding cystourethrogram.

Results

During the three-year period, there were 19 patients (17 male and two female) with ARF in the first month of life. The mean age at presentation was 14 days (range, birth to 30 days).

There were two groups of patients; Group A patients (12) were those with structural malformation of the kidneys or urinary tract, while Group B patients (seven) were without structural malformation (Table). The clinical features common to both groups were non-specific symptoms including lethargy, vomiting and poor feeding. No patient presented with oedema.

TABLE

Conditions Associated with Acute Renal Failure in 19 Neonates

Condition	*No of Cases*
Group A: Malformations	
Posterior urethral valves	6
Prune belly syndrome	3
Renal dysplasia	2
Hydronephrosis	1
Group B: Non-malformations	
Acute gastroenteritis and septicaemia	3
Septicaemia	2
Acute gastroenteritis	1
Meningitis	1

Patients with Structural Malformations (Group A)

Abdominal distension was present in all the patients in this group. The causes of the distension were distended urinary bladder in six infants with posterior urethral valves (PUV); palpable and enlarged kidneys in four cases (prune belly syndrome (PBS) 3, hydronephrosis 1), and ascites in two patients with renal dysplasia. One patient with PUV and septicaemia presented with convulsion. Lax abdominal wall with wrinkled skin was the presenting symptom in infants with PBS. Additional anomalies present in patients with PBS included undescended testes, hernia and absent leg.

Patients without Structural Malformations (Group B)

The seven babies in this group were acutely ill; four were febrile (temperature 38.5–39.2°C). They were all moderately dehydrated although only four of them had gastroenteritis on admission. However, oliguria was recorded only terminally in three patients. The two infants with septicaemia alone, had uvulectomy a few days prior to admission.

Investigations

Urinalysis showed variable proteinuria, microscopic haematuria and casturia. The highest levels of serum urea (31–33 mmol/l, 185–200 mg/dl) were found in patients with PUV and in those with septicaemia. Variable degree of hyperkalaemia was found in all the patients. No difference was detectable in biochemical results between infants with and those without structural malformation of the kidneys or urinary tract. IVP in patients with PBS showed dilated and tortuous ureters. In those with PUV, voiding cystourethrogram showed bladder trabeculation, distension and an obstruction below the bladder neck. Organisms were isolated from blood culture in three babies with septicaemia: *Klebsiella* in two and *E. coli* in one.

Course and Outcome

Group A Patients

The six children with PUV had bladder catheterization and intravenous fluids to correct electrolyte imbalance, followed by resection or diathermy excision of the valve. Three of these

patients died of septicaemia (*E. coli* 2, *Staphylococcus pyogenes* 1) within a week of the operation. The remaining three are alive and well, with normal serum urea and creatinine. Two of the three patients with PBS are alive. One of these survivors has been followed up for over three years; he has normal serum urea but a repeat IVP showed no evidence of improved renal function. The second survivor, now aged one year, has slowly progressive impairment of renal function manifested by a rising serum urea and creatinine and falling urine specific gravity. The third patient with PBS was an asphyxiated premature baby who was resuscitated but died at four days of age. The necropsy findings were hyaline membrane disease, pneumonia, dilated ureters and renal tubules. The two infants with renal dysplasia died at 18 hours and 20 days of age, respectively.

At necropsy, in addition to bilateral dysplastic changes in the kidneys, there were renal microcysts in one, and interstitial fibrosis of the kidney and the liver in the other infant. Permission for necropsy in the remaining babies was not granted.

Group B Patients

All the babies in this group had intravenous fluid replacement and maintenance. Patients with proven or suspected infection were treated with penicillin and kanamycin or gentamycin. The illness in these infants ran a fulminant course with rapid azotaemia. Six (85·7%) of the seven infants died, five in less than 24 hours of admission and the sixth on the fourth day of admission. The only survivor was a 30-day old infant admitted with septicaemia, clinical and laboratory features of haemolytic-uraemic syndrome. He was treated with intravenous fluids, blood transfusion, penicillin and kanamycin. Three months after discharge, he had normal renal function and normal haematological indices.

Discussion

The present study indicates that with an average of six cases a year, ARF in the newborn period is relatively common in our institution. This prevalence is probably an underestimation, since renal function tests are not carried out routinely on all newborn infants with asphyxia, septicaemia and gastroenteritis; nor are autopsies performed frequently in the institution.

The mortality of 63% in the present study was slightly higher than the 56% reported by Reimold, Don and Worthen,[7] but much higher than one of 45% reported by Norman and Asadi.[5] In contrast to the finding of Reimold, Don and Worthern,[7] the mortality in our series was higher in infants without structural malformation of the renal tract than in those with anomalies. It is noteworthy that none of the cases reported by Norman and Asadi[5] had congenital renal anomalies. This may be partly explained by the fact that only few patients with renal anomalies, specifically PUV, are symptomatic during the first year of life.

Non-specific clinical features in the present series such as lethargy, vomiting, and poor feeding were not helpful in the diagnosis of renal failure because these features also occur commonly in other conditions in newborn babies. However, the possibility of renal failure was considered in those babies with structural malformation of the kidneys or renal tract because of additional signs of abdominal distension and palpably enlarged kidneys. In contrast, babies without congenital renal anomalies were admitted with shock secondary to severe dehydration or septicaemia, presumably resulting in renal hypoperfusion and subsequent renal shut-down.

It is possible that late presentation, inappropriate intravenous fluid therapy, and the use of potentially nephrotoxic drugs (kanamycin and gentamycin) contributed to the poor prognosis

in some of the patients. Although oliguria is a most useful sign of ARF, the sign is easily overlooked or ignored,[8] especially in a busy, understaffed newborn nursery or children's emergency unit. It is worthy of note that none of our patients presented with oedema. One patient had convulsion, a feature which was relatively common in the series reported by others.[3–7] The reason for this difference is not clear.

Infection appears to be a common cause of ARF in early life, although its relative importance varies from place to place.[9] In the present series, ARF was associated with infection in 37% of the cases. Asphyxia and respiratory distress syndrome respectively, are reported to cause ARF in the newborn.[8–10] However, only one infant in the present series had PBS, asphyxia and hyaline membrane disease. The importance of asphyxia as a cause of ARF in neonates might have been underestimated because renal function tests were not carried out routinely, in all asphyxiated newborn babies in the institution.

Recent studies have demonstrated the usefulness of renal failure index, fractional excretion of sodium, and osmolality of urine to plasma ratio in the diagnosis of ARF in the newborn.[5 6 9] However, the results of these tests may not be readily available for the clinician to initiate appropriate therapy before irreversible renal failure occurs. Therefore, a high index of suspicion and simple laboratory tests including serum urea, creatinine and electrolytes especially, potassium, are particularly useful for an early diagnosis. The volume, appearance and composition of urine should be determined routinely, in all high-risk infants such as asphyxiated babies and those with severe dehydration and shock as previously suggested by Daniel and James.[1]

References

1. Daniel SS and James LS. Abnormal renal function in the newborn infant. *J Pediat* 1976; **88:** 856–8.
2. Fawer CL, Torrado A and Guignard JP. Maturation of renal function in full-term and premature neonates. *Helv Paediat Acta* 1979; **34:** 11–21.
3. Griffin NK, McElnea J and Barratt TM. Acute renal failure in early life. *Arch Dis Childh* 1976; **51:** 459–62.
4. Barratt TM. Renal failure in the first year of life. *Brit Med Bull* 1971; **27:** 115–21.
5. Norman ME and Asadi FK. A prospective study of acute renal failure in the newborn infant. *Pediatrics* 1979; **63:** 475–9.
6. Mathew OP, Jones AS, James E, Bland H and Groshong T. Neonatal renal failure: usefulness of diagnostic indices. *Pediatrics* 1980; **65:** 57–60.
7. Reimold EW, Don TD and Worthern HG. Renal failure during the first year of life. *Pediatrics* 1977; **59:** (**suppl**): 987–94.
8. Dauber IM, Krauss AN, Smychych PS and Aud PAM. Renal failure following perinatal anoxia. *J Pediat* 1976; **88:** 851–5.
9. Gordillo-Paniagua G and Velasquez-Jones L. Acute renal failure. *Pediat Clin North Am* 1976; **23:** 817–28.
10. Guignard JP, Torrado A, Mazouni SM and Gautier E. Renal function in respiratory distress syndrome. *J Pediat* 1976; **88:** 845–50.

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