

Chronic Renal Failure in Children

S. Jamro, N. A. Channa, A. H. Shaikh, A. Ramzan (Department of Paediatrics, Chandka Medical College, Larkana and National Institute of Child Health*, Karachi.)

Introduction

In CRF there is irreversible and progressive loss of renal function with residual glomerular filtration rate (GFR) less than 30%. Metabolic disturbances occurring in CRF reflect an adaptation to the loss of renal function.¹

The exact prevalence and incidence of CRF in children of Pakistan is not known but available figure suggest that it accounts for 10-12% of all renal cases.² According to United States Renal Data System (USRDS), 20 new ESRD cases per million population of children per year are registered for renal replacement therapy.³

The goals of management of CRF in children is not only to prevent progression to ESRD but to fulfil the physiological and emotional needs of children to the best possible quality of life. These goals can be achieved by:

1. Early and appropriate treatment of reversible causes of CRF like vesicoureteric reflux (VUR), posterior urethral valves (PUV) and urolithiasis.
2. Early and appropriate conservative management of CRF may help to achieve normal growth and development.
3. Periodic monitoring for rate of progression to end stage renal disease helps to plan for renal replacement therapy.^{3, 4}

In Pakistan there are very few studies on chronic renal failure in children. Further more, there are very limited facilities for dialysis and renal transplant for children with ESRD. This study was planned with the objectives: (a) to determine the clinical presentation of CRF in Pakistani children and (b) to find out the etiology of CRF.

Patients and Methods

This is a descriptive prospective study of children of 1-12 years of age, attending children hospital, Chandka Medical College Larkana and National Institute of Child Health (Nephrology Unit) Karachi from January 1993 to December 1996.

Seventy eight cases, confirmed as CRF on the basis of residual glomerular filtration rate (GFR) less than 30% for more than 3 months were included in this study. Detailed history and thorough examination was done and recorded in a special proforma. Blood CP, urine analysis, blood urea, serum creatinine, serum electrolytes, serum calcium, serum phosphorus and alkaline phosphatase as well as X-ray hands, ultrasound for kidneys, ureters and bladder (KUB) were done in all cases. GFR was determined by the height/serum creatinine plotted on nomogram.⁵ Other specific tests like micturating

cystourethrogram, intravenous pyelography, diethylene triamine pentacetic acid (DTPA) and dimercapto succinic acid (DMSA) renal nuclear scans, serum complement (C3) level, antinuclear antibodies (ANA), Anti double stranded DNA and renal biopsy were done when indicated to confirm the underlying cause.

Results

Seventy eight cases of CRF were included in the study. Majority (90%) were over 3 years of age with male to female ratio 1.8:1 (Table 1). Most common clinical presentations were anaemia, growth retardation and urinary complaints like dysuria, frequency of micturation, dribbling of urine and weak urinary stream (Table 2). Most common causes of CRF were reflux nephropathy, posterior urethral valves, chronic glomerulonephritis and urolithiasis (Table 3).

Discussion

CRF is an irreversible progressive renal disorder, which ultimately leads to end stage renal failure (ESRF). Renal replacement therapy in the form of dialysis or renal transplant are the ultimate options for the management of these children. These treatment options are very costly and are available in only few centres in Pakistan. The age of presentation of CRF correlates closely with the underlying cause. CRF in children under 5 years of age is commonly the result of congenital nephropathies and obstructive uropathy, whereas after 5 years of age, acquired glomerular diseases (chronic glomerulo nephritis, haemolytic uremic syndrome) or hereditary disorders (Alport's syndrome, Juvenile nephronophthiasis) are common causes.^{6,7} Majority of the cases (64%) in our study presented after 6 years of age. There was a male predominance (M:F ratio 1.8:1) which may be due to little higher number of posterior urethral valves and Alport's syndrome. Anemia was invariably present in all cases (93.3%), which is similar to a study by Nawarro M. et al.⁸ Our children had more growth retardation (74.3%) as compared to 50% in Belts and Margrath Study.⁹ This may be due to late diagnosis and more prevalence of malnutrition in general population of our country. Hypertension with or without cardiac failure was found in 53.8% of cases, which is similar to 49% in Schorer's study.¹⁰

The most common primary cause leading to CRF in our study was reflux nephropathy (24.5%) which is similar to Indian (20%) and U.K (21%) studies.^{1,11} We had more cases of posterior urethral valves (23%) as compared with Indian (7%) and U.K (12%) studies.^{1,11} This may be due to more referrals, as we are the only referral centers in Sindh and Balochistan with facilities of PUV fulguration. Chronic glomerulonephritis (15%) as a cause of CRF is similar to U.K (13%) and Indian (17%) studies.^{1,11} Urolithiasis (14%) was similar to the Indian study¹¹ but there was no case reported in U.K study.¹

The prevalence of congenital dysplasia/cystic renal diseases in this study is low as compared to other two studies, but it is comparable to figures of USA where polycystic kidney disease is responsible for 4.2% of ESRD in children.

Conclusion

The most common clinical presentation of CRF is anaemia, growth retardation, urinary complaints and hypertension in our study. The common cause of CRF include reflux

nephropathy, posterior urethral valves, chronic glomerulonephritis and urolithiasis in this study. Early diagnosis and management of these conditions may prevent or delay the progress to end stage renal disease. Preventable cause of CRF like VUR, PUV and urolithiasis should be detected early and managed promptly before they cause irreversible damage to kidneys.

References

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