

Evaluation of Clinical Presentation to Recognize Chronic Renal Failure in Children

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ABSTRAK

Latarbelakang: Gagal ginjal kronik (GGK) ialah kehilangan fungsi ginjal progresif dan ireversibel mulai dari tahap ringan sampai gagal ginjal tahap akhir. Diagnosis dini GGK pada anak sering sulit ditegakkan karena presentasi klinis awal bervariasi dan tidak spesifik. Diagnosis dan pengobatan konservatif sejak awal penyakit dapat memperlambat progresifitas penyakit dan penggunaan pengobatan pengganti ginjal.

Tujuan : Melacak presentasi klinis gagal ginjal kronik pada anak yang dirawat di RSUP dr. Wahidin Sudirohusodo Makassar. **Subjek dan cara kerja :** Penelitian retrospektif dilakukan pada anak-anak dengan GGK yang dirawat di RSUP dr. Wahidin Sudirohusodo Makassar mulai Januari 1999 sd. Desember 2001. Kriteria inklusi yaitu penderita dengan kreatinin serum di atas normal atau laju filtrasi glomerulus $< 30 \text{ ml/ menit/ } 1,73 \text{ m}^2$ sesuai rumus Schwartz selama 3 bulan atau lebih dan penderita dengan catatan medik lengkap. Presentasi klinik dikumpulkan dari catatan medik dan selanjutnya dianalisis. **Hasil :** 27 anak berumur 2,5 sampai 15 tahun memenuhi kriteria penelitian dengan usia rata-rata $10,30 \pm 3,22$ tahun; 88,90% di atas 5 tahun. Laki-laki (60,70%) lebih banyak dari anak perempuan dengan rasio 2:1. Presentasi klinis yaitu pucat (92,60%), retardasi pertumbuhan (51,9%), dispneu (48,15%), edema (44,4%), demam (37,04), hipertensi (36,3%), anuria (36,3%), hematuria gros (35,7%), muntah (29,6%), kejang (25,92%), ensefalopati hipertensi (22,2%), dan oliguria (18,2%). Penyebab GGK terbanyak pada penelitian ini yaitu glomerulopati (70,37%) meliputi sindrom nefrotik primer (58,74%), glomerulonefritis kronik (30,15%) dan nefritis lupus (11,11%) sedangkan sisanya tidak diketahui (29,63%). Tujuh belas anak (62,96%) mendapat pengobatan konservatif sedangkan 37,03% anak dengan gagal ginjal tahap akhir diberikan pengobatan dialisis yaitu dialisis peritoneal (22,9%) dan hemodialisis (7,41%). **Simpulan :** Walaupun pucat dan retardasi pertumbuhan merupakan gejala umum GGK pada anak, gejala klinis lain dan data laboratorium perlu dilacak untuk menyokong diagnosis dini GGK.

Kata kunci: Presentasi klinis, gagal ginjal kronik, anak

INTRODUCTION

Chronic renal failure (CRF) is defined as the stage at which the irreversibly damaged kidney are unable to maintain the homeostasis of the body.¹ The progressive and irreversible loss of renal function may range in severity from mild renal insufficiency to end stage renal disease (ESRF).^{2,3} The first clinical sign of functional deterioration if the nephrons are progressively damaged is a diminution of renal reserve and a decrease of GFR. During the initial phase of loss of renal function, the body uses various physiological mechanisms to maintain homeostasis so that the clinical manifestation still remains within normal variation.¹

The incidence of progressive renal diseases leading to CRF in children varies from 1.5 to 3.0 children per million

total population in patients less than 16 years of age. 1 ESRF at Pakistan occurs in 100/million population/year.⁴

CRF in children is usually asymptomatic initially, until the GFR falls to 10-25 ml/min. Gulati S et al reported that children with CRF had been symptomatic for a mean of 33.2 months and 54% of them were in ESRF on the initial presentation.⁵ Early diagnosis of CRF is therefore very crucial unless a delay diagnosis may lead to delay initiating conservative management to retard the progression of the disease and to delay the need for renal replacement therapy.⁶

The present study evaluates the clinical presentations of CRF in children hospitalized at the Department of Child Health, Dr Wahidin Sudirohusodo Hospital, Makassar

PATIENTS AND METHODS

This was a retrospective study conducted on children with CRF hospitalized at the Department of Child Health, Dr Wahidin Sudirohusodo Hospital, Makassar from January 1992 to December 2001. CRF was defined as irreversible deterioration of renal function with persistent elevation of serum creatinine or GFR <30 ml/min/1.73m² for ≥ 3 months. The inclusion criteria in this study was patients aged ≤ 15 years with a sustained elevation of serum creatinine or GFR < 30 ml/min/1.73m² calculated with Schwartz's formula, during ≥ 3 months⁷ and those who have sufficient medical data to be evaluated. The clinical presentation of patients with CRF at the time of admission were collected from the medical record and evaluated thereafter.

RESULTS

During the study period, 27 patients aged from 2.5 to 15 years were evaluated. Their mean age was 10.30±3.22 years, 88.90% were over 5 years and 66.7% were boys with a boy-to-girl ratio of 2:1. The common presentation of childhood CRF in the present study was pallor (92.60%) and failure to thrive (51.9%), followed by other symptoms; generalized edema (44.4%) consisted of periorbital, facial, pretibial, pedal, genital edema, and ascites (92.6%, 44.4%, 85.2%, 55.6%, 44.4%, and 70.4%) (Table 1) The laboratory data indicated Hb < 10 g/dl in 92.6% cases and 10.5g/dl 7.4% cases, increased leukocyte count 37.04%, elevated BSR 87.5%, severe hypoalbuminemia 59.26%, hypercholesterolemia 33.33%, proteinuria 2+ or more 74.07%, and microscopic hematuria (red blood cells > 5 per HPF) in 74.07%.

The most frequent causes of CRF in this study was glomerulopathy (70.37%) including primary nephrotic syndrome (58.74%), chronic glomerulonephritis (30.15%) and Lupus nephritis (11.11%); whereas the remaining causes (29.63%) were unidentified. Seventeen patients (62.96%) were on conservative medical treatment and 37.03% cases with ESRF were treated with peritoneal dialysis (22.9%) and hemodialysis (7.41%), respectively. Few patients with ESRF died and some of them were lost on follow up.

DISCUSSION

The mean age at hospitalization (10.30 ± 3.22) in the present study are slightly higher than the study among Italian children (6.9 ± 5.4)⁷, Iranian children (7.9±4.5)⁸, and Jordanian children (7.5 ± 3.9)⁹ CRF in children in this study is mostly over 5 years (88.90%) almost similar to the study in India(96%);¹⁰ whereas the Italian report stated that 58.6% of children are over 10 years.¹¹

The boy-to-girl ratio (2:1) in the present study is similar to the Italian study⁷ but different from the Chilean study (1:1).¹¹

The most common clinical presentation in the present study (pallor - 92.6% and failure to thrive - 51.85% vs. 17.6% and 14.8% in Pakistan⁸) differs from Pakistan study (dyspnea, 54.8% and fever 52.6%³ vs. 48.15% and 37.04% in this study). (Table 2)

Table 1. Distribution of the clinical presentation of CRF according to age

Clinical presentation	Age (years)			Total (%)
	0-5(%)	5-10(%)	10-15(%)	
Pallor	2(7.40%)	11(40.74%)	12(44.44%)	25(92.6%)
Failure to thrive	1(3.7%)	8(22.22%)	7(25.92%)	14(51.85%)
Dyspnea	1(3.7%)	5(18.51%)	7(25.92%)	13(48.15%)
Edema	1(3.7%)	6(22.22%)	5(18.51%)	12(44.44%)
Fever	2(7.40%)	3(11.11%)	5(18.51%)	10(37.04%)
Vomiting	1(3.7%)	2(7.40%)	5(18.51%)	8(29.62%)
Hypertension	0(0.0%)	2(7.40%)	5(18.51%)	7(25.92%)
Seizures	0(0.0%)	3(11.11%)	4(14.81%)	7(25.92%)
Anuria	2(7.40%)	2(7.40%)	2(7.40%)	6(22.22%)
Gross hematuria	1(3.7%)	3(11.11%)	1(3.7%)	5(18.51%)
Oliguria	0(0.0%)	0(0.0%)	2(7.40%)	2(7.40%)

Pallor, noted usually in children with CRF, is the clinical sign of anemia caused by renal dysfunction in producing erythropoietin;¹ the use of subcutaneous recombinant erythropoietin, therefore may improve the anemia and intellectual performance as well.¹²

The etiology of CRF differs from one to another study. The leading cause of CRF in this study is glomerulopathies (70.37%); similar to the leading cause of CRF reported in the literature¹ and in India⁵ but different from other reports documenting the most frequent causes of CRF are obstructive uropathy (18.1%),¹¹ hypodysplasia associated with urinary tract anomalies (53.6%),⁷ urological abnormalities and malformations (42.1%),⁹ and congenital urological malformations⁸ (Table 3).



Table 2. Comparison of clinical presentation of CRF in children between this study and Pakistan study

Clinical presentation	This study (%)	Pakistan (%) ⁶
Pallor	92.60	17.80
Failure to thrive	51.90	14.80
Dyspnea	48.15	54.80
Fever	37.04	52.50
Edema	44.44	29.60
Vomiting	29.60	29.60
Hypertension	25.92	00.00
Seizures	25.93	17.80
Anuria	22.22	-
Gross hematuria	18.50	-
Oliguria	18.20	13.30
Polyuria	-	10.40
Tetany	-	4.40
Bone fractures	-	0.70
Abdominal pain	-	7.40
Polydipsia	-	5.90

Table 3. Comparison of etiology of CRF in children between this study and others⁷

Etiology	This study %	India ^{5,10} %	Chile ¹¹ %	Iran ⁸ %	Jordan ⁹ %
Glomerulopathy	70.37	37.50 / 27.54	18.3	10	14.4
Renal hypo/dysplasia	-	- / 8.5	18.7	-	5
Obstructive uropathy	0	52.00 / 31.80	18.1	46.98	42.1
Reflux nephropathy	-	- / 18.72	18.7	-	-
Hereditary nephropathy	-	6.3 / 6.55	-	21	29.7
Hemolytic uremic syndrome	-	- / 1.64	-	-	4.5
Unidentified	29.63	4.2 / -	-	-	4.5

Seventeen children (62.96%) in the present study were given conservative medical treatment and 37.03% with ESRF either with peritoneal dialysis (22.9%) or hemodialysis (7.41%). Forty eight percent of CRF among Chilean children⁹ and 21% of CRF among Indian children⁵ were on conservative medical treatment; whereas 42.2% of Chilean children⁹ and 14.6% of Indian children, who were in ESRF, had undergone dialysis and renal transplantation.

CONCLUSION

This study demonstrates that although pallor and failure to thrive as the common clinical presentation of CRF can be used to recognize CRF in children, other clinical presentations as well as laboratory data is still required for the early recognition of children with CRF.

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