Continuous ambulatory peritoneal dialysis for infantile chronic renal failure
(A case report)

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ABSTRACT

Pungky AK, Damanik MP, Iljima K - Continuous ambulatory peritoneal dialysis for infantile chronic renal failure (A case report).

A two month old male infant with lethargy, vomiting, and loss of body weight was referred to Kobe University Hospital, Kobe, Japan. He had increased levels of BUN and serum creatinine, and severe metabolic acidosis. Ultrasonography exhibited hypoplasia kidney. Treatment with continuous ambulatory peritoneal dialysis (CAPD), recombinant human erythropoietin, and endogenous human growth hormone was started immediately on the admission day. All of his symptoms were disappeared and he grew up well. He was discharged 3 months after admission and he had been treated with the above therapy in Out Patient Clinic. CAPD on CRF patient is the most essential management. CAPD system should be introduced to save infants and children with chronic renal failure in Indonesia.

Key words: chronic renal failure - peritoneal dialysis - infant - hypoplasia kidney - recombinant growth hormone

ABSTRAK

Pungky AK, Damanik MP, Iljima K - Ambulatory peritoneal dialysis pada gajal ginjal kronik bayi

Seorang anak laki-laki, 2 bulan dengan keadaan lemah, muntah dan penurunan berat badan, dengan peningkatan BUN dan kreatinin serum dan asidosis. Dari ultrasonografi didapatkan ginjal hipoplisis. Dengan terapi CAPD, eritropoeitin dan hormon pertumbuhan recombiant, kejadian gajal ginjal meningkat dan pasien tumbuh semakin dengan baik.


INTRODUCTION

Chronic renal failure is characterized by a decreased glomerular filtration rate (GFR) and histologic evidence of the reduction of nephron population. The clinical course is typically one of the progressive and inevitable loss of nephron function, ultimately leading to end stage renal disease (ESRD). The incidence of CRF among children less than 16 years of age varies between 1.5 and 3.0 per million2. In Europe and USA, using established data bases and allowing for the differences in age definition between the data bases, the reported incidence in the 0-15 year age group is approximately five million child population per year2. The distribution of primary renal diseases in the child differs markedly from an equivalent adult population structural renal disease and hereditary and familial nephropathies predominates in the 0-15 year age group and particularly in the 0-2 year age group.
We present a case of chronic renal failure infant due to congenital abnormality, treated with continuous arteriovenous dialysis, recombinant human erythropoietin, and recombinant human growth hormone.

The problem is CAPD in infant is very rare1. The aim of this study is to report that CAPD system should be introduced to save Indonisian infants and children with chronic renal failure.

CASE REPORTS

A two month old infant with normal gestation, he has the first-born child from healthy young parents at 19 days old was admitted for the first time to Kobe Hospital Japan in February 1999, because of performed lethargy, decreased appetite to drink, loss of body weight to 600 g, tachycardia, decreased urine, blood pressure 100/58, anemia (hemoglobin 8.7 g/dl), leukocyte count 8000/UL, platelet count 525,000/UL. Laboratory findings were metabolic acidosis, blood urea nitrogen (BUN) 141.9 mg/dl, serum creatinine 5.22 mg/dl, total serum protein 6.8 g/dl, serum albumin 4.3 g/dl, serum sodium 142 mEq/l, serum potassium 3.1 mEq, serum chloride 114 mEq. The clinical diagnosis was chronic renal failure. Ultrasound examination exhibited dilated renal pelvis, but not severe, and moderate hydronephrosis. Consultation with nephrologist showed that there was congenital renal failure with hypoplasia kidneys, nevertheless the nephrologist didn’t advice to perform an operation but suggested a conservative therapy instead. Unfortunately BUN, serum creatinine, and potassium increased and the patient developed into severe metabolic acidosis. Therefore, he was managed to start with continuous ambulatory peritoneal dialysis (CAPD) using Tenckhoff catheter with peritoneal dialysis solution 60 ml, 8 cycle and 40 minutes in abdomen.

Two weeks later, the baby suffered form acute otitis media and peritonitis with symptoms of nausea, vomiting, and cloudy in the dialysis solution. The culture of acute otitis media showed the presence of (MRSA) and Pseudomonas aeruginosa and that of dialysis solution showed the presence of Pseudomonas aeruginosa as well. White blood count in the dialysate was more than 100/UL, C-reactive protein was 4.41 and leukocyte count was increased. We treated the patient with antibiotics, piperacillin and vancomycin 10 mg/kg/dose 6 hrly (based on the sensitivity test). Anemia (hemoglobin 7.2 g/dl) was treated with recombinant erythropoietin 50 u/kg body wt 3 times wkly twice a week and recombinant growth hormone was used for growth therapy. Acute otitis media disappeared, but peritonitis recurred with increased C-reactive protein and white blood count from the dialysate. We tried another therapy with third generation of cephalosporin and tobramycin based on pseudomonas aeruginosa is sensitive to this therapy and we changed of the catheter as well.

The condition of the baby improved, all of his symptoms disappeared, the body weight was increased and laboratory findings showed CRP: 0.14, the dialysis solution, is clear culture from dialysis was negative, and he grew up well.

DISCUSSION

Renal failure develops when renal function is diminished to the point at which body fluid homeostasis can no longer be maintained. Chronic renal failure in children under five years of age is commonly the result of anatomic abnormalities (hypoplasia, dysplasia, abruption, malformations) whereas after five years of age it is the result of acquired disease; glomerular disease (glomerulonephritis, hemolytic uremic syndrome) or hereditary disorders (Alport syndrome, cystic disease) predominates. This incidence is resulted from defect congenital anomaly in the kidney or urinary tract, due to developmental abnormalities (cystic disease, hypoplasia, dysplasia, agenesis), obstruction lesion5. In the present patient, the cause of renal failure in the newborn baby might derive from congenital abnormality of hypoplastic kidney. Renal hypoplasia is defined as a reduction in the number of nephrons and ducts, and/or decreased nephron size, with normal development and differentiation of those nephrons and ducts that are present, Hypoplasia is regarded as non genetic, although it can be part of malfunction syndromes such as fetal alcohol syndrome and several autosomal syndromes. Clinical finding in patient related to the renal failure includes pallor (anemia), diminished urine output, vomiting, and lethargy due to uremic encephalopathy. Laboratory findings were anemia
(dilution), hyperkalemia, metabolic acidosis, increased levels of BUN and serum creatinine. Renal ultrasonography and radiographic scan and renal biopsy may ultimately be required to determine the precise cause of renal failure.

The development of renal failure may be insidious; however, symptoms with anatomic abnormalities may present specific complaints. If renal failure has developed slowly over several years, growth retardation and rickets may develop as well. Prior to the advent of dialysis and transplantation, understanding of the physiological changes that accompanied chronic renal disease was rudimentary. Nutritional status may be monitored by periodic evaluation of the serum albumin, zinc, transferrin, folate acid, and iron level.

The management of the child having chronic renal failure required close monitoring. Clinical and laboratory status/blood studies (hemoglobin, electrolytes, BUN, creatinine, calcium, phosphorus level and alkaline phosphates activity, parathyroid hormone), and radiography are needed to detect osteodystrophy. The present patient had decreased urinary, increased levels of BUN and serum creatinine, i.e. the usual manifestations in patients with renal failure and it was likely to be the end stage renal failure.

General therapeutic measures for predialysis phase: One of major aims of the clinician dealing with any chronic disease in childhood is to maximize growth and development potential. The pathogenesis of growth failure is complex and not yet fully understood. The defect however may be overcome by the use of exogenous human growth hormone (HGH). The complex metabolic and hormonal disturbances in uremia, and the proportionately increased energy and protein requirements of the infant and child tend to mitigate against the fundamentally important role of nutritional management. Regular clinical, nutritional, biochemical, and anthropometric review are necessary.

Growth failure can be marked with decreased glomerular filtration rate, but may be improved by appropriate dietary management, limiting protein intake may decrease renal perfusion and progression. Caloric intake can be enhanced by adding it to the diet without restricted of carbohydrate and fat as tolerated by the patients. High calorie (35 cal/kg/day) and low protein (0.6 – 0.8 g/kg/day) with high protein food like biological value (eggs and milk followed by meat, fish, and fowl).

Secondary hyperparathyroidism and metabolic bone disease, hyperphosphatemia may be controlled by low phosphate formula and by enhancing fecal excretion. Hypocalcemia may result from hyperphosphatemia, inadequate dietary intake and decreased intestinal calcium absorption due to a deficiency in the active form of vitamin D.

Recombinant human growth hormone therapy combined with optimal dialysis improves linear growth. If height is lower than -2 SDS it may be treated with recombinant human growth hormone. Human growth hormone has been successful in improving growth velocity in patients on CAPD. Enzyme protein to maintain hemoglobin within the range of 16 - 11 g/dl, (r - Hu EPO) is effective in patients on CAPD.

Indication for dialysis is based on plasma creatinine, in under two year old is 300 - 450 u mol/l (3.5 - 5 mg/dl) in the year old 450 - 600 mmol/l (5 - 6.8 mg/dl), in the 5 - 10 year old 600 - 800 mmol/l (6.8 - 9 mg/dl). The patient presented serum creatinine 5.2 mg/dl, uremic encephalopathy (vomiting and lethargy) and decreased urine. The management of children on peritoneal dialysis is complex and time consuming. Many of these difficulties are not so much technical dialysis problems but rather the problems of feeding, nutrition, psychomotor development, parental support and complication. Some modification of peritoneal dialysis equipment, parental is necessary for small infants and the circulation of small dialysis volume through a blood warming coil is important in order to avoid patient from hypothermia. The standard regime of three or four day time exchange at 4 - 5 hours intervals and one overnight exchange, has been applied successfully on infant of less than 5 kg. Dialysis volume should start at 26 ml/kg and should be increased toward 40 ml/kg if required. Prior to the dialysis, many children are receiving 10 or 15 % dextrose solution IV to prevent hypoglycemia. Most parents, usually16, become exceptionally well trained as dialysis experts for their own child. Continued treatment over many years is possible with chronic peritoneal dialysis (CPD). Chronic peritoneal dialysis (CAPD) beginning in infancy should now be accepted as worthwhile until successful renal transplantation can be achieved.
Complications inherent to the basic CAPD system - peritonitis, catheter related infections, ultrafiltration failure, inadequate dialysis and hernia are the reasons for more than 60% of drop out technique. Peritonitis is the most common acute complication of peritoneal dialysis and is the major cause of morbidity, temporary cessation of peritoneal dialysis and technique failure. Catheter exit site and tunnel infections frequently precede peritonitis and are important factors in the etiology of refractory or recurrent peritonitis. Pathogenesis of peritoneal dialysis associated peritonitis is multifactorial with range of causative organisms, and the clinical peritonitis depends on the balance between the pathogenicity defenses of the peritoneal cavity. Organisms may reach the peritoneal cavity by several exogenous and endogenous portals of entry, each route of infection being associated with a different spectrum of causative organisms. Contamination with skin or air borne commensals usually occurs as the time of an exchange but many also follow accidental disconnecting, changing of the transfer set and development of cracks in the tubing of catheter aseptic method to avoid infection. Culture of the dialysate effluent is important. CAPD patient with human immunodeficiency has increased the risk of peritonitis which is caused by *Pseudomonas aeruginosa* and fungus. In this case we found *Pseudomonas aeruginosa*. Most of the reported experiences of first line antibiotic therapy are various combination of aminoglycosides and second generation cephalosporins and vancomycin.

CONCLUSION

CAPD on CRF patient is the most essential management system should be introduced to save infants and children with chronic renal failure in Indonesia.

REFERENCES