Locally Improvised Acute Peritoneal Dialysis Using Nasogastric Tube and Fluids in a Resource-Limited Setting in Southern Nigeria: A Case Report

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ABSTRACT

Acute gastroenteritis remains a major reason for consults to the emergency room and can be complicated with electrolyte derangements, sepsis and acute kidney injury. We present a 24-month-old boy who had acute gastroenteritis with sepsis and who developed oliguric acute kidney injury. He had improvised acute peritoneal dialysis (PD) done using a nasogastric feeding tube and peritoneal dialysis fluids (Ringers Lactate and Dextrose intravenous fluids). He had 18 sessions of PD for 4 days with complete recovery of renal functions and discharged after 11 days. Children with gastroenteritis and sepsis that develop acute kidney injury should be offered improvised PD in resource-limited settings, as illustrated in this case report.

Keywords: *Acute kidney injury, improvised peritoneal dialysis, improvised peritoneal fluid, gastroenteritis*

INTRODUCTION

Acute kidney injury (AKI) is a sudden loss of kidney function in children that can occur as a result of a variety of illnesses. Septicemia, acute diarrheal disease, severe malaria and haemolytic uraemic syndrome are the main contributors.[1–4] AKI is a quiet but significant cause of morbidity and mortality among children in Sub-Saharan Africa.[1,3,5–7] AKI is usually treated based on the likely cause. Administration of fluids or blood products to expand the intravascular volume when due to a pre-renal cause, fluid restriction with avoidance of precipitating agents when AKI is established and due to structural damage to the kidneys (intrinsic renal) while correcting dyselectrolaemia or relieving obstruction of urinary flow (post-renal) are some of the principles of AKI management.[8]

If all of these procedures fail and renal function continues to deteriorate, life-saving interventions such as renal replacement therapy (RRT), which includes dialysis, become the only option.[9] The two main modalities available to sick children are acute peritoneal dialysis and haemodialysis. Many dialysis modalities are available to nephrologists in resource-rich settings, and which modality to adopt depends primarily on competence. However, in resource-constrained settings, the lack of such therapies is a serious barrier, resulting in increased mortality in children with AKI who require dialysis.[9]

Peritoneal dialysis (PD) is a straightforward and safe technique of renal replacement treatment.[10]

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The filtering membrane is the peritoneum in this case. Water and solutes are removed by the processes of diffusion, osmosis, and convection after cycles of instilling suitable constituent and osmotic concentration solutions into the abdomen and permitting timed contact with the peritoneal membrane.[10] In several therapeutic contexts, this has been demonstrated to improve AKI-induced dyselectrolytaemia, metabolic acidosis, and volume overload. It can be done in places where standard PD catheters and solutions are unavailable.[9] PD continues to be widely utilized in many resource-limited countries, despite the availability of newer modalities in resource-rich settings.[4] This article shows how improvised acute PD was carried out successfully on a child, in a place where there were very limited resources for the standard PD setup and procedure.

CASE PRESENTATION

A 2-year-old-male presented to the emergency room of a tertiary facility in Southern Nigeria with complaints of persistent vomiting and passage of watery stools of ten days and a fever of 8 days duration. Vomiting was non-projectile, vomitus consisted of recently ingested feeds at onset, and had 5-7 bouts per day with each bout described as moderate volume, non-bilious, and non-bloody. Vomiting subsided before the presentation. The stool was predominantly watery, moderate volume, mucoid and non-bloody. Had 7-9 episodes of bowel motions per day as against 1-2 times in his premorbid state. The patient has been on a normal family diet and eats 5-6 times a day, the meal is prepared and given by the mother, however, there is a history of poor hygiene as the mother does not always wash hands before preparing food or giving him his food. No other family member with a history of diarrhoeal illness. There is a history of increased thirst, and weakness; mother was unsure of a reduction in urine volume. Watery stool persisted till presentation. Fever was high grade, intermittent, and transiently relieved by paracetamol and tepid sponging. No history of rigours or seizures. The fever persisted till presentation. No history of cough, runny nose, ear discharge, dysuria, or hematuria.

Following the onset of symptoms, he was given water-based herbal concoctions and also over-the-counter oral drugs (metronidazole). However, with the persistence of symptoms, he was taken to a private hospital where he was admitted for three days and received intravenous medications and fluid. He was subsequently brought to this facility for expert care, as his clinical condition persisted.

At the presentation in the children's emergency room, the significant findings were that he was ill-looking, febrile (38.1C), severely pale, had non-pitting pedal oedema, angular stomatitis, had mild ascites with tender hepatomegaly (8cm below right costal margin, liver span 13cm), excoriated perineum, tachypneic (respiratory rate 40cpm), tachycardia (heart rate 140bpm), pedal oedema. SPO_2 = 98% in room air. His weight was 10kg (80% of expected), Length was 83cm (< -2 Scores), MUAC = 15cm, BSA= 0.48.

He was admitted for acute gastroenteritis with no dehydration, severe anaemia in heart failure and sepsis. A CBC done showed HB 7.3g/dl, PCV 20%, TWBC 12400/mm^3 (Neutrophils:64.9%, lymphocytes:28.06%, normocytes: 5.3%, eosinophils: 0.2%, basophils: 1.6%) platelets: 162,000/mm3, Malaria Parasite showed nil *plasmodium falciparum spp*. He was commenced on intravenous 5% Dextrose Ringers solution at 100% maintenance, intravenous artesunate, intravenous cefotaxime, gentamicin, tabs zinc and had an aliquot of blood transfusion.

On 2nd day of admission, he was observed to have low urine output and following urethral catheterization was discovered to be 0.8ml/kg/hr and clinical suspicion of AKI was entertained. Urinalysis done showed protein trace and blood 80 Ca Cells/uL. serum revealed findings in keeping with acute kidney injury: sodium 130mmol/L, potassium

2.6mmol/L, bicarbonate 16mmol/L, chloride 107mmol/L, urea 159mg/dl (26.47mmol/l) and creatinine 6.6mg/dl (583.57 umol/L). An abdominal ultrasound scan was not done due to financial constraints.

On the 3rd day of admission, the paediatric nephrologist was informed and significant review findings revealed generalised peripheral oedema, ascites demonstrable by shifting dullness, blood pressure was 85/50mmHg (< 90th percentile), no crepitations on auscultation of the lungs, output was 0.5ml/kg/hr, but the weight had increased to 12kg, after 48 hours of admission. A clinical diagnosis of Acute gastroenteritis with hypokalaemia and sepsis with AKI stage III was made. Continuous intravenous frusemide infusion at 2mg/kg/day and intravenous ciprofloxacin were commenced and gentamicin was discontinued. A repeat serum chemistry done showed sodium 135mmol/L, potassium 2.5mmol/L, bicarbonate 15mmol/L, chloride 113mmol/L, Urea 164mg/dl (27.3mmol/L), creatinine 6.5mg/dl (574.73umol/L), eGFR = 5.7ml/min/1.73m^2.

On the 4th day of admission, urine output improved to 0.8ml/kg/hr, however, the child's clinical condition remained unsatisfactory as he was noticed to become lethargic with worsening azotemia. Repeat serum chemistry showed: sodium 139mmol/L, potassium 2.4mmol/l, bicarbonate 12mmol/L, chloride 107mmol/L, urea 186mg/dl (30.96mmol), creatinine 6.3mg/dl (557.05umol/L). Therefore, with persistently elevated urea and creatinine as well as acidosis, a decision was made to commence acute peritoneal dialysis.

RESULTS

An improvised PD catheter - a size 10 nasogastric feeding tube to which some fenestrations were added manually was inserted by the surgeon using the modified Seldinger technique at the bedside (Fig 1). Improvised dialysis fluid using 25mls of 50% dextrose water and 500mls of Ringers lactate to make a 1.5% PD solution, to which IV ceftriaxone 500mg was added (Fig 2). No heparin was used, the bladder was catheterized before the insertion and hospital urine bags were used to improvise for the drain set. The peritoneal dialysis exchanges were carried out manually. A fill volume was initially at 10ml/kg, with an initial shorter and more frequent dwell times, then followed 20ml/kg fill volumes and 40minute dwell time, before draining for 20 – 30minutes. Dwell times were

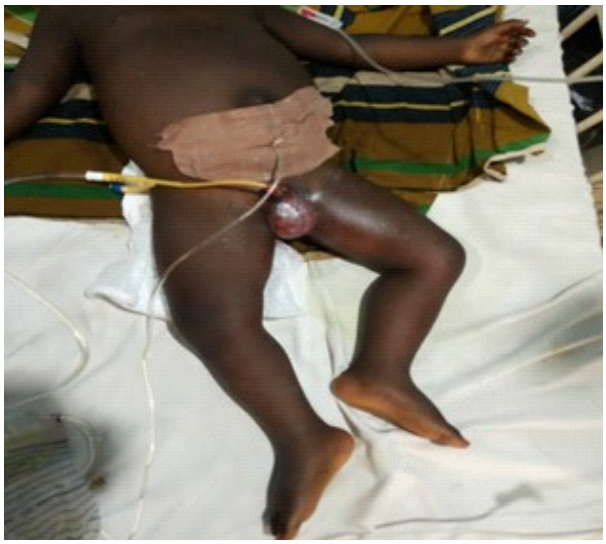

Fig 1: Modified Nasogastric tube inserted in the suprapubic region as the improvised catheter

Figure 2: PD fluid made at the bedside using 500ml of Ringer's lactate, 25ml of 50% dextrose water and 500mg of ceftriaxone

subsequently increased for up to 60 minutes before draining, depending on the child's clinical state. He had between 4 – 5 sessions daily and a total of 18 sessions in 4 days. Between the 5th and 8th day on admission and while being dialysed, his urine output improved and ranged from 2-3ml/kg/hr. The child's general condition improved. Repeat serum chemistries done during and after the acute improvised PD is shown in Table 1.

PD was complicated by improvised catheter blockage. However, with the evidence of improving renal function and resolved oliguria, PD was discontinued. The hypokalemia and acidosis were subsequently corrected using Half strength Darrow's solution. By the 11th day on admission, repeat serum chemistry had returned to normal (Table 1). The patient was discharged home and at the last follow-up visit had no residual renal impairment.

Table 1: Serial serum electrolytes, urea and creatinine levels during and post dialysis

Day of PD	Na (umol/L)	K (mmol/L)	Cl (mmol/L)	HCO_3 (mmol/L)	Urea (mmol/L)	Creatinine (mmol/L)
1	139	2.4	107	12	30.96	557.05
3	134	2.4	110	18	10.15	106.1
4	135	2.7	104	17	3.33	44.21
11th day on admission	138	4.0	103	20	1.49	35.37

DISCUSSION

Due to the relative ease of setup and administration, as well as the lack of need for highly trained personnel or specialized machinery, acute peritoneal dialysis remains the primary treatment option for acute kidney damage in resource-limited settings.[4] In children under the age of five, PD is the preferred method of renal replacement treatment. In children living in resource-limited situations, early diagnosis of AKI and the ability to offer peritoneal dialysis as a method of renal replacement therapy are important for preventing kidney-related deaths.

Although the peritoneum was first used as a dialyzing membrane in the 1940s, over the years, advancement in technologies have resulted in automated machines, standardized commercial PD solutions, and peritoneal catheters to be developed.[11] In resource-limited settings, these are not readily available, hence, paediatric nephrologists have had to resort to locally adapting resources to curb kidney-related deaths in children with acute kidney injury by improvising either the PD catheter or peritoneal dialysis solutions, or both.[3,12–14] This was the case in this patient we report. We provided peritoneal dialysis for this 24-month-old male with an improvised nasogastric tube which was inserted at the bedside using the Seldinger technique. The use of adapted nasogastric PD catheters has similarly been used successfully in other facilities in Nigeria.[12,15] Due to constraints of funds and unavailability of standard commercial PD solutions, locally made PD fluids were constituted for this acutely ill child. PD cycles were few per day because of the unavailability of doctors and nurses to perform more cycles. Although there were occasional leaks from the improvised catheter, the set-up worked. Our report is similar to what was also previously documented by other authors in Nigeria. Our patient, on the other hand, did not develop features of peritonitis, which was a potential consequence. This was likely due to the addition of antibiotics to the PD fluid and efforts to maintain asepsis within the constraints of available resources. It could have also

been because an improvised catheter was used for a very short duration. This was similar to the finding by Obiagwu et al[12] in Kano state and Ibitoye et al[16] in Sokoto state, both in North-Western Nigeria but contrasted with reports by Ademola et al,[15] in Oyo state, South-western Nigeria. The authors from Oyo state attributed the occurrence of peritonitis to the use of improvised nasogastric PD catheters and connectors, despite using standard commercial PD solutions and also the fact that PD was done manually by doctors who were also carrying out other duties. The only complication encountered was blockage of the improvised catheter which was probably due to a fibrin clot, as heparin was not used. Our findings were similarly reported in most of the studies where improvised PD catheters were used including nasogastric tubes,[12,15] foley's urinary catheters,[17] and intercostal drains[3] but were not observed in the study by Okoronkwo et al[13], in Abia State in south-eastern Nigeria where a double lumen haemodialysis catheter and in the report by Ikpeme in Akwa-Ibom State, South-south Nigeria where a size 16 silicone catheter was used as the adapted PD catheter. As reported by most authors that promptly utilized improvised catheters, the outcome was good with the recovery of renal function in a considerable proportion of patients.[18,19]

This study adds to the growing body of data that improvised acute PD is a cost-effective RRT option for paediatric AKI therapy in resource-limited settings and when performed with caution, the use of improvised dialysis fluids and catheters where available can reduced deaths from AKI.

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