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Case Report

The use of an Improvised Nasogastric Tube as a Peritoneal Dialysis Catheter and Challenges of Adaptation-A case report

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Abstract

Background: Paediatric dialysis for acute kidney injury (AKI) in developing countries may be a challenge because of limited access to standard gadgets. Adapted gadgets such as nasogastric tubes have been used to provide peritoneal dialysis (PD)in such settings. The nasogastric tube is usually inserted by the trocar or surgical technique which is cumbersome. We describe passage of the nasogastric tube for PD at the bedside by a technique that is considered easier than the trocar or surgical method.

Case report and Treatment: A two-and-a-half-year-old girl presented with oliguric AKI secondary to malaria and intravascular haemolysis with suspected uraemic encephalopathy. Her serum creatinine on admission was 9.4mg/dl. A nasogastric tube was adapted as PD catheter, and was inserted by the modified Seldinger technique on post-admission day 1.

Results: She received 39 cycles of PD over 5 days. Complications of PD included catheter outflow obstruction on post admission day 2 on account of which the catheter was replaced. She also developed *Klebsiella pneumoniae* peritonitis and on account of which the procedure was discontinued on post admission day 6. She regained normal urine output on post admission day 7. She was managed with iv meropenem based on the antibiotic sensitivity pattern with resolution of peritonitis. She showed significant improvement and her serum creatinine on post admission day 15 was 0.5 mg/dl.She was discharged home on post admission day 18.

Conclusion: Nasogastric tube, adapted as PD catheter and inserted by modified Seldinger technique may be life-saving in patients with AKI in developing countries.

Keywords: Peritoneal dialysis, acute kidney injury, nasogastric tube, developing countries, children

Introduction

Acute kidney injury is an important cause of morbidity and mortality among children in low resource settings. A prospective study found AKI defined by KDIGO serum creatinine criteria in about a 3rd of all children who had serum creatinine estimation and about 8.3% of admissions to the emergency room.[1] Another study of AKI among patients with malaria, gastroenteritis and pneumonia in Uganda found AKI incidence of 13.5%.[2] While yet another prospective study among patients with severe malaria found prevalence of 45.5% among patients with severe malaria.[3]

Renal replacement therapy may be lifesaving and is frequently indicated in children with AKI. Previous studies showed that renal replacement therapy may be indicated in about 20-66% of children with acute kidney injury.[1,4-7] and lack of dialysis access has been identified as an important cause of mortality in children with acute kidney injury in many centres in low resource settings.[4-9].

Modalities for renal replacement therapy in developed countries include continuous renal replacement such as continuous venovenous haemodiafiltration, haemodialysis and peritoneal dialysis.[10-12] However challenges with provision of renal replacement in developing countries include cost and access to standard paediatric size consumables for paediatric dialysis.[4-9] Peritoneal dialysis is particularly suited for renal replacement therapy for paediatric acute kidney injury in developing countries because the technique is simple, and it is not as technologically demanding as the other modalities.

Some of the adaptations that have been utilized include use of chest tubes, Redon drains, suprapubic catheters, temporary haemodialysis vascular catheters, the Foley catheter and nasogastric tubes as peritoneal dialysis catheter.[1,13-20] The nasogastric tube in particular, when it is adapted as peritoneal dialysis catheter, is usually inserted by a trocar or surgical technique and some paediatric nephrologists may find these approaches cumbersome.[18,21] We therefore describe the insertion of the nasogastric tube for peritoneal dialysis by the modified Seldinger technique,[13,22-24] a technically simple procedure, and the outcomes in terms of associated complications and patient survival in a 2-and-a-half-year-old girl with oliguric acute kidney injury.

Case report

A 2 and a half year old girl presented with fever of 6 days', jaundice and reduction in urine output of 4 days', passage of dark coloured urine and reduced activity of 3 days' duration. She had one episode of generalized tonic-clonic convulsion, which occurred 5 days before presentation. The convulsion lasted for about 30 minutes and she had no other history of seizures. She was taken to a peripheral health facility where she was admitted for two days and had intravenous ceftriaxone, metronidazole, furosemide, intramuscular artemether and oral medications but she was noted to have become drowsy, and started vomiting. She was suspected to have cerebral malaria and was referred for further management. Her developmental history was normal. At presentation she was mildly pale, not jaundiced, had periorbital oedema, but no pedal oedema. She weighed 12.3kg, her length was 88 cm, temperature was 36.6^{0}C, and she had an oxygen saturation of 98% in room air. She had respiratory rate of 36/minute, she was not dyspnoeic and her breath sounds were vesicular. She had normal volume regular pulses, blood pressure was 90/50mmHg, and heart rate was 100/minute, she had normal heart sounds. Her abdomen was full and soft, her liver was palpable 6cm below the costal margin, soft smooth, non-tender. She had normal bowel sounds and she was lethargic.

Rapid diagnostic test for malaria carried out at presentation was positive, while her blood film for malaria parasite was negative. Her packed cell volume was 21%. Her urinalysis showed blood 2+, protein 1+, but the other parameters on dipstick urinalysis were normal. Her urine microscopy showed red blood cells: 4386/ high power field (HPF), white blood cells 117/HPF, but there was no growth on urine culture. Her full blood count showed white blood cell 12,650/mm^3, neutrophil 56%, lymphocyte 27%, monocyte 16%, haematocrit 19%, and platelet count was 38,000/mm^3. Her haemoglobin electrophoresis was A. Her electrolyte urea and creatinine showed serum sodium 123 mmol/L, potassium 3.1mmol/L, chloride 92 mmol/l, bicarbonate 9 mmol/L, urea 398 mg/dl and creatinine of 9.4 mg/dl. Her abdominal ultrasound done post admission day 1 showed both kidneys normal in position and outline. The right kidney measured 8.3cm x 4.1cm, while the left kidney measured 7.8cm x 4.3cm. They both showed increased parenchymal echogenicity higher than that of the liver and spleen on the right and left respectively with preservation of their corticomedullary differentiation and normal central sinus echoes consistent with bilateral grade 2 renal parenchymal disease. No pelvicalyceal or ureteric dilatation was seen. The urinary bladder was under filled making its assessment difficult.

The initial diagnosis was acute kidney injury (AKI) secondary to intravascular haemolysis and malaria with suspected uraemic encephalopathy. She was oliguric with urine output of 0.2ml/kg/hr. She had boluses of 20mls/kg (250mls) of normal saline twice at presentation with no improvement in her urine output. She was started on intravenous (i.v) artesunate and i.v ceftriaxone. She was commenced on peritoneal dialysis on account of the history of seizure, the lethargy, oliguria and marked azotaemia on post admission day 1.

A nasogastric tube was adapted as peritoneal dialysis catheter with additional fenestrations made at the distal end of the tube. The tube was inserted by the paediatric nephrology senior registrar using the modified Seldinger technique.[13,22,24] The peel away sheath, and guidewire used for the insertion were obtained from a tunneled vascular catheter kit. A three-way tap was used to connect the outside end of the nasogastric tube to the distal end of a burette that is connected to the peritoneal dialysis fluid. (Fig 1 shows nasogastric tube adapted as peritoneal dialysis catheter, Fig 2 illustrates the set up for peritoneal dialysis) The drain for the peritoneal dialysis effluent was connected to the third opening of the three-way tap. The peritoneal dialysis fluid exchanges were carried out manually. Commercially available continuous ambulatory peritoneal dialysis fluid (CAPD) were used for the exchanges with a dwell volume of 180 mls and dwell time of 45 minutes. She was placed on intravenous fluids at ¾ of daily maintenance requirements when peritoneal dialysis was commenced. Peritoneal dialysis catheter outflow obstruction was noted on post admission day 2 and the 'catheter' was replaced with another adapted nasogastric tube using the same technique.

The second 'catheter' was passed by the paediatric nephrologist. She had a total of 39 cycles of peritoneal dialysis over a period of 5 days. The serum creatinine results while she was on peritoneal dialysis were as follows 9.1 mg/dl (post admission day 3), and 8.3 mg/d (post admission day 4) while serum urea results were 278 mg/dl (post admission day 3) and 212mg/dl (post admission day 4) The peritoneal dialysis was stopped on post admission day 6 on account of suspected peritonitis based on the recurrence of fever and the finding of cloudy peritoneal dialysis effluent and her antibiotic was changed from i.v ceftriaxone to i.v ciprofloxacin. Her urine output at the time of discontinuation of peritoneal dialysis was 0.6ml/kg/hr.

However, her urine output became normal (1.2ml/kg/hr) a day post discontinuation of peritoneal dialysis (post admission day 7), while her serum creatinine and urea on that day (post admission day 7) were 7.0 and 195 mg/dl respectively. The fever however persisted in spite of the i.v ciprofloxacin. Culture of the peritoneal dialysis effluent grew *Klebsiella pneumoniae*, sensitive to amikacin, meropenem but was resistant to augmentin, gentamicin, ceftriaxone, ceftazidime, levofloxacin and piperacillin tazobactam. Intravenous ciprofloxacin was changed to i.v meropenem on post admission day 1, she went on to have 7 days of i.v meropenem. Her temperature became normal 2 days after commencement of i.v meropenem (on post admission day 12). Her serum urea and creatinine recorded on post admission day 15 were 48 and 0.5mg/dl respectively. she was discharged home on post admission day 18 and was followed up in the clinic. She showed sustained clinical improvement. Her serum electrolyte urea and creatinine results three weeks post discharge were sodium of 135mmol/l, potassium of 4mmol/l, chloride of 98mmol/l, bicarbonate of 22mmol/l, serum urea of 15mg/dl, serum creatinine of 0.4mg/dl.

Discussion:

We provided peritoneal dialysis to a 2and a half year old girl with adapted nasogastric tubes inserted at the bed-side by the modified Seldinger technique. We adapted the introducer and peel away sheet from the tunneled vascular catheterization kit, and followed the procedure for insertion of the standard Tenckhoff catheter for peritoneal dialysis by modified Seldinger technique.[13,22,24] This method of catheter insertion can be done at the bed-side by the paediatric nephrologist or a trained resident and may be a much easier technique than the surgical technique or the trocar technique, and may further increase the uptake for peritoneal dialysis in our country.

Adapted catheters may be lifesaving, but may also carry a higher rate of complications than standard catheters.[11,19,21] Complications in our patient included catheter obstruction necessitating replacement of the catheter. Catheter outflow obstruction is seen less frequently when soft catheters such as the Tenckhoff or Cook catheter (Cook Medical Inc, Bloomington, IN, USA) are used for peritoneal dialysis compared to rigid or adapted catheters.[14,21,23,25-27] However in Nigeria, Tenckhoff and Cook catheters are very expensive and most parents or caregivers are not able to afford them. An additional complication in our patient was peritonitis.

The use of adapted gadgets may be associated with higher rates and earlier onset of clinical peritonitis. There is wide variation in the reported rates of peritonitis for patients receiving acute peritoneal dialysis and can range from 1-78.3%.[28,29] Some of the factors which influence rates of peritonitis include the definition of peritonitis, type of peritoneal dialysis catheter, the method of insertion of catheter, the rate of catheter manipulation, the use of open or closed peritoneal dialysis drainage systems, formal PD training of the staff carrying out the exchanges, duration of peritoneal dialysis, hypoalbuminaemia and possibly the use of automated peritoneal dialysis.[12,14,25-28] Replaced catheters also appear to be associated with episodes of peritonitis more frequently than primary catheters.[26]

The occurrence of peritonitis in our patient may be due to the use of peritoneal dialysis catheter adaptations such as the nasogastric tube and the three way tap rather than standard catheters such as the Tenckoff catheter with the extension tubing. The drainage systems were also adapted. We did not use tunneled access for the catheters as we did not use the standard Tenckhoff catheter. An additional challenge which would have increased the risk of peritonitis is the manual PD exchanges by doctors who do shifts on the patients while concurrently carrying out other assignments. Acquiring and utilizing an automated PD machine may reduce the infection rate.[13]Presently,we also do not yet have a formal training program for the staff who carry out PD exchanges. In spite of these limitations we were able to carry out renal replacement therapy for acute kidney injury in our patient for about 5 days. Her successful outcome may have been partially influenced by the aetiology of acute kidney injury. Malaria is a recognized cause of reversible acute kidney injury, however, she might not have survived if she did not receive renal replacement therapy at all. Use of standard of care catheters and gadgets, training in the placement of tunneled Tenckhoff catheters, and training of nurses in carrying out the exchanges may help to reduce rates or delay onset of complications in paediatric patients on acute PD.[30] Sustained advocacy should be made to government and non-governmental societies to support access to standard peritoneal dialysis equipment and training for the management of children with acute kidney injury in developing countries.

Conclusion: Our case report illustrates that nasogastric tube, adapted as peritoneal dialysis catheter, may be passed by the modified Seldinger's technique and used in the successful management of children with acute kidney injury when the standard peritoneal dialysis catheter is not available. However, attention needs to be paid to the early detection and management of potential complications. Continued advocacy should be made to government and NGOs to support access to standard gadgets for peritoneal dialysis for paediatric patients.

Authors' contributions

ADA- Wrote the first draft, and reviewed the manuscript, AOA- Critically reviewed the manuscript, RMA- Conceptualized the paper and reviewed the manuscript, All the authors approved the final draft.

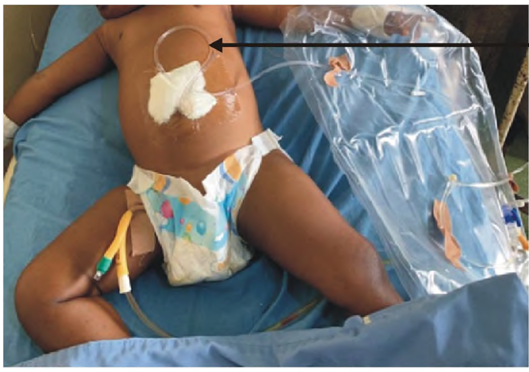
Nasogastric tube adapted as Peritoneal dialysis catheter

Fig 1: Peritoneal dialysis for acute kidney injury with the use of adapted peritoneal dialysis catheter inserted by modified Seldinger technique (nasogastric tube)

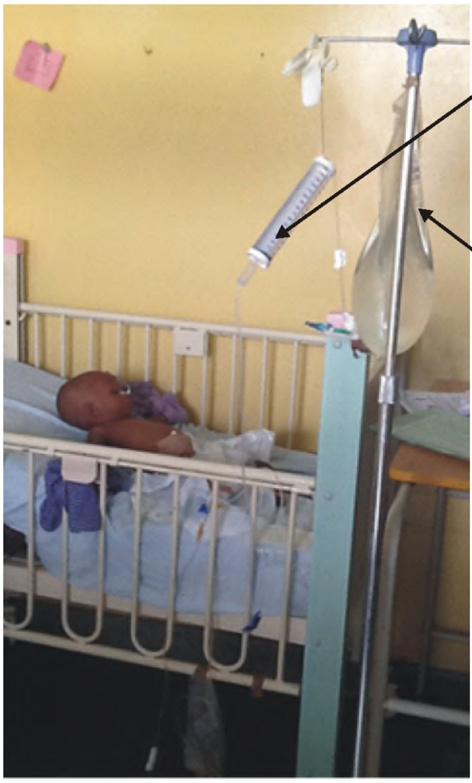

Fig 2: Picture illustrating the peritoneal dialysis set-up

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