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Case Report

Peritoneal Dialysis in Acute Renal Injury in Neonates with Nonimmune Hydrops Fetalis

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Abstract

Acute kidney injury (AKI) in newborns may be pre-renal, intrinsic, post-renal and is the cause 0.4-3.5% of hospital admissions, and up to 8% of admissions to a neonatal intensive care unit (NICU). Most newborns with AKI are preterm or critically ill babies, having serious problems. Nonimmune hydrops fetalis is a rare condition contributing significantly to neonatal and perinatal mortality. Peritoneal dialysis (PD) is a renal replacement therapy for children at all ages including neonates with acute renal injury and can be performed in the neonatal intensive care unit. There is less experience in PD in the critically ill newborns. The aim of the report is to discuss the use of PD in two cases with nonimmune hydrops fetalis. We report our experience with acute renal injury secondary to non-immune hydrops fetalis (NIHF) in two critically ill neonates managed with PD, a safe and effective treatment in the neonatal population.

Keywords: *Peritoneal Dialysis; Acute Renal Injury; Neonates; Nonimmune Hydrops Fetalis*

Introduction

Acute kidney injury (AKI) in newborns can be pre-renal, intrinsic and post-renal. AKI is the cause 0.4 to 3.5% of hospital admissions, and nearly 8% of admissions to a neonatal intensive care unit (NICU). Most newborns with AKI are preterm or critically ill babies, having serious problems [1-2].

Nonimmune hydrops fetalis is a rare condition contributing significantly to neonatal and perinatal mortality. These infants must receive prompt, aggressive therapy to survive. Peritoneal dialysis (PD) is a renal replacement therapy for children at all ages including neonates with acute renal injury and can be performed in the neonatal intensive care unit (NICU). Peritoneal dialysis allows slow removal of fluid and solute, while avoiding hemodynamic instability. Acute PD is relatively easy to perform, does not require heparinization and also the neonate does not need to be hemodynamically stable [3-6]. There isn't so much experience in PD in the critically ill newborns. Our aim with this case report is to discuss the use of PD in two critically ill neonates with nonimmune hydrops fetalis.

Patients

We report our experience with acute renal injury secondary to non-immune hydrops fetalis (NIHF) in two neonates managed with PD.

Case 1: A 23-year-old gravida 2-para 2-abortus 1 woman was admitted at 36 weeks and, an emergency cesarean section was performed because of fetal distress. The mother had a prenatal diagnosis of hydrops fetalis in the last trimester of her pregnancy. The male neonate was severely asphyxiated at birth. The Apgar scores were 0, 0 and 3, at the 1st, 5th and 10th minutes, respectively. The birth weight was 2850g. After successful resuscitation including intubation, he was placed on high frequency oscillatory ventilation with 100% oxygen

and inotropic support was started. The infant had a generalized edema, abdominal distention with ascites and no anomalies on the physical exam. No identifiable cause could be determined for nonimmune hydrops fetalis. The blood types were A Rh (+), A Rh (-) for the mother and infant, respectively. Selective head cooling was started for the treatment of perinatal asphyxia at the 5th postnatal hour. At the 18th hour the urine output was 0.5 mL/kg/hour, uric acid was 7.9 mg/dL, creatinine 1.66 mg/dL, blood urea nitrogen (BUN) 9 mg/dL. The infant had a bleeding diathesis. [INR: 2, partial thromboplastin time (PT): 25.50 seconds, activated partial thromboplastin time (APTT): 32.60 seconds, platelet count: 83000/mm^3]. The infant had generalized edema with progressive weight gain and worsening respiratory functions in the 2nd postnatal day. The urine output was 0.1 mL/kg/hour. Peritoneal dialysis was postponed for severe bleeding. On the 4th day, he was still on supportive treatment with a urine output of 0.3 mL/kg/hour, uric acid 10.7 mg/dL, creatinine 2.2 mg/dL, BUN 26 mg/dL. His blood pressure was 68/42 (mmHg). On the 5th day, erythrocyte and platelet transfusions, cryoprecipitate, dopamine, dobutamine and antibiotic infusions was given and also PD was started with the dialysate solution of %1.36 dextrose concentration was used. Initial exchange volumes were 10 mL/kg, which is subsequently increased to 45 mL/kg. The vital signs including pulse and blood pressure, temperature, electrolytes were monitored closely. On the 9th day of PD, the urine output was 5 mL/kg/hour, uric acid 2 mg/dL and creatinine 0.26 mg/dL, PD was discontinued.

Case I	Urine output (mLkg/h)	Uric acid (mg/dL)	Creatinine (mg/dL)	Blood Urea Nitrogen (BUN) (mmHg)
18th hour	0.5	7.9	1.5	9
2nd day	0.1	9.1	1.8	15
4th day	0.3	10.7	2.2	26
16th day	5.0	2.0	0.2	23

Case 2: An infant with a birth weight of 2580g was born by cesarean section at 33 weeks of gestation, to a 28-year-old gravida 1-para 1 mother. She had a prenatal diagnosis of hydrops fetalis in the last trimester of the pregnancy. At birth, the infant was cyanotic and hypoxic, was successfully resuscitated including intubation. The Apgar scores were 3 and 6, at the 1st and 5th minutes, respectively. Both the infant's and the mother's blood types were 0 Rh (+). The infant was diagnosed as NIHF and had compaction cardiomyopathy on the echocardiogram. On the 33rd day of her NICU stay, she was consulted to the pediatric nephrologist for a decreased urine output and hyperuricemia (serum uric acid level: 8.6 mg/dL, creatinine: 0.21 mg/dL, BUN :16 mg/dL). Renal ultrasound was normal. While she was on medical treatment, including furosemide, she progressively gained weight and on the 39th day of life, her serum uric acid level and creatinine levels increased to 11.6 mg/dL and 1.22 mg/dL respectively. Her platelet count was 57000/mm^3, INR,1.7, PT 19.5 seconds, and APTT 32.5 seconds. The renal functions began to deteriorate progressively thereafter. On the 45th day, she was anuric and the creatinine level increased to 2.54 mg/dL. A peritoneal dialysis of 10 mL/kg exchange volumes with %1.36 dextrose solution was started. After the 4th day of dialysis, her uric acid level was 5.5 mg/dL and creatinine level 1.98 mg/dL. PD exchange volume was increased to 45 mL/kg. On the 9th day of the PD, when the urine output was 0.5 mL/kg/hour; uric acid 3.8 mg/mL, creatinine 0.69 mg/mL PD was discontinued.

Case II	Urine output (cc/kg/h)	Uric acid (mg/dL)	Creatinine (mg/dL)	Blood Urea Nitrogen (BUN) (mmHg)
33rd day	1.9	8.6	0.2	16
39th day	0.2	11.6	1.2	78
45th day	no	8.4	2.5	106
49th day	0.2	5.5	1.9	67
56th day	0.5	3.8	0.6	53

Discussion

Regardless of etiology, NIHF is a serious condition with high mortality and morbidity. 90% of hydrops cases in the western world is of non-immune origin. The basis of the disorder is an imbalance in the regulation of fetal fluid movement between the vascular and interstitial space [6]. In recent years, management of HF has improved. However, NIHF is still associated with an overall perinatal mortality rate of 50 to 98% [7-10]. The incidence of acute renal injury is 8% among neonates admitted to NICU. The decision to begin dialysis is crucial, because in neonates, the shorter the time from the ischemic insult to the dialysis, the higher the survival rate [11]. The need for PD should be early recognized, hence it may reduce the mortality in neonates with AKI in NICU [4]. Peritoneal dialysis is effective in the newborn period in the management of metabolic disturbances as well as renal injury [12]. Reznik., *et al*. reported fifty neonates and children with acute renal injury treated with acute PD successfully. The mortality however was 50% reflecting the impact of underlying disease [14]. Caldwell., *et al*. reported two NIHF cases successfully treated with PD. Using PD in especially NIFH neonates whose urine output is below 1cc/kg/hour is strongly recommended by the authors [16]. In another study reported by Hakan., *et al*. a 7-year single-center experience with PD in NICU was documented. Peritoneal dialysis in neonates with acute renal injury was reported to be safe and effective renal replacement therapy. They further pointed out that PD is the most inexpensive of all renal replacement therapies in this group of patients [4]. Other advantages of PD included technical simplicity, avoiding anticoagulation or placement of a central venous catheter, and excellent tolerance in hemodynamically unstable patients. On the other hand, the major limitations of PD in the intensive care units are the slow and inefficient nature of the treatment, which preclude its use in children with severe, life-threatening peritonitis, pulmonary edema, severe hyperkalemia and the need for surgery to place a PD catheter [17,18].

In both cases reported here, there were life-threatening conditions such as severe bleeding diathesis. PD had to be started after medical stability is achieved, which were time keeping. Overall, PD was useful in recovery from acute kidney injury in both of our cases. Unfortunately, both cases were lost due to other medical problems after renal functions recovered.

In conclusion, early PD can be performed even in critically ill neonates including NIHF, PD is a safe and effective treatment in the neonatal population.

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