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Peritoneal dialysis after pediatric cardiac surgery: benefits and risks

Abstract

Introduction: Neonates and infants having surgical repair for congenital heart disease are at risk of developing acute kidney injury (AKI). Our objectives were to determine surgeries most associated with AKI, to compare effect of peritoneal dialysis (PD) and conventional treatment, and to study the risk factors associated with PD mortality.

Materials and methods: Records of Children who underwent cardiac surgery from November 2016 until December 2017 were reviewed. Clinical and biological effects of PD and conventional treatment were compared. In PD group, subgroups of survivors and non-survivors were compared to study risk factors for mortality associated with PD. We compared mortality between early and late PD (more than 24 hours after surgery).

Results: 134 children were operated during the study period. 27 (20%) developed AKI and 9 of those (33%) received PD. Arterial switch was most associated with AKI (71.4%). PD had better effect in decreasing creatinine and blood urea nitrogen (BUN) levels after 48 hours treatment than conventional treatment (creatinine: 28.8±14.5 vs 7.5±12.1micromol/L, p=0.003) (BUN: 3.08±2.1 vs 0.91±1.5mmol/L, p=0.017). In PD group, survivors (n=5) had higher mean arterial pressure in the 6 hours prior to PD that non-survivors (n=4) (55.3±9.6 vs 40.0±3.6 mmHg, p=0.019). Survivors had also higher pH 24 hours after PD (7.37±0.03 vs 7.31±0.02, p=0.014), better creatinine variation (-3.6±5.8 vs 29.0±13.0micromol/L, p=0.02), and better diuresis improvement (4.4±3.2 vs 0.23±1.1ml/kg/h, p=0.039). There was no mortality difference between early and late PD. There were no major complications with PD.

Conclusion: PD is safe for AKI after heart surgery. It has better outcome on BUN and creatinine levels. PD mortality is higher with low cardiac output, persistence of acidosis and absence of creatinine or diuresis improvement.

Keywords: acute kidney injury, peritoneal dialysis, congenital heart disease, children, mortality

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Abbreviations: PD, peritoneal dialysis; AKI, acute kidney injury; PICU, pediatric intensive care unit

Introduction

Incidence of acute kidney injury (AKI) in pediatric intensive care units is around 3 to 5%, with 20 to 30% mortality.[1] In order to decrease AKI morbidity, renal replacement therapy techniques are used. Peritoneal dialysis (PD) is the preferred choice, especially in children under 6years of age.[1,2] Advantages of PD are a minimal cost compared to hemodialysis, ease of technique and hemodynamic tolerance in children.[1,3] Furthermore, PD does not need anticoagulation or central vascular line.[4,5] PD applies short cycles in which low volume of dextrose solutions are used. Congenital heart disease represents the main cause of congenital abnormalities.[6] With the development of surgical techniques, children can now benefit from pediatric cardiac surgery. However, increase in pediatric cardiac procedures is accompanied with an increase in post-operative complications rate, particularly renal complications such as AKI. PD use for post-operative AKI treatment has been the subject of many studies in order to establish its security and efficacy.[4,7–15] After cardiac surgery, mortality rate was between 6 and 57% in case of AKI, and between 11 to 46% in case of PD use.[4,9–12,14–17]

The objectives of this study were the following:

A. Comparison of clinical and biological effects between PD and conventional treatment (diuretics, hydration…)

B. Identification of high risk procedures

C. Identification of mortality risk factors for patients who receive PD

D. Comparison of mortality between patients who received early PD (in the first 24hours after surgery) and patients who received late PD (more than 24hours after surgery).

Materials and methods

We conducted a retrospective study between November 2016 and December 2017. We registered demographic data of operated children, type of surgery and complications of PD. Children included in the study are those who had cardiac surgery and who developed AKI later. AKI was defined as a 50% elevation of creatinine level in comparison to baseline levels, or oliguria with diuresis below 1ml/kg/h for more than 6 hours. No exclusions were made upon type of surgery, age or sex of children. We compared children receiving conventional treatment and PD. We registered hospital and PICU (Pediatric

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Intensive Care Unit) length of stay, duration of mechanical ventilation and vasopressor drugs use. In addition, we registered improvement of creatinine and blood urea nitrogen (BUN) levels after 24 and 48hours of treatment, improvement of diuresis and hydric balance after 24hours of treatment (both expressed in ml/kg/h) and the time needed for creatinine normalization. We also compared survivors and non-survivors among children who received PD. We studied the following variables to identify risk factors associated with mortality: age and weight of children, duration of cardiopulmonary bypass and aortic clamp, timing and duration of PD, creatinine level at the initiation of PD, pH at start of PD and pH 24hours after PD, improvement in creatinine levels and diuresis after 24 hours of treatment, and finally the low cardiac output syndrome defined by the measurement of mean arterial pressure in the 6 hours prior to PD.

We used pediatric catheters for PD, type Argyle TM (CovidienTM), that were placed during or after the surgery. Dialysate solutions were standard commercial solutions (DIANEAL Baxter®) with dextrose concentrations of 1.36%, 2.27% and 3.86%. Dwell volume was between 10 to 20 ml/kg. Cycles were 1hour each with a dwelling time of 30minutes. 500units of Heparin were added to every liter of dialysate solution. We analyzed mortality rate and compared the difference in mortality in case of early introduction of PD versus late introduction of PD. We also noted survival rate at 1month and 3months follow-up after PD. We used SPSS version 22.0 for the statistical analysis and presented the results as mean and standard deviation. We used Student test to compare effects of conventional treatment and PD. Same test was used to assess mortality risk factors associated with PD when comparing groups of survivors and non-survivors among children who received PD. Finally, we used Fisher test to compare mortality between children who received early and late PD.

Results

During the time of the study, 134 children were operated, with 72boys (53.7%) and 62girls (46.3%). 27children presented post-operative AKI with a rate of 20%. Among these children, nine needed PD (33.3%). Global mortality rate was 4.5%. Mortality in case of AKI development was 14.8% and mortality in case of PD treatment was 44.4%. Table 1 shows the classification of patients according to surgery type and incidence of AKI. Clinical and biological outcomes of PD and conventional treatment are presented in Table 2. Children with conventional treatment needed vasopressors for a mean time of 143.2hours while patients with PD received vasopressors for 388.2 hours. Application of Student test did not show a significant difference with a p-value of 0.22. The same test was used to compare the time for creatinine normalization in both groups. It did not show any difference with a p-value of 0.74 (92.3hours with conventional treatment and 82.4hours with PD). Table 3 shows the factors associated with mortality when compared between survivors and non-survivors among patients receiving PD. In addition to the factors presented in table 3, cardiopulmonary bypass time was 151.33±114.02 minutes for survivors and 165.0±16.09 minutes for non survivors (p=0.84). Aortic clamp time was 74.0±37.16 minutes for survivors and 88.75±52.19 minutes for non-survivors (p=0.7).

Mortality rate in children who received early PD was 50% while it was 40% in patients receiving late PD. The application of Fisher test did not show any difference between the two groups. Table 4 shows the distribution of children according to the timing of PD and mortality. Follow-up of patients, who received PD and survived, showed a survival rate of 100% after 1 and 3months. As for complications, we noted only metabolic complications, with three cases of hyperglycemia and three cases of hypokalemia.

Table 1 Classification by type of procedure and acute kidney injury incidence

Type of surgery	Sample	AKI (n)	AKI Percentage (%)
Ventricular Septal Defect	28	3	10.7
Tetralogy of Fallot	18	2	11.1
Atrioventricular Septal Defect	12	2	16.7
Atrial Septal Defect	9	1	11.1
Glenn	9	1	11.1
Blalock	8	3	37.5
Banding	7	2	28.6
Crafoord	6	1	16.7
Pulmonary atresia	4	2	50.0
Persistent Ductus Arteriosus	4	0	0.0
Total Anomalous Pulmonary Venous Return	4	2	50.0
Fontan	3	2	66.7
Infundibular Stenosis	3	0	0.0
Arterial Switch	7	5	71.4
Aortic Stenosis	2	0	0.0
Arc interruption	2	0	0.0
Rastelli	2	0	0.0
Ablation of Prosthesis	2	0	0.0
Other	4	1	25.0
Total	**134**	**27**	

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Table 2 Comparison of both treatments effects on clinical and biological outcomes

Factors	Conventional (n=18)	PD (n=5)	P-Value
Hospital Stay (days)	21.3±10.4	38.2±29.9	0.05
PICU Stay (days)	10.7±3.7	35.2±29.4	0.002
Mechanical Ventilation (hours)	113.3±108.9	312.2±247.5	0.013
BUN variation after 24h (mmol/L)	0.2±1.4	0.22±2	0.98
BUN variation after 48h (mmol/L)	-0.91±1.5	-3.08±2.1	0.017
Creatinine variation after 24h (micromol/L)	-2.8±9.3	-3.6±5.8	0.86
Creatinine variation after 48h (micromol/L)	-7.5±12.1	-28.8±14.5	0.003
Diuresis Variation after 24h (ml/kg/h)	2±2.5	4.4±3.2	0.082
Hydric balance after 24h (ml/kg/h)	-2.3±3.1	-4.4±3.8	0.22

Table 3 Comparison of mortality risk factors between groups of survivors and non-survivors treated with peritoneal dialysis

Factors	Survivors (n=5)	Non-Survivors (n=4)	P-Value
Age (months)	6.3±5.9	4.6±7.6	0.72
Weight (kg)	5.1±1.9	4.0±1.3	0.38
Timing PD (hours)	45.0±51.0	74.3±82.9	0.53
PD Duration (hours)	124.4±199.0	67.5±100.4	0.621
Mean Arterial Pressure (mmHg)	55.3±9.6	40.0±3.6	0.019
pH at start of PD	7.34±0.1	7.30±0.2	0.66
pH 24h after PD start	7.37±0.03	7.31±0.02	0.014
Initial Creatinine Level(micromol/L)	77.8±43.8	55.0±14.0	0.323
Creatinine Variation after 24h (micromol/L)	-3.6±5.8	29.0±13	0.02
Diuresis Variation After 24h (ml/kg/h)	4.4±3.2	0.23±1.1	0.039

Table 4 Distribution of children according to peritoneal dialysis timing and mortality

	Early PD	Late PD
Survivors	2	3
Non-Survivors	2	2

Discussion

AKI is one of the main complications of pediatric cardiac surgery. AKI rate in our study was 20% and was comparable to rates reported by other authors and varying from 3% to 26%.[4,7,9,10,12,17,18] Variability in AKI rate depends essentially on the definition used in each study. Using dynamic variation of creatinine level may limit the selection bias that can be caused by using a cutoff to define AKI, since some children can have slightly elevated creatinine levels prior to surgery (like neonates) and considered later to have AKI. Using oliguria for the definition of AKI is a good indicator, but oliguria range varied among studies from 0.5ml/kg/h to 1ml/kg/h. [4,9–11] We chose to use the cutoff of 1 ml/kg/h for the definition of oliguria to detect the occurrence of AKI at its beginning. We proved in our study that AKI rate depends on the type of procedure performed. Arterial switch is most commonly complicated with AKI, as others authors have noted, especially that it is performed in neonates who are vulnerable to hemodynamic instability.[9,10,14,15,19] In this case, young age of children and their low weight are favoring factors for AKI development.

Length of hospital stay, length of PICU stay, and duration of mechanical ventilation and vasopressor drugs use were shorter in the conventional treatment group. This can be explained by the complexity of surgeries in patients who needed PD. Plus, hospital and PICU stay are influenced by complications other than AKI. Advantages of PD compared to conventional treatment were noted with improvement of BUN and creatinine levels after 48 hours of treatment. This can be explained by active filtration across the PD membrane, while the renal function is recuperated. Many factors were determined as risk factors for mortality associated with PD. Low cardiac output syndrome in the 6hours preceding PD installation was associated with a greater mortality in our study. Low cardiac output syndrome is defined by a mean arterial pressure below 60 mmHg in infants and below 50mmHg in neonates despite treatment by vasopressor drugs.[9] Although two other studies showed that low cardiac output syndrome was related to the development of AKI and not to mortality, we found that it was related to mortality while using PD.[9,11] In fact, low cardiac output syndrome signs the failure of cardiac pump, thus resulting in hypoperfusion of the organs, in particular renal hypoperfusion leading to AKI. On the other hand, the severity of low cardiac output syndrome will make the recuperation of renal function more difficult, and may affect other organs, which raises the risk of mortality. Acidosis was considered as a mortality factor in two studies.[4,9] We demonstrated that persistence of acidosis after 24hours of PD was associated with a greater mortality. Presence of acidosis at start of PD was not correlated to mortality. Acidosis is a marker of shock severity in post-operative period, and persistence of acidosis will increase morbidity and mortality.[4] We noted also that absence of improvement in creatinine levels and diuresis after 24hours of treatment was associated with a greater mortality. This can be explained by the absence of renal function recuperation despite adequate treatment. Creatinine levels at PD initiation were not related to mortality although two other studies proved that they were.[4,9] However, high creatinine levels before start of PD result from surgery and not from PD and remain a better marker of mortality due to surgery. Despite the inflammatory reaction caused by cardiopulmonary bypass, the latter was not correlated to mortality, nor was the aortic clamp time. Those results are similar to the ones found by Chan et al.[11] Young age was reported to be a factor favoring the development of AKI and not mortality. This is due to immaturity of kidney autoregulation mechanisms.[17] We did not find a relation between young age and mortality by PD. We had the same result for young patient weight. These results were compatible what Chien et al[4] have found.

Choice of PD timing is controversial until now. In the present study, there was no significant difference for the timing of PD introduction between survivors and non-survivors. Our results join what have

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been published previously since there are no arguments in favor of prophylactic PD or early PD.[10,20] As for duration of PD, analysis of its association with mortality is complicated. In fact, duration of PD can be sometimes short in survivors when they retrieve their renal function earlier. On the other hand, PD use can be short in non-survivors if death occurs earlier. In our study, there was no association between PD duration and mortality. Concerning the comparison of mortality between early PD and late PD, 3 studies showed a decrease of mortality rate with early PD, with mortality ranging from 6% to 25%, while mortality was ranging between 14% and 44% with late PD (7,14,15). In contrary to these results, we had a greater mortality rate with early PD although the difference with late PD was not significant. The main reason for this finding was the early deaths of two children with early PD in whom the severity of the post-operative clinical status and the importance of AKI and multiple organ failure were enough to cause death despite PD use.

As for PD complications, we did not find major ones. The only complications that we found are metabolic (hyperglycemia, hypokalemia), with no repercussion on the patient, which is in favor of PD safety. Several limitations deserve comment. First, the retrospective aspect of this study makes it difficult to analyze the cause-effect relationship between PD and the different variables. Second, the small number of patients enrolled in the PD group did not help us to do a multivariate analysis to establish a stronger bond between PD and mortality, if present. Third, other complications than AKI might have occurred in the post-surgery period, thus affecting the morbidity and mortality. The factors related to these complications were not analyzed.

Conclusion

We found that PD was a safe method to treat AKI after open-heart surgery in children. The main advantages of PD over the conventional treatment were better capacity to improve creatinine levels and BUN after 48hours of treatment. Four main factors were associated with a greater mortality in children who received PD. We mention persistence of acidosis, absence of improvement in creatinine levels and urinary output after 24hours of treatment and low mean arterial pressure in the 6hours prior to PD with low cardiac output syndrome. Finally, it is worth to mention that AKI development depends on the type of surgery, with arterial switch found to be the most related to post-operative AKI.

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Conflict of interest

The author declares that there are no conflicts of interest.

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