PERITONEAL DIALYSIS AS THE FIRST-LINE RENAL REPLACEMENT THERAPY IN PATIENTS WITH AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE.

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Introduction and objectives

Autosomal dominant polycystic kidney disease (ADPKD) is the most common inherited renal disease, which affects more than 12.5 million individuals worldwide(1). Although not every patient with ADPKD ends up requiring dialysis, more than 50% of patients progress to end-stage renal disease (ESRD) (2). ADPKD is the fourth most common cause of ESRD in western world and it is responsible for 3-10% of all patient treated for renal replacement therapy (RRT).(1) In Europe the annual incidence rate of RRT due to ADPKD is 7.8 cases per million in men and 6.9 in women(3). Transplantation is the optimal RRT, but when it is not an option or for patients awaiting transplantation(1) the RRT modality is often based on patients choice, physician-related factors and source availability(2). Traditionally ADPKD is considered a contraindication to peritoneal dialysis (PD)(2). because of progressive development of kidney cysts that might reduce intraperitoneal space and increase intraperitoneal pressure resulting in abdominal wall hernias, leaks and poor dialysis efficacy(1-3). Moreover cysts infection or diverticulitis may increase the risk of peritonitis especially that caused by Gram negative bacteria(1).

Methods

We conducted a retrospective analysis of 40 consecutive incident PD patients who chose PD as their fist-line RRT in our Unit between January 2010 to December 2016. A total of 9 patients (6 females and 3 males) with a median age of 50±10 years had a history of ADPKD, 31 patients (12 females and 19 males) with a median age of 60±16 years had other causes of ESRD. Five ADPKD patients were in incremental CAPD (iCAPD) whit nocturnal dwell of icodextrin solution for 9-10 hours and four patients were in nocturnal intermittent peritoneal dialysis (NIPD). In control group 17 patients were in iCAPD(1-2 dwell) and 14 patients in NIPD. We analyzed demographic parameters, biochemical and clinical data, comorbidity, details of peritonitis, abdominal wall hernia or leaks, mortality, PD technique failure and transplantation.

	ADPKD (n=9)	CONTROL GROUP (n=31)
Age (yrs)	50 +/- 10	60 +/- 16
Male (%)	33%	61%
BMI	24	25
Blood Pressure (mmHg)		
Systolic	136	140
Diastolic	87	83
Comorbid conditions (%)		
Hypertension	88	90
Diabetes	0	9
Heart disease	0	25
Cerebrovascular disease	11	13
Baseline biochemistry		
Haemoglobin (g/dl)	11.6	11.5
Serum albumin (g/dl)	4.1	3.9
Serum uric acid (mg/dl)	5.6	5.4
Calcium (mg/dl)	9.2	8.9
Phosphate (mg/dl)	5	5.4
PTH (pg/ml)	298	291
Triglycerides (mg/dl)	142	141
Total cholesterol (mg/dl)	211	180
GFRe- CKD EPI (ml/min)	7	8
Mean duration of PD	34 +/- 19	28 +/- 20

Tab.1

Results and conclusions

There were no statistically significant differences in relevant biochemical investigations, mean duration of PD and estimated glomerular filtration rate at the beginning of PD therapy. A higher percentage of patients with Diabetes mellitus (9% vs 0%) and heart disease (25% vs 0%) was detected in the control group, probably due to different age of the two patients groups (*Table1*).

	ADPKD (n=9)	CONTROL GROUP (n=31)	*p value*
24h- urine output (ml)	1900	1600	*>0.05*
Total Kt/V	2.7	2.7	*>0.05*
Transporter status (n-%)			
High average and high	4-44	13-42	*>0.05*
Low average and low	5-56	18-58	
Total weekly creatinine clearance	102	98	*>0.05*

Tab.2

No differences were found in residue urine output, baseline assessment of dialysis clearance and membrane function including Kt/V and weekly endogenous and dialysis creatinine clearance (*Table 2*). During observational period 9 episodes of peritonitis were identified (1 in ADPKD group and 8 in 7 patients of the control group). The peritonitis rates were 0.01 and 0.03 episodes per patients-year for ADPKD and control group respectively. In control group 6 episodes of peritonitis were caused by gram-positive bacteria and three by gram-negative organisms. There was a single episode of negative culture peritonitis that occurred in the ADPKD group. 3 patients with ADPKD developed 4 new abdominal hernias, despite of this they were able to resumed peritoneal dialysis after abdominal surgical procedure. In control group only 1 patient developed abdominal hernia, but it was not necessary surgical procedure. The two major causes of interruption of peritoneal dialysis treatment were shift in HD for ultrafiltration failure that occurred in 2 ADPKD patients and 7 patients of control group after an average of 41 months and 24 months respectively and transplant (5 patients in control group and 2 patients in ADPKD group). 2 female patients (1 with ADPKD and 1 in control group) had acute hydrothorax, thus they had to begin HD. No nephrectomy was performed in patients with ADPKD including who received transplant later. There was no record of bowel perforation. Patients survival was similar for the ADPKD and control group (*Tabel 3*).

In this retrospective study we find that survival and long term outcomes of patients with ADPKD receiving PD is essentially the same as that for other PD patients. Even though it is widely believed that patients with ADPKD in PD are susceptible to Gram-negative peritonitis (2) our analysis reveal that incidence of peritonitis, including Gram negative peritonitis, is not increased in ADPKD patients. In present study the major difference between the ADPKD patients and control group is an higher incidence of hernia, even if the incidence rate in our ADPKD patients is substantially lower that in previous reports (4). Our findings show that PD is a feasible treatment option for most ADPKD patients with ESRD.

	ADPKD	Control Group
Death	0	1
Transplant	2	5
Peritonitis		
n° patients	1	7
n° episodes	1	8
Hernias		
abdominal	2	1
inguinal	2	0
Shift HD	2	7
Nephrectomia	0	0

Tab.3

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