DR. ALBERTO MARCACUZCO QUINTO (Orcid ID : 0000-0001-6266-8792)

DR. CARLOS JIMENEZ-ROMERO (Orcid ID : 0000-0002-1965-0666)

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1.Title Page

"Outcome of patients with hemodialysis or peritoneal dialysis undergoing simultaneous pancreas-kidney transplantation. Comparative study"

Author listing

Alberto Marcacuzco[a]. Staff member of HPB Surgery and Abdominal Organ Transplantation

Carlos Jiménez-Romero[ab]. Head of HPB Surgery and Abdominal Organ Transplantation. FACS. Professor of Surgery. UCM

Alejandro Manrique[a] Staff member of HPB Surgery and Abdominal Organ Transplantation. Associate Professor of Surgery. UCM

Jorge Calvo[a] Staff member of HPB Surgery and Abdominal Organ Transplantation. Associate Professor of Surgery. UCM

Félix Cambra[a] Staff member of HPB Surgery and Abdominal Organ Transplantation. Associate Professor of Surgery. UCM

Óscar Caso[a] Staff member of HPB Surgery and Abdominal Organ Transplantation. Associate Professor of Surgery. UCM

Álvaro García-Sesma[a] Staff member of HPB Surgery and Abdominal Organ Transplantation. Associate Professor of Surgery. UCM

Anisa Nutu[a] Resident of General and Digestive Surgery

Iago Justo[a] Staff member of HPB Surgery and Abdominal Organ Transplantation. Associate Professor of Surgery. UCM

Institution:

Unit of HPB Surgery and Abdominal Organ Transplantation[a]. University Hospital "Doce de Octubre". Department of Surgery. Faculty of Medicine. Complutense University (UCM). 28041-Madrid. Spain. Madrid

Correspondence information:

[b]Carlos Jiménez-Romero. MD, PhD, FACS. Head of Unit of HPB Surgery and Abdominal Organ Transplantation. Hospital Universitario Doce de Octubre. 4ª Planta. Ctra Andalucía Km 5,4. 28041-Madrid. Spain. Phone: +91-3908294. Fax: 91-390-8077. e-mail: carlos.jimenez@inforboe.es

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2.Author Contributions:

Alberto Marcacuzco. Study design and drafting of the article

Carlos Jiménez-Romero. Critical revision, design and writing the paper

Alejandro Manrique. Data collection and drafting article

Jorge Calvo. Data analysis and design of the study

Félix Cambra. Data collection and statistics

Óscar Caso. Data analysis and interpretation

Álvaro García-Sesma. Approval of article and interpretation

Anisa Nutu. Data collection

Iago Justo. Concept, design and data analysis

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3.Abbreviations Page:

BMI: body mass index

CMV: cytomegalovirus

HD: hemodialysis

ICU: intensive care unit

PD: peritoneal dialysis

SPKT: simultaneous pancreas kidney transplantation

Key Words: Hemodialysis, peritoneal dialysis, pancreas-kidney transplant, intraabdominal infections, pancreas-kidney transplant mortality, pancreas-kidney complications

4. Abstract

ABSTRACT

Background. Controversy remains with regard to the higher risk of intraabdominal infections and lower patient and graft survival when peritoneal dialysis (PD) rather than hemodialysis (HD) is used in simultaneous pancreas-kidney transplantation (SPKT).

Methods. From March 1995 to December 2015 we performed 165 SPKTs. Prior to transplant, patients received hemodialysis (Group HD; n=98) or peritoneal dialysis (Group PD; n=67). A comparison was performed to analyze posttransplant complications and patient, pancreas and kidney graft survivals.

Results. Donor, pretransplant and perioperative recipient variables were similar in both groups. Overall rates of infections (69.4% in HD vs. 73.1% in PD; p=0.50) and intraabdominal infections (31.6% in HD vs. 35.8 in PD; p=0.57) were similar in both groups. The rates of pancreatitis, hemorrhage or thrombosis of the graft, duodenal graft leak, relaparotomy, transplantectomy, pancreas rejection and retransplantation were similar in both groups.

One, 3, and 5-year patient survival (95.9%, 93.9% and 93.9% in HD vs. 95.5%, 92.2% and 90.4% in PD; p=0.54), and pancreas graft survival (83.6%, 78.0%, and 71.8% in HD vs. 79.2%, 77.4%, and 71.0% in PD; p=0.8) were similar in both groups. Kidney graft survival was similar in both groups.

Pancreas graft thrombosis, rejection and relaparotomy for intraabdominal complications were independent predictors of lower pancreas graft survival, but dialysis modality did not influence patient or graft survival.

Conclusions. Pre-SPKT modality of dialysis does not significantly influence overall or intraabdominal infection and patient, pancreas or kidney graft survivals.

1. INTRODUCTION

Simultaneous pancreas-kidney transplantation (SPKT) is the treatment of choice for patients who suffer diabetes type 1 or type 2 at end-stage renal disease, because it is the only proven method to restore long-term glycemic control.[1-3] Glycemic control after a successful pancreas transplant can reduce the development or progression of many secondary diabetic complications (retinopathy, neuropathy, nephropathy, and cardiovascular disease).[4]

Recipients with pretransplant dialysis show a higher risk of death compared to non-dialyzed recipients, and therefore a preemptive SPKT transplant should be considered whenever possible.[5] There is some controversy regarding the best modality of dialysis before SPKT. Thus, several authors have compared the outcomes of peritoneal dialysis (PD) and hemodialysis (HD) before SPKT, showing discordant results mainly with regard to patient and graft survival and the development of intraabdominal infections.[6-13]

The aim of this study, to our knowledge the largest series considering patients who have undergone SPKT with previous HD versus PD, is to compare both groups regarding the rates of posttransplant complications, patient survival, and survival of pancreas and kidney grafts.

2. PATIENTS AND METHODS

From March 1995 to December 2015 we performed 215 pancreas transplants from brain-death donors, and 173 of these were SPKTs. The sample size for this retrospective, longitudinal cohort study comprised 165 patients, after exclusion of eight patients who underwent SPKT in predialysis status. The sample was divided into two groups according to the dialysis modality before transplant: group HD (n= 98; 59.4%) and group PD (n=67; 40.6%). This study was approved by the institutional review board.

The whole pancreas with duodenum and first jejunal limb, and kidney grafts were recovered and preserved in Belzer or Celsior solution. The pancreas graft was placed intraperitoneally, in a caudal or cephalad position, on the right iliac fossa. The portal vein was anastomosed end-to-side to the common iliac vein (first 27 SPKTs) or to the distal caval vein, and the arterial Y graft of the pancreas was anastomosed end-to-side to the common iliac artery of the recipient. A side-to-side stapled duodeno-cystostomy or a side-to-side hand-sewn duodeno-enterostomy was performed. At the end of pancreas implantation, PD catheters were removed in all recipients, but catheter tip culture was not routinely performed. Only in cases of suspicion of intraperitoneal fluid contamination a culture was carried out. The kidney graft was implanted extraperitoneally through a second oblique incision in the left iliac fossa.

Immunosuppression comprised thymoglobulin (1.5 mg/kg/d) induction for 7 days or basiliximab (20 mg/kg/d at the 1st day, and 20 mg/kg/d at the 4th day), tacrolimus (maintenance levels of 10-15 ng/ml during the first 6 months and 5-10 ng/ml thereafter), azathioprine (100 mg/d) during the first three months and thereafter substituted by mycophenolate mofetil (500 mg/12 h), and steroids.

Prophylaxis of infections consisted of intravenous ceftazidime and vancomycin for bacterial infections, ganciclovir or valganciclovir for cytomegalovirus (CMV) infections, fluconazole for fungal infections, and trimethoprim-sulfamethoxazole for *Pneumocystis jirovecii infections*.

Initially, thrombosis prophylaxis was performed with perioperative low-molecular-weight dextran (250 ml/d) or perioperative low-dose of intravenous heparin (50 U/kg for 5-7 days), and acetylsalicylic acid (300 mg/d) beginning at the 5th day (n=23 HD; n=19 PD); thereafter, we changed to enoxaparin (4000 IU/d) and acetylsalicylic acid (100 mg/d) beginning at the 5th day. Currently we follow the latter prophylaxis regimen.

Posttransplant intraabdominal infection was classified into four grades: 1) evidence of fluid collections by drainage that were managed by antibiotic therapy alone; 2) intraabdominal collections managed by percutaneous drainage; 3) peritonitis or infected collections that required relaparotomy for surgical drainage; and 4) intraabdominal infections that required transplantectomy. Patients who suffered grades 2, 3 or 4 were also treated with antibiotics.

The chronology followed in this comparative study was the registration of pretransplant recipient and donor characteristics, intraoperative and posttransplant variables, postoperative complications, and patient and pancreas and kidney graft survivals.

2.1 Statistical analysis

Quantitative variables with normal distribution were expressed as mean values plus or minus standard deviation (SD), or as median 25 and 75 percentiles when the distribution was abnormal. Qualitative variables were described as percentages. Differences in properties between qualitative variables were assessed by chi-square test or Fisher´s exact test. Comparison of quantitative variables was made by Student´s *t* test. Graft and patient survivals were estimated using the Kaplan-Meier method. Comparison of survival curves was performed using the log-rank test. Pretransplant and perioperative variables showing statistically (near-) significant differences in the univariate analysis were subsequently investigated in a multivariate analysis using the Cox regression method to assess the effect of dialysis modality on patient and graft survival. Hazard ratios (HRs) and 95% confidence intervals (CIs) were determined by adjusting for the dialysis type. A P value less than 0.05 was considered statistically significant. Analysis of these data was performed with the SPSS 15.0 statistical software package.

2. RESULTS

In the comparative analysis between HD and PD recipients we did not find any significant differences regarding gender, age, body mass index (BMI), duration of diabetes, time on dialysis, personal background, and laboratory parameters (Table 1).

Regarding donor characteristics (gender, age, BMI, cause of death, intensive care unit (ICU) stay, hypotension, cardiac arrest, blood transfusion, vasoactive drugs, and laboratory data) there were no statistically significant differences between the groups (Table 2).

With respect to perioperative variables (type of preservation solution, warm and cold ischemia times, frequency of donor CMV+/recipient CMV-, venous and exocrine drainage of the pancreas graft, graft thrombosis prophylaxis, induction therapy with antibodies, and maintenance immunosuppression with calcineurin inhibitors) we did not find any significant differences between the groups (Table 3).

Culture of intraperitoneal fluid was performed in 3 recipients of the PD group because of suspicion of contamination or infection; *Candida albicans* was detected in 2 cases. The overall rate of infections was similar between the groups: 68 (69.4%) patients showed at least one episode of infection in the HD group vs. 49 (73.1%) patients in the PD group (P=0.50). The rate of intraabdominal infections was also similar in both groups: 31 (31.6%) patients in the HD group vs. 24 (35.8%) in the PD group (P=0.57). There was no significant difference in the rate of patients with light intraabdominal infections (grade 1) who were managed with antibiotic therapy alone:

23 (23.5%) patients in the HD group vs. 13 (19.4%) in the PD group (P=0.51). Surgical drainage of intraabdominal collections (peritonitis and abscesses) was more frequently carried out in the PD group than in the HD group: 8 (11.9%) patients in PD vs. 4 (4.1%) in HD. In the PD group, 4 patients underwent 2 relaparotomies and 2 patients needed 3 relaparotomies for drainage of intraabdominal collections. In the HD group, 1 patient underwent 2 relaparotomies and 2 patients needed 3 relaparotomies. The mean period from SPKT to the first reoperation was 7±1.7 days in the HD group versus 13±3.4 days in the PD group.

Percutaneous drainage was indicated in 4 (4.1%) patients of HD group vs. in 2 (3.0%) patients in the PD group. Finally, transplantectomy was only necessary in 1 (1.5%) patient of the PD group because of pancreas graft abscesses.

The rates of other complications, such as wound and CMV infections, overall relaparotomy, graft pancreatitis, graft hemorrhage, graft venous or arterial thrombosis, duodenal leak, transplantectomy, and acute or chronic rejection of the pancreas graft and retransplantation did not reach any significant differences between the groups. The only complication that was almost significantly more frequent in the PD group was the rate of duodenal fistula (6% in HD vs. 15% in PD; P=0.06). The rates of pancreas graft loss because of pancreatitis, hemorrhage, thrombosis, duodenal leak, and rejection were also similar in both groups. The median hospital stay was 26 days in both groups (Table 4). The rates of bacterial and fungal organisms that were isolated from patients with intraabdominal infections were similar in both groups. The most frequently encountered organisms are shown in Table 5. Five patients developed antibiotic resistance to *Pseudomona aeruginosa*

(3 patients in the HD group and 1 in the PD group) and *Klebsiella pneumoniae* (1 patient in the PD group).

One, 3, and 5-year actuarial patient survival was similar in the HD and PD groups (95.9%, 93.9% and 93.9% in HD vs. 95.5%, 92.2% and 90.4% in PD; P=0.54). Likewise, we did not find any significant differences between the groups comparing the rates of actuarial pancreas graft survival at 1, 3, and 5 years (83.6%, 78.0%, and 71.8% in HD vs. 79.2%, 77.4%, and 71.0% in PD; P=0.83), or kidney graft survival at 1, 3, and 5 years (93.8%, 88.1%, and 86.9% in the HD group vs. 95.3%, 92.0%, and 92.0% in the PD group; P=0.71) (Figure 1).

Multivariate Cox regression analysis demonstrated that patient and pancreas graft survivals were not adversely affected by the modality of pre-SPKT dialysis. On the other hand, pancreas graft thrombosis or rejection, and the necessity of relaparotomy for management of intraabdominal complications were confirmed as independent predictors of pancreas graft loss (Table 6).

3. DISCUSSION

There is some controversy regarding the relative mortality risk for end-stage renal disease patients treated with either PD or HD. However, several studies have shown improvements in prognosis for both HD and PD patients.[14-16] In a recent review, it was established that patient survival is similar in PD and HD, but older patients, diabetic patients and those with comorbidities may have a worse prognosis on PD versus HD, whereas younger patients with no comorbidities show higher survival when treated with PD versus HD.[17]

The results are also controversial when a comparison is made between HD and PD patients who undergo kidney transplantation alone, because some authors do not find any significant differences between the groups,[18,19] whereas others find worse results when PD is used before kidney transplant.[20,21]

Several potential risks of PD in diabetic patients have been described (fluid overload, dysregulation of metabolic response to glucose, hyperinsulinemia, obesity, dyslipidemia and peritoneal infection), but important benefits have also been reported (home-based continuous therapy, more liberal diet, intraperitoneal insulin administration, better preservation of residual renal function and blood pressure control, preservation of vascular access, avoidance of myocardial stunning and circulatory stress, and no need for systemic anticoagulation).[22] A series of more than 400 patients showed that survival rate of diabetics on PD was equal to that of nondiabetics on HD, and diabetics on HD had the worst survival.[23] It has been suggested that new PD solutions with low accumulation of glucose degradation products may reduce the risk of cardiovascular disease in diabetic patients, by preserving residual renal function, optimizing volume control, and possibly reducing local and systemic inflammation.[22]

Series comparing PD and HD prior to SPKT have also shown discordant results with respect to patient and pancreas graft survival and the rate of posttransplant intraabdominal infections.[6-13]

Our present series of SPKTs shows homogeneity with respect to the distribution of donor, recipient, and perioperative variables between the groups, and intraabdominal infections, thrombosis, pancreatitis, bleeding and leak of the pancreas graft are the most frequent surgical complications after SPKT. Overall infection rate after SPKT has been reported in up to 80% of patients,[24-26] whereas intraabdominal infections can represent up to 70% of all infections after pancreas transplant,[27] and are associated with higher risk of mortality and pancreas graft loss.[28,29] Ninety per cent of patients who suffer severe intraabdominal infections will need a relaparotomy, and 70% of these will require a pancreas explant.[30] Many risk factors can contribute to the development of intraabdominal infections after SPKT, such as pancreas graft age >45 years, prolonged graft ischemia time,[27,30] long duration of diabetes, PD (association of progressive peritoneal fibrosis), enteric exocrine drainage, immunosuppression,[8,9,31] retransplantation, older recipient, prolonged time of surgery,[27,30,32,33] graft pancreatitis,[34] and leakage from the duodeno-enteric anastomosis.[30]

Several authors have reported that the use of PD compared to HD before SPKT constitutes a higher risk factor for the development of intraabdominal infections,[6-9,13] showing an incidence between 23.5% and 44% in PD groups versus 7.7% to 34% in HD groups.[8,9] However, other studies do not find any significant differences between the groups in the overall incidence of intraabdominal infections (9.6% to 28% in PD groups versus 8.8% to 30% in HD groups).[10-12] In our series the overall rate of infections was 71%, and 55 (33%) patients showed intraabdominal location with no significant difference between the groups (31.6% in HD vs. 35.8% in PD). The majority of our infected patients corresponded to grades 1 and 2, and were managed

by minimally aggressive therapies, such as antibiotic therapy alone in 36 (21.8%) or percutaneous drainage in 6 (3.6%) patients.

Our overall rate of patients undergoing surgical drainage for intraabdominal infections (grade 3) was 7.3% (12 patients), corresponding 8 (11.9%) patients in the PD group vs. 4 (4.1%) patients in the HD group, and only 1 patient needed transplantectomy (grade 4) because of pancreas abscesses.

Other authors have reported rates between 5-29% of surgical drainage of intraabdominal collections,[10,12,13] with the main causes being the presence of infection, bleeding, and thrombosis.[13]

Although we did not find a significant difference in overall incidence of intraabdominal infections between the PD and HD groups, there is a tendency to develop more severe intraabdominal infections in the PD group. Therefore, additional studies with a larger sample size are required to substantiate this preliminary finding. As in other series,[13] pancreatitis, hemorrhage, thrombosis and rejection of the pancreas graft, and duodenal graft leakage were our main causes of graft loss, but we did not observe any significant differences between the HD and PD groups. Moreover, the rate of duodenal leakage was close to being significantly higher in our PD group. Our rates of wound and CMV infections were also similar between the groups.

In a recent report, significantly lower patient and kidney graft survivals were seen in the PD group.[13] However, as other authors,[10,12] we did not find any significant differences with respect to patient, pancreas and kidney graft survivals between the HD and PD groups.

Multivariate Cox regression analysis demonstrated that graft thrombosis, pancreas rejection and mandatory relaparotomy are independent predictors of lower pancreas graft survival. Moreover, the dialysis modality did not affect patient or pancreas graft survival. Other authors found in a multivariate analysis that concomitant cardiovascular disease, pancreas and/or kidney failure, and PD are independent predictors of patient death.[13]

In conclusion, the current study confirms that the pre-SPKT modality of dialysis does not influence the overall rate of infections, or specifically of intraabdominal infections, and patient, pancreas and kidney graft survivals.

Multivariate analysis demonstrated that pancreas graft thrombosis or rejection, and the necessity of relaparotomy for management of intraabdominal complications are independent predictors of pancreas graft loss.

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Table 1. Pre-transplant recipient characteristics

	Sample (n=165)	HD (n=98)	PD (n=67)	P
Gender:				
• **Male**	99 (60%)	61 (62.2%)	38 (56.7%)	0.47
• **Female**	66 (40%)	37 (37.8%)	29 (43.3%)	
Age (yr)	38.9 ± 7.5	38.8 ± 7.9	39.1 ± 6.9	0.76
BMI (kg/m^2)	23.7 ± 3.7	23.4 ± 3.8	24.3 ± 3.4	0.14
Time of diabetes (yrs)	23.6 ± 7.5	23 ± 7.3	24.3 ± 7.8	0.30
Time on dialysis (mos)	21 [13-29]	21 [14-32.5]	21 [11.7-25.4]	0.90
Personal background:				
• **Retinopathy**	157 (95.7%)	92 (93.9%)	65 (98.5%)	0.24
• **Neuropathy**	121 (73.8%)	69 (70.4%)	52 (78.8%)	0.23
• **Cardiopathy**	50 (30.5%)	33 (33.7%)	17 (25.8%)	0.28
• **Gastroparesis**	28 (17.1%)	18 (18.4%)	10 (152%)	0.59
Laboratory data:				
• **Creatinine (mg/dl)**	7.2 ± 2.3	6.8 ± 2.0	7.5 ± 1.5	0.30
• **Hemoglobin (g/dl)**	12.3 ± 1.6	12.5 ± 1.6	12.1 ± 1.4	0.40
• **Serum glucose (mg/dl)**	192 [119-315]	193 [119–336]	192 [116-295]	0.94
• **HbA1c (%)**	8 ± 1.3	8 ± 1.4	8.2 ± 1.2	0.47
• **CMV +**	114 (69%)	72 (74%)	42 (63%)	0.14

Table 2. Donor characteristics

	Sample (n=165)	HD (n=98)	PD (n=67)	P
Gender:				
• **Male**	111 (67%)	67 (68%)	44 (66%)	0.7
• **Female**	54 (33%)	31 (32%)	23 (34%)	
Median age (yr)	29 [21-35]	28.5 [21-36]	29 [12-35]	0.97
BMI (kg/m^2)	23.7 ± 3.7	24.1 ± 3	23.2 ± 2.8	0.07
Cause of death:				
• **Head trauma**	103 (62%)	66 (67%)	37 (55%)	0.41
• **Intracranial bleeding**	47 (29%)	24 (25%)	23 (34%)	0.40
• **Other**	15 (9%)	8 (8%)	7 (11%)	0.45
ICU stay (hr)	48.9 ± 45	52.7 ± 50.7	42.5 ± 32.8	0.19
Hipotension	71 (43%)	42 (43%)	29 (44%)	0.89
Cardiac arrest	20 (12%)	14 (14%)	6 (9%)	0.31
Blood transfusion	44 (27%)	30 (31%)	14 (21%)	0.36
Vasoactive drugs:				
• **Norepinephrine**	135 (82%)	81 (83%)	54 (81%)	0.89
• **Desmopressin**	87 (53%)	45 (46%)	42 (62%)	0.26
• **Dopamine**	38 (23%)	23 (23%)	15 (22%)	0.91
Laboratory data:				
• **Creatinine (mg/dl)**	0.79 ± 0.24	0.80 ± 0.24	0.78 ± 0.22	0.58
• **Serum glucose (mg/dl)**	145 ± 42	147 ± 41	148 ± 34	0.94
• **Serum amylase (IU/l)**	106 [55-209]	107 [55-208]	106 [56-213]	0.93
• **CMV (+)**	120 (73%)	71 (75%)	49 (80%)	0.41

Table 3. Perioperative variables

	Sample (n=165)	HD (n=98)	PD (n=67)	P
Preservation solution:				
• **Celsior**	120 (73%)	71 (72%)	49 (73%)	0.92
• **Belzer**	45 (27%)	27 (28%)	18 (27%)	
Warm ischemia time (min)	70 [60-85]	70 [60-82]	75 [60-85]	0.32
Cold ishemia time (min)	519 ± 114	517 ± 110	521 ± 121	0.83
Donor CMV+/Recipient CMV-	39 (23.6%)	18 (18.4%)	21 (31.3%)	0.54
Venous drainage:				
• **Porto-caval**	138 (84%)	84 (86%)	54 (81%)	0.38
• **Porto-iliac**	27 (16%)	14 (14%)	13 (19%)	
Exocrine drainage:				
• **Intestinal**	106 (64%)	65 (66%)	41 (61%)	0.49
• **Bladder**	59 (36%)	33 (34%)	26 (39%)	
Graft thrombosis prophylaxis:				
• **Enoxaparin**	123 (74.5%)	75 (76.5%)	48 (71.6%)	
• **Dextran**	29 (17.6%)	17 (17.3%)	12 (17.9%)	0.68
• **Heparin i.v.**	13 (7.9%)	6 (6.1%)	7 (10.4%)	
Induction therapy:				
• **Thymoglobulin**	161 (97.6%)	94 (96%)	67 (100%)	0.15
• **Basiliximab**	4 (2%)	4 (4%)	0 (0%)	
Maintenance therapy:				
• **Tacrolimus**	162 (98.2%)	97 (99%)	65 (97%)	0.56
• **CyA**	3 (1.8%)	1 (1%)	2 (3%)	

Table 4. Posttransplant complications

	Sample (n=165)	HD (n=98)	PD (n=67)	P
Overall rate of infections:	117 (71%)	68 (69.4%)	49 (73.1%)	0.50
• **Intraabdominal**	55 (33.3%)	31 (31.6%)	24 (35.8%)	0.57
-Antibiotic therapy	36 (21.8%)	23 (23.5%)	13 (19.4%)	0.51
-Percutaneous drainage	6 (3.6%)	4 (4.1%)	2 (3%)	
-Surgical drainage	12 (6.6%)	4 (4.1%)	8 (11.9%)	
-Transplantectomy	1 (0.6%)	-	1 (1.5%)	
• **Wound**	27 (16%)	17 (17%)	10 (15%)	0.71
• **CMV**	22 (13%)	11 (11%)	11 (16%)	0.31
Overall relaparotomy rate	53 (32.1%)	29 (29.6%)	24 (35.8%)	0.40
• **Transplantectomy**	29 (17.6%)	16 (16.3%)	13 (19.4%)	0.61
Graft pancreatitis:	44 (27%)	29 (30%)	15 (22%)	0.28
• **Graft loss**	7 (4.2%)	5 (5.1%)	2 (3%)	0.70
Graft hemorrhage:	31 (19%)	21 (21%)	9 (13%)	0.31
• **Graft loss**	2 (1.2%)	1 (1%)	1 (1.5%)	0.77
Pancreas graft thrombosis	21 (12.7%)	12 (12.2%)	9 (13.4%)	0.8
• **Vein**	15 (9.1%)	8 (8.2%)	7 (10.4%)	0.83
• **Arterial**	6 (3.6%)	4 (4.1%)	2 (3%)	0.98
Duodenal leak	16 (10%)	6 (6%)	10 (15%)	0.06
• **Graft loss**	2 (1.2%)	1 (1%)	1 (1.5%)	1
Pancreas graft rejection	18 (11%)	12 (12.4%)	6 (9.1%)	0.51
• **Graft loss**	13 (7.9%)	8 (8.2%)	5 (7.5%)	0.87
Retransplant rate	8 (5%)	4 (4%)	4 (6%)	0.57
Median hospital stay (d)	26 [19-39]	26 [20-37]	26 [18-41]	0.96

Table 5. Organisms cultured in patients with intraabdominal infections

	Sample (n=165)	HD (n=98)	PD (n=67)	P
Bacterial organisms (n (%))	40 (24.2%)	25 (25.5%)	15 (22.4%)	0.40
-Bacteroides fragilis	7	4	3	
-Enterococcus faecium	6	2	4	
-Enterococcus faecalis	6	3	3	
-Staphylococcus coag. Negative	6	4	2	
-Escherichia coli	4	4	0	
-Pseudomona aeruginosa	4	3	1	
-Klebsiella Pneumoniae	1	0	1	
-Other	6	5	1	
Fungal organisms (n (%))	29 (17.6%)	15 (15.3%)	14 (20.9%)	0.17
-Candida Albicans	22	10	12	
-Candida Glabrata	3	2	1	
-Candida Parapsilosis	3	2	1	
-Candida Lusitaniae	1	1	0	

Table 6. Multivariate Cox regression analysis of predictors of patient and pancreas graft survival

	HR	(CI 95%)	p
Patient survival:			
• **Transplantectomy**	2.64	(0.88-7.84)	0.08
• **Dialysis modality**	1.3	(0.52-3.39)	0.55
• **Time of diabetes (yr)**	1.05	(0.99-1.12)	0.06
Pancreas graft survival:			
• **Graft thrombosis (venous/arterial)**	8.62	(2.51-29.61)	0.001
• **Pancreas rejection**	4.66	(2.22-9.73)	0.000
• **Relaparotomy**	2.99	(1.39-6.43)	0.005
• **Dialysis modality**	1.04	(0.58-186)	0.87

Figure 1a. Patient survival

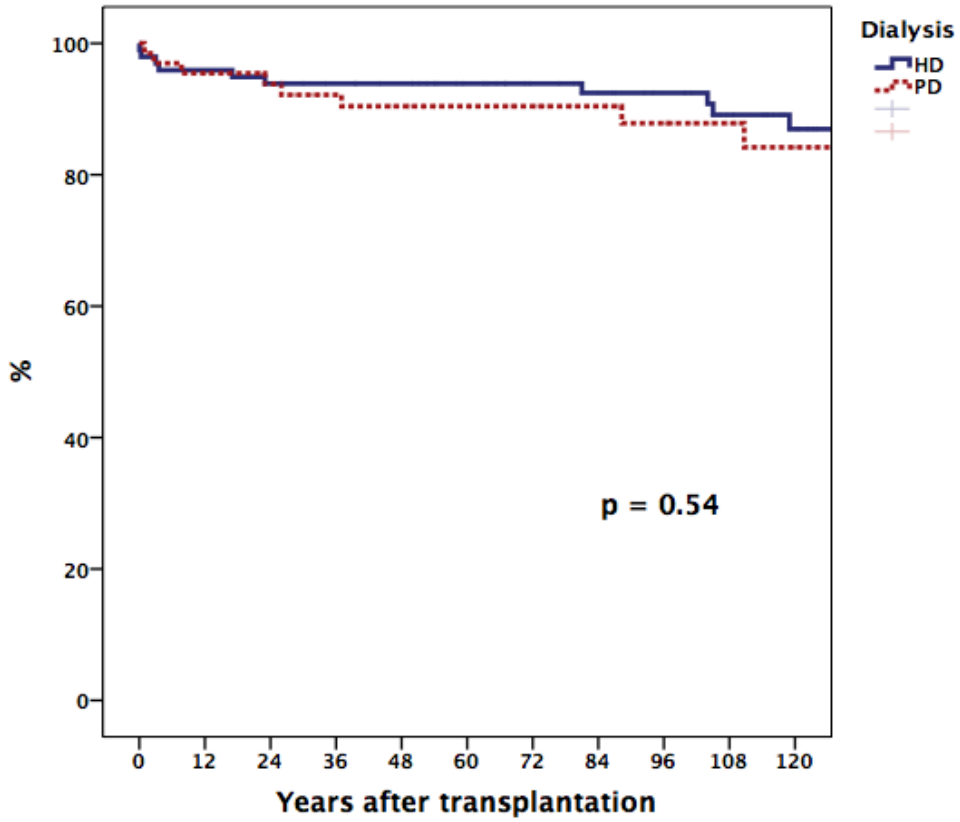

Figure 1b. Pancreas graft survival

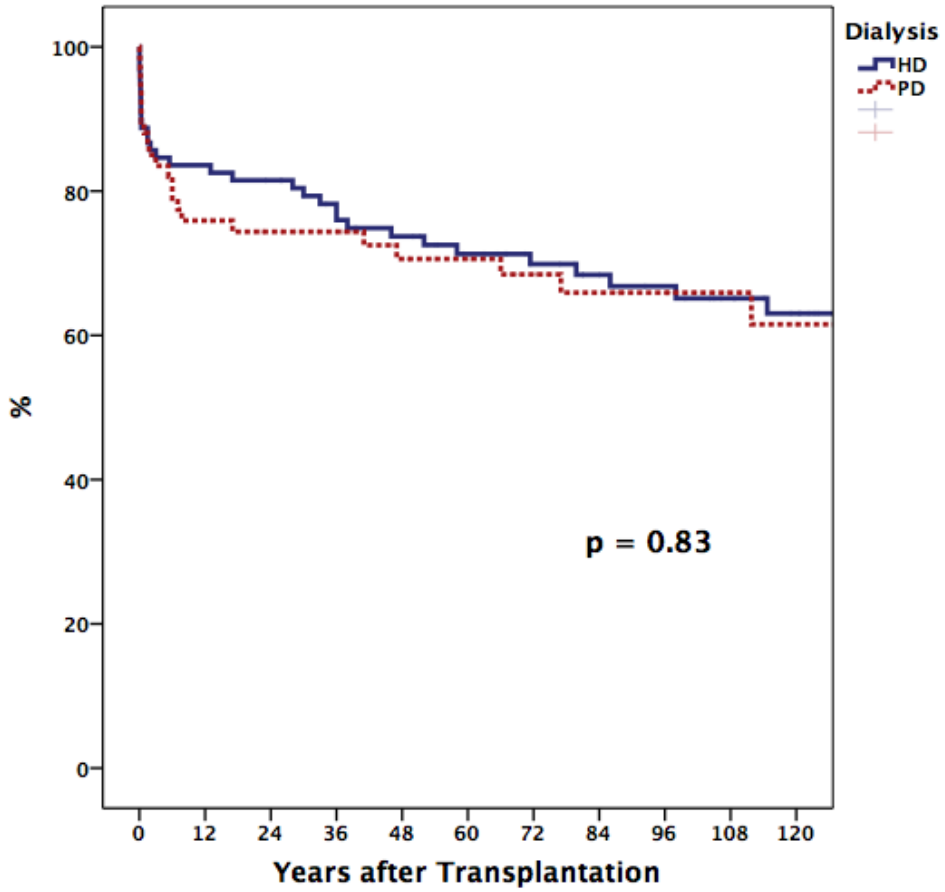

Figure 1c. Kidney graft survival