The impact of kidney function and intra-abdominal infection on patient 1-yr survival after simultaneous pancreas-kidney transplantation (SPKT)

**ABSTRACT**

Introduction: Simultaneous pancreas-kidney transplantation (SPKT) is one of the treatments for insulin-dependent patients with chronic renal failure. Methods: One-year patient and kidney allograft survival rates of 150 patients submitted to SPKT analyzed by COX regression and Kaplan-Meier. Uni- and multivariate analysis identified the risk factors involved with either allograft or patient survival. Results: One-year patient and kidney allograft survival rates were 82% and 80%, respectively. Delayed graft function from kidney (DGF) (P = 0.001, HR 5.41), acute kidney rejection (P = 0.016, HR 3.36) and intra-abdominal infection (IAI) (P < 0.0001, HR 4.15) were related to the 1-yr patient survival. One-year kidney allograft survival was also related to DGF (P = 0.013, OR 3.39), acute rejection (P = 0.001, OR 4.74) and IAI (P = 0.003, OR 6.29). Main risk factors for DGF: time on dialysis > 27 months (P = 0.046, OR 2.59), kidney cold ischemia time > 14 hours (P = 0.027, OR 2.94), donor age > 25 years (P = 0.03, OR 2.82) and donor serum sodium > 155 mEq/l (P < 0.0001, OR 1.09). Conclusions: Delayed kidney allograft function and IAI had an important impact on either patient or kidney allograft survival rates. Improving deceased donor care may reduce DGF occurrence.

Keywords: survival, medical complications, surgical complications, pancreas-kidney transplantation.

**INTRODUCTION**

Insulin-dependent diabetic patients with renal failure present higher survival rates after simultaneous pancreas-kidney transplantation (SPKT) compared to dialysis treatment. Recent data from United Network for Organ Sharing (UNOS) and International Pancreas Registry (IPTR) have shown that patient’s one-year survival after SPKT is higher than 95%. One-year survival after pancreas transplantation is improving over the years and is around 85%, which is attributed to a decrease in technical failure (thrombosis, infection, pancreatitis, fistula of anastomosis or bleeding) and immune loss rates. Death with functioning graft, however, is one of the major causes of pancreas allograft loss after the first year, corresponding to approximately 50% of loss causes following SPKT. Regarding kidney allograft, one-year survival after SPKT is approximately 90%.

On the other hand, some data in the literature have shown higher survival rates in insulin-dependent diabetic patients with renal failure after kidney transplant alone compared to SPKT, which may probably be related to increased short-term mortality after SPKT. In other words, after SPKT, recipients have a higher mortality compared to kidney recipients from living-donor within the first 18 months, but later the survival is greater after SPKT. In long-term, that is, within 7 to 10 years, SPKT recipients have lower incidence of myocardial ischemia, stroke and amputation in comparison to recipients of kidney alone. This difference is attributed to a reduction in carotid intima-media thickness and coronary atherosclerosis, as well as due to an improvement in echocardiographic parameters after SPKT.

However, there are few data in the literature considering survival rates in patient, kidney and pancreas allograft after SPKT in hospitals outside US. The identification of risk factors related to those survival rates would help to explain the higher mortality rates and loss of kidney and pancreas allograft following SPKT and, consequently, establish better strategies for improving outcomes in Brazilian sites.

In the present study, we performed a retrospective analysis of 150 patients submitted to SPKT in only one Brazilian transplantation center during a 6-years follow-up. In addition, a multivariate analysis was performed...
to identify the risk factors that influenced the survival rates within one year for patients as well as for kidney and pancreas allograft after SPKT.

PATIENTS AND METHODS

PATIENTS

From December 2000 to August 2006, 150 simultaneous pancreas-kidney transplantations were performed in the Universidade Federal de São Paulo, São Paulo, Brazil. All patients had history of insulin use prior transplantation. Approximately 95% of the diabetic patients submitted to SPKT were diagnosed with type 1 diabetes, and about 5% were diagnosed as MODY (Maturity Onset Diabetes of the Young) or LADA (Latent Autoimmune Diabetes in Adults) types. In Brazil, the allocation of pancreas-kidney for SPKT is based on registration time, and the waitlist time is around 2 years. Enteric exocrine derivation was performed in 143 patients (in 7 patients, the exocrine derivation was vesical). Vascular anastomosis was carried out in common or external iliac veins or inferior vena cava. Belzer and Euro-Collins solutions were used for pancreas and kidney preservation, respectively. Only donors that presented no cardiac arrest were selected, as well as those less than 45 years of age and no history of diabetes mellitus in first-degree relatives.

IMMUNOSUPPRESSION AND POST-OPERATIVE CARE

Prophylactic anti-infective therapy was made with ceftriaxone (1 g every 12 hours) and ampicillin (1 g every 6 hours) at least for one week or until the removal of intra-abdominal drain. Intraoperatively, a bolus of 1-g methylprednisolone was given. Heparin prophylaxis was started 12 hours after surgery and continued up to one week following transplantation, since no bleeding was noted. Subcutaneous maintenance doses ranged from 10,000 to 15,000 U/day, and partial thromboplastin time was kept close to reference range. Aspirin in a dose of 100-200 mg/day was started 2 to 3 days following transplantation. Immunosuppression started on the second day and included: Tacrolimus 0.15 mg/kg/dose, adjusted for transplantation time (serum levels 10-15 ng/ml within the first 30 days, 8-10 ng/ml between days 31 and 90 and subsequently 5-10 ng/ml), prednisone 30 mg/day (decrease of 5 mg monthly up to a maintenance dose of 5 mg/day about the sixth month) and mycophenolate mofetil 2 g/day or mycophenolate sodium 1.44 g/day in all cases. Mono or polyclonal antibody induction was not used in a routine basis, only in retransplant cases, in cases of lymphocyte reactivity panel higher than 20% or when cold ischemia time exceeded 24 hours. Thirteen out of the 150 patients (8.7%) received induction: 5 received thymoglobulin (4%), 2 received OKT3 (1.3%), and 4 received basiliximab (3.3%).

ENDPOINTS

Primary endpoints were patient, kidney and pancreas one-year survival after SKPT. Secondary endpoints included the determination of risk factors that have had influence on patient and allograft survival. All deaths occurred during the first 90 days were considered as transplant-related.

Traditional risk factors of recipients included sex, age, race, dialysis time, time of diagnosis of diabetes mellitus, dialysis type (hemodialysis vs. peritoneal dialysis), body mass index (BMI), calculated as body weight (kg) divided by the square of the patient’s height (m²), and presence of coronary atherosclerotic disease prior to transplantation, defined as need of coronary angioplasty or myocardial revascularization. Patients with a history of diabetic longer than 20 years or presenting other risk factors for coronary artery disease, such as smoking habit, family history of sudden death or previous cardiovascular event underwent a coronary angiography. Diabetic patients with low cardiovascular risk were submitted to non-invasive tests, such as a dobutamine stress echo or dipyridamole myocardial scintigraphy. Traditional risk factors for deceased donors included sex, age, BMI, and cause of brain death, which was divided in cardiocerebrovascular and non-cardiocerebrovascular causes. Transplant-related risk factors for recipients included ischemia time for kidney and pancreas allograft, delayed kidney graft function (DGF, defined as need of dialysis in the first week after transplantation), kidney acute rejection classified according to the Banff criteria, 1997, cytomegalovirus infection diagnosed by antigenemia test (detection of pp65 protein in peripheral blood leukocytes), acute pancreatitis (clinically defined by the presence of fever, local pain, increase in serum amylase, and/or hyperglycemia), peripancreatic abscess, fistula of anastomosis, and vascular thrombosis. Intra-abdominal infection (IAI) included the presence of peripancreatic abscess and fistula of enteric anastomosis. Venous thrombosis of pancreatic allograft is rarely reversible and usually presents with graft edema, sudden onset of abdominal pain as well as a sudden increase in glucose levels and serum amylase. Arterial thrombosis of pancreatic allograft may affect splenic artery, superior mesenteric artery or both. It is characterized by a sudden increase in glucose levels and decrease in serum amylase, in the absence of abdominal pain or discomfort. Data from deceased-donors included serum creatinine (mg/dL), serum amylase (U/l), serum sodium (mEq/L), and use of vasoactive drugs (noradrenaline and dopamine), expressed in µg/kg/min.
Kidney allograft biopsy was indicated whenever there was an increase in creatinine levels and/or a reduction in diuresis, while pancreatic allograft biopsy was performed in the presence of an unexplained increase in glucose levels or a persistent increase in pancreatic enzymes (amylase and lipase).

**Statistical Analysis**

All results are shown as mean ± SD. Statistical analysis was carried out in SPSS 12.0 software (Chicago, IL, USA). Fisher's exact t-test and ANOVA were used for numerical variables, and Pearson's X² test in categorical variables. Correlations were tested by Pearson’s correlation coefficient. Kaplan-Meier curves were used to determine the survival of the patients and grafts. For graft survival analysis, death was considered as graft loss regardless of its functioning status at the time of death. In the event that a graft was removed before death, data were analyzed as a death with non-functioning graft.

To determine traditional and transplant-related risk factors and those that had a correlation with one-year survival for patient and kidney pancreas allograft, as well as to the occurrence of acute pancreatitis, intra-abdominal infection and delayed kidney graft function, all variables with a P value ≤ 0.3 in univariate analysis were included in the binary logistic regression model, and those factors were considered as dependent variables. Results were described as odds ratio (OR) or relative risk (RR) using a 95% confidence interval (CI). Statistical analysis was considered significant for P < 0.05.

**Results**

Demographic data of recipients and donors are described in Table 1. One-year survival rates for patients and for kidney and pancreas allograft were 82%, 80% and 76.7%, respectively. Mean time to death after transplantation was 78.2 ± 82.1 days (ranging from 3 to 259 days). Approximately 70% of deaths occurred in the first 90 days after transplantation, and eight deaths that occurred after this period were related to infection.

**Patient Survival**

The analysis of variables obtained by Cox regression showed that presence of delayed kidney graft function (DGF) (P = 0.001, RR 5.4, 95% CI 1.98-14.8), kidney acute allograft rejection (P = 0.016, RR 3.36, 95% CI 1.25-9.1) and intra-abdominal infection (IAI) (P < 0.0001, RR 4.15, 95% CI 2-10.4) were the main risk factors correlated with one-year survival of patients. Patient’s survival curves according to log-rank analysis are shown in Figures 1A-C. Risk factors related to deceased-donors did not have impact on survival rates of patients. Although kidney DGF has not increased the incidence of kidney acute rejection (P = 0.81), one-year survival of patients without DGF and without kidney acute rejection was 92.6%, compared to 77.1% in cases without DGF but with kidney acute rejection. One-year survival of patients with DGF and without kidney acute rejection was 69.6%, compared to 45.4% when both DGF and kidney acute rejection were present (log-rank = 13.91, P = 0.0002).

There was a slight increase in the one-year survival of the patient in the first 75 cases when compared to the last 75 cases from 81.3% to 82.7%, respectively, but without statistical significance (P = 1.0). However, the incidence of kidney DGF did not change significantly (21.3% vs. 24%, P = 0.7), as well as the incidence of kidney acute rejection (26.7% vs. 34.1%, P = 0.29) and peripancreatic abscess (14.7% vs. 6.7%, P = 0.11).

The main cause of death after transplantation was sepsis (66.7%). IAI was more frequent (55.6%), followed by pulmonary infection (44.4%). Other causes of death included hemorrhagic shock (18.5%) and cardiovascular complications (14.8%).

**Kidney Allograft Survival**

With respect to kidney allograft survival, there were 30 losses in the first year. The main cause of loss was death with functioning graft (83.4%), followed by kidney acute rejection (16.6%). Logistic regression analysis identified kidney DGF (P = 0.013, OR 3.39, 95% CI 1.3-8.9), kidney acute rejection (P = 0.001, OR 4.74, 95% CI 1.9-11.9) and IAI (P = 0.003, OR 6.29, 95% CI 1.9-20.8) as the main risk factors influencing one-year kidney allograft survival.

For kidney DGF occurrence, the multivariate analysis showed that dialysis time greater than 27 months (P = 0.046, OR 2.59, 95% CI 1.02-6.8), kidney cold ischemia time greater than 14 hours (P = 0.027, OR 2.94, 95% CI 1.13-7.6), age of donor greater than 25 years (P = 0.03, OR 2.82, 95% CI 1.1-7.2) and serum sodium value from donor higher than 155 mEq/L (P < 0.0001, OR 1.09, 95% CI 1.04-1.15) were the main determinants. Female to male deceased donors occurred in 17% of cases, but were not related to kidney DGF event. In this study, we found a positive correlation between deceased-donors serum creatinine and sodium values (P < 0.0001) (Figure 2). In other words, the more severe the donor’s clinical presentation, the higher the creatinine and sodium serum levels, leading to the occurrence of kidney DGF. In addition, the
### Demography of Recipient and Deceased-Donor

#### Recipient data

<table>
<thead>
<tr>
<th>Demography data</th>
<th>Value or %</th>
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<tr>
<td>Sex, M:F</td>
<td>75:75</td>
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<tr>
<td>Age (years)</td>
<td>34.6 ± 7.9 (range, 18 to 55)</td>
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<tr>
<td>Race</td>
<td>24% Black</td>
</tr>
<tr>
<td>Duration of diabetes (years)</td>
<td>21.9 ± 5.9 (range, 10 to 37)</td>
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<tr>
<td>Time on dialysis (months)</td>
<td>26.9 ± 18.6 (range, 0 to 108)</td>
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<tr>
<td>Hemodialysis</td>
<td>76.7% (n = 115)</td>
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<tr>
<td>Peritoneal dialysis</td>
<td>17.3% (n = 26)</td>
</tr>
<tr>
<td>Preemptive</td>
<td>6% (n = 9)</td>
</tr>
<tr>
<td>Body mass index (kg/m2)</td>
<td>21.4 ± 2.9 (range, 17.2 to 37.4)</td>
</tr>
<tr>
<td>Atherosclerotic heart disease prior transplantation</td>
<td>11.3% (13 coronary angioplasty and 4 CABG)</td>
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#### Donor data

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<td>Sex, M:F</td>
<td>92:58</td>
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<tr>
<td>Age (years)</td>
<td>25.6 ± 9.4 (range, 10 to 46)</td>
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<td>Cause of death</td>
<td>24.7% (n = 37)</td>
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<td>Cerebrovascular</td>
<td>75.3% (n = 113)</td>
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<tr>
<td>Body mass index (kg/m2)</td>
<td>23.7 ± 3.2 (range, 12.5 to 28.1)</td>
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<tr>
<td>Use of vasoactive drugs, yes:no</td>
<td>92.8%:72%</td>
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<tr>
<td>Dopamine</td>
<td>45.4% (mean dose 7.6 µcg/kg/min)</td>
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<tr>
<td>Noradrenaline</td>
<td>41.5% (mean dose 0.42 µcg/kg/min)</td>
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<td>Dopamine + Noradrenaline</td>
<td>12.3%</td>
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<td>Dopamine + Dobutamine</td>
<td>0.8%</td>
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#### Laboratory data

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<tr>
<td>Mean serum creatinine (mg/dl)</td>
<td>1.2 ± 0.6 (range, 0.3 to 3.7)</td>
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<tr>
<td>Mean serum sodium (mEq/l)</td>
<td>154.6 ± 16.5 (range, 119 to 206)</td>
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<tr>
<td>Mean serum amylase (U/l)</td>
<td>163.4 ± 229.3 (range, 22 to 1406)</td>
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#### Transplant-related factors

<table>
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<tr>
<td>Mean kidney cold ischemia time (hours)</td>
<td>14.03 ± 3.9 (range, 6 to 27)</td>
</tr>
<tr>
<td>Mean pancreas cold ischemia time (hours)</td>
<td>14.3 ± 4.6 (range, 6 to 29)</td>
</tr>
<tr>
<td>Delayed renal graft function</td>
<td>22.7%</td>
</tr>
<tr>
<td>Acute renal rejection</td>
<td>30.7%</td>
</tr>
<tr>
<td>Cytomegalovirus infection</td>
<td>42%</td>
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#### Pancreatic graft complications

<table>
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<tr>
<td>Pancreatitis</td>
<td>13.3%</td>
</tr>
<tr>
<td>Fistula of enteric anastomosis</td>
<td>4%</td>
</tr>
<tr>
<td>Pancreas abscess</td>
<td>11%</td>
</tr>
<tr>
<td>Vascular thrombosis</td>
<td>4%</td>
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cause of death in donors under the age of 25 years was non-cerebrovascular in 87.6% vs. 12.4% cerebrovascular, compared to patients aged over 25 years, which had 45% of deaths due to non-cerebrovascular vs. 55% cerebrovascular ($P < 0.0001$, OR 8.7, 95% CI 3.85-19.5).

In the present study we did not identify correlation of kidney acute rejection to previously known risk factors, such as the value of lymphocyte reactivity panel, dialysis time, and race, although 40% of black patients developed rejection compared to non-black patients ($vs. 27.8\%$, $P = 0.17$). Also, panel value was 10-20% in 3 patients and greater than 20% in 5 patients. There were only 2 patients who had been previously submitted to kidney transplantation.

**Survival of Pancreas Allograft**

In the same period, surgical complications related to pancreatic allograft were described in 60 patients (40%), and they included postoperative bleeding (10.7%), peripancreatic abscess (8%), reoperation due to intestinal sub-occlusion (6%), vascular thrombosis (5.3%, 3 of them were arterial thrombosis and 5, venous thrombosis), evisceration (4%), anastomosis fistula (2.7%), non-therapeutic laparoscopy (2%), and reoperation due to perforating acute abdomen (1.3%). Five out of 12 patients (41.7%) presenting peripancreatic abscess evolved to death due to intra-abdominal infection and sepsis. Acute pancreatitis was diagnosed in 20 cases (14%) with enteric derivation and complication due to peripancreatic abscess occurred in 50%.

Along the first year after transplant there were 35 pancreatic allograft losses (Figure 3). Similarly to kidney allograft, the main cause for pancreas allograft loss was death with functioning graft (54.3%), followed by vascular thrombosis (20%), death with non-functioning graft (14.3%), peripancreatic abscess or fistula of enteric anastomosis (5.8%), and pancreatic acute rejection asynchronous to kidney rejection (2.8%). The five cases of death with non-functioning graft included 2 cases of pancreatectomy secondary to peripancreatic abscess, 2 cases...
of pancreatectomy secondary to fistula of enteric anastomosis, and 1 case secondary to venous thrombosis. There was one case of intraoperative pancreas loss due to lack of perfusion and major atherosclerosis of recipient.

Risk factors for pancreatic allograft loss during the first year included IAI (P < 0.0001, OR 12.83, 95% CI 3.2-52.2), vascular thrombosis (P = 0.002, OR 40.55, 95% CI 3.85-426.6), kidney acute rejection (P = 0.027, OR 3.06, 95% CI 1.13-8.3), serum donor sodium greater than 155 mEq/L (P = 0.02, OR 3.27, 95%CI 1.2-8.9), and donor dopamine doses greater than 7.6 µcg/kg/min (P = 0.046, OR 2.85, 95%CI 1.01-8). Logistic regression analysis used to identify the risk factors involved in IAI event showed that kidney DGF was the only factor (P = 0.027, OR 3.56, 95% CI 1.2-10.6).

Peritoneal dialysis as treatment modality was also the only risk factor associated with the occurrence of acute pancreatitis (P = 0.035, OR 3.2, 95% CI 1.08-8.9). Fifty per cent of patients with acute pancreatitis had IAI as complication. No other risk factor was identified in the event of vascular thrombosis of pancreatic allograft.

**DISCUSSION**

In the present study, we evaluated the one-year survival rates for patients and kidney and pancreas allograft after SPKT in a single transplantation center. Intra-abdominal infection (IAI) and kidney DGF had an impact on survival of kidney and pancreas allograft. The incidence of IAI and kidney DGF in the first 75 patients was similar to the 75 subsequent patients, justifying the impact persistence of these complications on patients’ survival in the two groups analyzed. In addition, death with functioning graft was the main cause for renal and pancreas allograft losses.

Kidney allograft survival after SPKT was influenced by kidney DGF, acute rejection, and IAI. In the present study, we noted that DGF incidence of 22.7% was greater than that described by UNOS data (4%), in spite of...
the fact that the cold ischemia time was the same. This difference motivated us to identify the potential risk factors in donors and recipients that might be involved in the incidence of kidney DGF.

The impact of kidney DGF on patient survival may be explained by the effect of immunosuppressant use in uremic patients under hemodialysis, so that the risk of infection has increased. Also, the treatment for acute rejection has increased even more the risk of infection due to high doses of corticosteroids.

The impact of kidney DGF on kidney survival is controversial. Nonetheless, kidney DGF is an independent risk factor for kidney allograft loss in one year and long-term, although other authors reported that this impact was on kidney function and not on survival. Several risk factors from recipients and donors are related to the occurrence of DGF after a kidney transplant, which may also be present after SKPT. In the present study, the risk factors for kidney DGF included dialysis time, cold ischemia time and age of donor, and, interestingly, donor serum sodium level. There are some aspects that should be discussed in relation to this finding. Firstly, the mean cold ischemia time of kidney allograft in our patients was 14 hours, while in the literature this time exceeds 24 hours at the time transplant is performed. Therefore, we would expect that the incidence of kidney DGF would be lower in this study, since, as described by other authors, at each 6-hours increment in cold ischemia there is a 23% increase in the risk of kidney DGF. Consequently, the factors already known to increase DGF, such as those affecting the donor kidney during the diagnostic workout to establish brain death, had greater impact in our cases, which would, at least in part, explain the greater incidence of observed kidney DGF.

Brain death diagnosis is associated with hypotension, which in its turn is secondary to hypovolemia, cardiac dysfunction, and vasodilation, so an appropriate treatment would minimize ischemic damage in donors’ organs. In Brazilian hospitals, most of the time the use of vasopressin is not available, due to high cost. Thus, our donors, who in 2/3 of times had a cranial trauma as the etiology of brain death, were not properly treated for diabetes insipidus. This may be one of the possible explanations for the association of high levels in donor serum sodium and occurrence of kidney DGF. This is the first report in the literature of this association after a SKPT, although after liver transplantation hypernatremia has a negative impact on short-term allograft survival. However, it is described that a correction in donor hypernatremia may reduce liver allograft loss. The mechanisms by which hypernatremia leads to hepatocytes damage, and probably renal tubular cells, are not fully elucidated, but they seem to involve mitochondrial ultrastructural changes, in addition to activation of apoptotic pathways and inhibition of antiapoptotic pathways.

Secondly, it is described that renal tubular cells in renal medulla are resistant to sodium chloride and urea-induced hyperosmolarity, because of an increase in HSP 70 (heat shock protein 70). However, our donors, although hypernatremic, were seriously hypovolemic and receiving vasoactive drugs, thus chaperones induction was overcome by the severe condition of renal hypoperfusion. In addition, even the kidney preservation in Euro-Collins solution, which is hyperosmolar (340 mOsm) and rich in intracellular ions resulting in minimization of cell edema secondary to Na+/K+ pump inhibition, was not sufficient to maintain renal tubular cells integrity. Therefore, inadequate handling of deceased-donor was probably one of the main determinants of kidney DGF event and may be an indicator of renal ischemic damage. However, the insufficient number of donors does not allow us to exclude it in these situations, but efforts should be made to minimize the ischemic damage. Thus, induction with antithymocyte globulin could be a useful strategy to reduce the occurrence of ischemia-perfusion damage and, subsequently, the rate of kidney DGF, and also to allow a delay in starting calcineurin inhibitors. On the other hand, antithymocite globulin use could increase the risk of infection.

The second risk factor correlated to patient and kidney allograft survival was acute rejection. UNOS data are consistent with a worse kidney allograft survival when both kidney and pancreas rejection are present compared to kidney rejection alone after SKPT. However, our data should be analyzed with caution, since in the majority of cases only kidney allograft was biopsied, due to some operational difficulty to perform biopsies in pancreas allograft. The association between kidney acute rejection and DGF was not seen in our study, although it is well established in the literature. No association between kidney acute rejection and known factors was found, since most of our patients were not sensitized and there were few cases of retransplant and a predominance of non-black patients. But, according to other authors, the presence of both kidney acute rejection and DGF is related to the worst survivals of kidney allograft.

The third risk factor related to kidney allograft survival was the presence of IAI, which certainly reflects the number of patients who died with a functioning graft. The main causes of pancreatic allograft loss included death with functioning graft, technical loss and
immunosuppressant therapy. Even though it is self-sepsis, ischemia-perfusion injury, surgical trauma and cordance with our cases. Similarly, IAI may have a impact on pancreatic allograft survival, which is in accord- ance with our cases. In the present study, the main risk factor for IAI was kidney DGF, which could be explained by a greater immunosuppression in patients under dialysis or those with partial function of renal al-lograft after transplant, as well as to reflect the intensity of ischemia-reperfusion damage of pancreatic allograft similar to would be occurring with kidney allograft.

We did not find correlation of IAI with other risk factors previously established in the literature, such as donors aged over 45-50 years, donor’s cause of death other than non- cardiocerebrovascular, donor and re-cipient BMI greater than 30 kg/m², and cold ischemia time greater than 24 hours. In Brazil, the age limit of deceased-donor for pancreas donation is 45 years, and there was only one case of a donor aged 46 years in this study. In addition, the cause of brain death in 2/3 of times was attributed to cranial trauma, while donor and recipient’s BMI were different in relation to those described by those authors, which would explain the lack of association with IAI occurrence.

However, although dopamine dosage and donor se-rum sodium in the occasion of donation did not directly affect the incidence of IAI and vascular thrombosis, but they contributed negatively to pancreatic allograft sur-vival by mechanisms similar to those described for kidney allograft. The use of dopamine may have contributed to pancreatic hypoperfusion in deceased-donors.

The association of kidney acute rejection and pan-creatic allograft loss can be explained by the effect of immuno-suppressant therapy and by the simultaneous oc-currence of pancreatic acute rejection, though this asso-ciation should be cautiously analyzed, provided that pan-creatic allograft biopsy was not available in most cases.

In the present study, pancreatitis of pancreas allo-graff was seen in 14% of cases with enteric derivation, which is similar to data in literature. When present at the immediate postoperative period, it is related to sepsis, ischemia-perfusion injury, surgical trauma and immunosuppressant therapy. Even though it is self-limited, complications such as pancreatic collection or necrosis of the adjacent fat may occur, and they can lead to more severe infections requiring surgical treat-ment. The only risk factor associated with acute pan-creatitis in our study was peritoneal dialysis prior to transplant. Yet, data in literature are controversial in relation to peritoneal dialysis and to the higher risk of infection after pancreatic transplant, so more stud-ies are needed to confirm this association. Our incidence of thrombosis (5.3%) is in accordance to literature, even though we did not find an associa-tion with risk factors already described.

In conclusion, kidney DGF and IAI had a negative impact on patient and kidney and pancreas allo-graff survivals after SPKT. The difference in mortality rates and allograft losses in the present study may explain lower results in Brazilian centers compared to American and European centers. Therefore, new strategies should be established to optimize deceased-donor cares, as well as a more effective treatment in IAI events; that is, an earlier indication for pancreatectomy to avoid sepsis, in addition to minimization of ischemia-perfusion injury with antithymocyte globu-lin, preferential use of Belzer solution to promote pres-ervation, and use of perfusion equipment.

REFERENCES


