

Impact of Pancreatic Allograft Function on 1-Year Survival Rates After Simultaneous Pancreatic-Renal Transplant

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Abstract

Objectives: Simultaneous pancreatic-renal transplant is an effective treatment for insulin-dependent patients with chronic renal failure. We sought to identify the main influences on pancreatic and patient survival rates after simultaneous pancreas-kidney transplants.

Patients and Methods: The 1-year patient and pancreas survival rates of 150 patients who had undergone simultaneous pancreas-kidney transplant were analyzed by the Cox proportional hazards regression model and the Kaplan-Meier method. Uni- and multivariate analyses were performed in terms of transplant-, recipient-, and donor-related risk factors.

Results: At 1 year, patient and pancreatic allograft survival rates were 82% and 76.7%, respectively. Delayed graft function in the kidney ($P = .001$, HR 5.41), acute kidney rejection ($P = .016$, HR 3.36), and intra-abdominal infection ($P < .0001$, HR 4.15) were the main factors related to 1-year patient survival. Pancreatic allograft survival at 1 year was related to intra-abdominal infection ($P < .0001$, OR 12.83), vascular thrombosis ($P = .002$, OR 40.55), acute kidney rejection ($P = .027$, OR 3.06), donor sodium greater than 155 mEq/L ($P = .02$, OR 3.27), and dopamine administration exceeding 7.6 $\mu\text{g/kg/min}$ ($P = .046$, OR 2.85).

Conclusions: Delayed kidney allograft function and intra-abdominal infection had an important effect on both patient and pancreatic allograft survival rates.

Key words: Pancreas-kidney transplantation, Survival, Complications

Insulin-dependent patients with chronic renal failure show greater survival rates with simultaneous pancreatic-renal transplant than with dialysis treatment (1).

Recent data from the United Network for Organ Sharing and the International Pancreas Registry showed 1-year patient survival to be higher than 95% after simultaneous pancreatic-renal transplant (2, 3). Improved 1-year pancreatic allograft survival, which is related to patient outcome, is approximately 85%; prolongation of graft survival is attributed to decreases in technical failures (thrombosis, infection, pancreatitis, anastomotic leak, or bleeding leading to removal) and in immunologic failures (2). Patient death despite a previously functioning graft is reported to be among the most important categories of pancreatic graft failure after 1 year after transplant, accounting for approximately 50% of grafts considered to have failed after simultaneous pancreas-kidney transplant (2).

Some studies show better survival rates for patients with chronic renal failure and diabetes after renal transplant alone than after simultaneous pancreatic-renal transplant (4, 5), which probably reflects greater mortality during the first 3 months from short-term infectious complications in simultaneous pancreatic-renal transplants (1). In addition, simultaneous pancreatic-renal transplant recipients had higher mortality than living-donor kidney recipients for the first 18 months after transplant, but subsequently, had lower relative risk (6). In the long term, such as 7 to 10 years after transplant, simultaneous pancreatic-renal transplant recipients show lower incidence of cardio- and cerebrovascular complications (7-11).

However, little information has been reported concerning patient and pancreatic allograft survival

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after simultaneous pancreatic-renal transplants performed at centers outside the United States. Identifying the main factors underlying higher mortality and poorer pancreatic allograft survival at such centers should help to develop effective strategies to improve results. We retrospectively analyzed 150 patients with nearly 6 years of follow-up who underwent simultaneous pancreatic-kidney transplant at a single South American center. We then performed a multivariate analysis to identify risk factors potentially compromising 1-year patient and pancreatic allograft survival rates.

Patients And Methods

Patients

Between December 2000 and August 2006, 150 patients with a pretransplant history of insulin dependence underwent a simultaneous pancreatic-renal transplant at the Universidade Federal de São Paulo in Brazil. Of these diabetic patients, 95% had type 1 diabetes, while 5% had maturity-onset diabetes of the young or latent autoimmune diabetes in adults. In Brazil, pancreas and kidney allocation for simultaneous pancreatic-renal transplant is determined by time on a waiting list, which averages 2 years. Enteric drainage was performed in 143 patients, and 7 patients underwent bladder drainage. Iliac vein or vena cava anastomosis was performed in all patients. Belzer and Eurocollins solutions were used for pancreas and kidney preservation, respectively. Only beating-heart donors were selected for organ donation, with those over 45 years old or having a history of diabetes in first-degree relatives being excluded.

Immunosuppression and postoperative management

Perioperative antimicrobial prophylaxis consisted of ceftriaxone (1 g q 12 h) and ampicillin (1 g q 6 h) for at least 1 week until the abdominal drain was removed. During surgery, a bolus of 1 g methylprednisolone was administered. Heparin prophylaxis begun 12 hours after surgery was continued for 1 week unless bleeding occurred; subcutaneous maintenance dosages ranged from 10 000 to 15 000 U/day, to maintain activated partial thromboplastin times close to normal values. Aspirin therapy (100 to 200 mg/day) was begun on day 2 or 3. Postoperative immunosuppression, begun on day 2, included tacrolimus at doses of 0.15 mg/kg,

adjusted according to interval after transplant (serum concentrations of 10-15 ng/mL for the first 30 days, 8-10 ng/mL between 31 and 90 days, and subsequently 5-10 ng/mL). Prednisone also was given (30 mg/day, with reduction by 5 mg each month until a maintenance dose of 5 mg/day was reached by the sixth month), as was mycophenolate mofetil (2 g/day) or mycophenolate sodium (1.44 g/day). Induction with mono- or polyclonal antibodies was reserved for repeat transplant when the panel reactive antibody was greater than 20% and for instances where renal cold ischemic time exceeded 24 hours.

Endpoints

Primary endpoints of the study were 1-year patient and pancreatic allograft survival rates. Secondary endpoints involved assessment of risk factors responsible for patient and pancreatic allograft survival rates. In general, all deaths within the first 90 days following transplant were assumed to be transplant-related.

Conventional risk factors for recipients included sex, age, race, duration of dialysis and of diabetes, dialysis modality (hemodialysis vs peritoneal dialysis), body mass index (BMI), and the presence of coronary atherosclerotic disease before transplant (defined as angioplasty or myocardial revascularization). Briefly, patients with diabetes duration exceeding 20 years or with well-known risk factors such as smoking, a family history of presumed sudden cardiac death, or a previous cardiac event, underwent coronary angiography. Lower-risk patients underwent non-invasive tests such as dobutamine stress echocardiography or a technetium-99m Sestamibi stress test. Body mass index was calculated as weight in kilograms divided by the square of height in meters. Conventional risk factors for deceased donors included sex, age, BMI, and general cause of brain death (cerebrovascular vs noncerebrovascular). Transplant-related risk factors included mean cold ischemic time for the pancreatic allograft, delayed graft function (DGF) for the kidney (defined as need for dialysis in the first week after transplant), acute kidney rejection according to the 1997 Banff classification, cytomegalovirus infection, acute pancreatitis (defined by a clinical presentation including fever, local pain, hyperamylasemia, and/or hyperglycemia), peripancreatic abscess, anastomotic leak, and pancreatic vascular thrombosis). Intra-abdominal infection (IAI)

included peripancreatic abscess and anastomotic leak. Rarely reversible, venous thrombosis generally presents with significant swelling of the graft, acute onset of pain, a sharp rise in the serum glucose, and increased serum amylase. Arterial thrombosis, which may involve the splenic artery, the superior mesenteric artery, or both, is characterized by an acute rise in serum glucose, a decrease in serum amylase, and usually absence of abdominal discomfort or pain. Data from deceased donors also considered included serum creatinine (mg/dL), serum amylase (U/L), serum sodium (mEq/L), administration and dose ($\mu\text{g/kg/min}$) of vasoactive drugs noradrenaline and/or dopamine.

As a matter of policy, we perform renal allograft biopsy when serum creatinine increases and/or diuresis decreases. A pancreatic allograft biopsy was performed only when unexplained hyperglycemia and/or persistently elevated serum amylase and lipase were noted.

Statistical analyses

All results are reported as the mean $\pm$ SD unless otherwise indicated. Statistical analyses were performed using SPSS software (Statistical Product and Services Solutions, version 12.0, SPSS Inc, Chicago, IL, USA). The Fisher exact test and an analysis of variance were performed for continuous variables, and the Pearson chi-square test was used for categorical variables. Correlation was assessed using a Pearson test and Cox proportional hazard regression. Log-rank analysis and Kaplan-Meier curves were used for analysis of patient and pancreatic allograft survival. For pancreatic allograft survival analysis, patient deaths were counted as graft losses irrespective of graft functional status at time of death. If the graft was removed before patient death, computation was carried out as for death with a nonfunctioning pancreas. To determine which conventional and transplant-related risk factors were associated with 1-year patient and 1-year pancreatic allograft survival, as well as with acute pancreatitis, IAI, or delayed renal graft function, all putative factors associated at P of .3 or less by univariate analysis were entered simultaneously in a backward binary logistic regression model with those factors analyzed as the dependent variable. Results were reported as odds ratio (OR) or hazard ratio (HR) with a 95% confidence interval. The statistical analyses were assumed significant for P values below .05.

Results

Demographic data from recipients and donors are described in Table 1. One-year patient, kidney, and pancreatic allograft survival rates were 82%, 80%, and 76.7%, respectively. Mean time of death was 78.2 ± 82.1 days (range, 3-259 days). Most deaths (70.4%) occurred during the first 90 days after transplant; only 8 patients died later, mainly of infection in all instances.

Table 1. Recipient and donor demographic data

Item	Value or %
Recipient items	
Sex, M:F	75:75
Age, years	34.6 ± 7.9 (range, 18-55)
Race	24% black
History of diabetes, years	21.9 ± 5.9 (range, 10-37)
Duration of dialysis, months	26.9 ± 18.6 (range, 0-108)
Hemodialysis	76.7% (n=115)
Peritoneal dialysis	17.3% (n=26)
Pre-emptive	6% (n=9)
Body mass index (kg/m^2)	21.4 ± 2.9 (range, 17.2-37.4)
Coronary atherosclerotic disease treated before transplant	11.3% (13 with angioplasty, 4 with myocardial revascularization)
Donor items	
Sex, M:F	92:58
Age, years	25.6 ± 9.4 (range, 10-46)
Cause of death	
Cerebrovascular	24.7% (n=37)
Noncerebrovascular	75.3% (n=113)
Body mass index (kg/m^2)	23.7 ± 3.2 (range, 12.5 to 28.1)
Vasoactive drugs, yes:no	92.8%: 7.2%
Dopamine	45.4% (mean dose $7.6 \mu\text{g/kg/min}$)
Noradrenaline	41.5% (mean dose $0.42 \mu\text{g/kg/min}$)
Dopamine + noradrenaline	12.3%
Dopamine + dobutamine	0.8%
Laboratory data	
Mean serum creatinine (mg/dL)	1.2 ± 0.6 (range, 0.3-3.7)
Mean serum sodium (mEq/L)	154.6 ± 16.5 (range, 119-206)
Mean serum amylase (U/L)	163.4 ± 229.3 (range, 22-1406)
Transplant-related items	
Mean kidney cold ischemic time, hours	14.03 ± 3.9 (range, 6-27)
Mean pancreas cold ischemic time, hours	14.3 ± 4.6 (range, 6-29)
Delayed kidney graft function	22.7%
Acute kidney rejection	30.7%
Cytomegalovirus infection	42%
Pancreatic complications	
Pancreatitis	13.3%
Anastomotic leak	4%
Pancreatic abscess	11%
Vascular thrombosis	4%

Cox regression analysis showed that the occurrence of DGF ($P = .001$; HR, 5.41; 95% CI, 1.98-14.77), acute renal allograft rejection ($P = .016$; HR, 3.36; 95% CI, 1.25-9.08), and IAI ($P < .0001$; HR, 4.15; 95% CI, 1.96-10.36) were the main factors that correlated with patient survival during the first year. Patient survival curves according to log-rank analyses are shown in Figure 1. Risk factors from

deceased donors had no direct influence on 1-year patient survival rates.

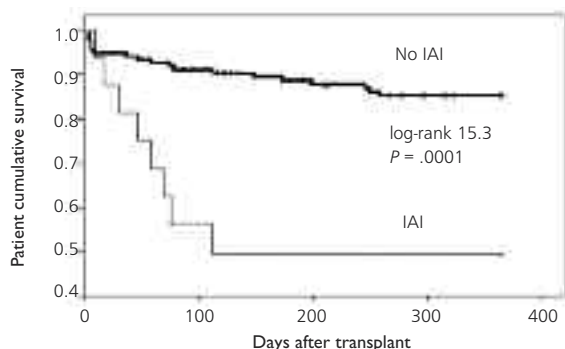


Figure 1. One-year patient survival in patients with versus without intra-abdominal infection (IAI).

A slight increase in 1-year patient survival rates, from 81.3% for the first 75 transplants to 82.7% for the last 75, was not significant ($P = 1.0$). Incidence of DGF did not change significantly (21.3% vs 24%, $P = .7$), which also was true for acute kidney rejection (26.7% vs 34.1%, $P = .29$) and peripancreatic abscess (14.7% vs 6.7%, $P = .11$).

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

Surgical complications related to the pancreatic allograft affected 60 patients (40%), requiring subsequent surgery because of bleeding (10.7%), peripancreatic abscess (8%), near-occlusion (6%), vascular thrombosis (5.3%, including 3 patients with arterial thrombosis and 5 with venous thrombosis), evisceration (4%), anastomotic leak (2.7%), negative exploration (2%), and small bowel perforation (1.3%). Of 12 patients with peripancreatic abscess, 11 required surgical drainage, including 5 with good outcomes after drainage and antimicrobial therapy. Only 1 patient required pancreatectomy. Five patients (41.7%) with peripancreatic abscess died, including 2 with sepsis secondary to IAI; 2 of the 5 previously underwent pancreatectomy. Three patients with anastomotic leaks were treated with pancreatectomy, 2 of whom subsequently died of sepsis. The fourth patient with anastomotic leak also died of sepsis, but did not undergo pancreatectomy. Acute pancreatitis occurred in 20 patients (14%) with enteric drainage and a pancreatic abscess complicated pancreatitis in 10 (50%).

During 1 year of follow-up, 35 pancreas allografts

failed (Figure 2). Death with a functioning allograft was the main category of pancreatic allograft loss (54.3%), followed by vascular thrombosis (20%), death with a nonfunctioning graft (14.3%), peripancreatic abscess or anastomotic leak (5.8%), and acute pancreatic rejection occurring asynchronously with kidney rejection (2.8%). The 5 patients dying with nonfunctioning grafts included 2 who underwent pancreatectomy for peripancreatic abscess, 2 who underwent pancreatectomy for anastomotic leak; and 1 who underwent pancreatectomy because of venous thrombosis. In 1 patient with pancreatic loss, vascular anastomosis was unsuccessful because of severe atherosclerosis in

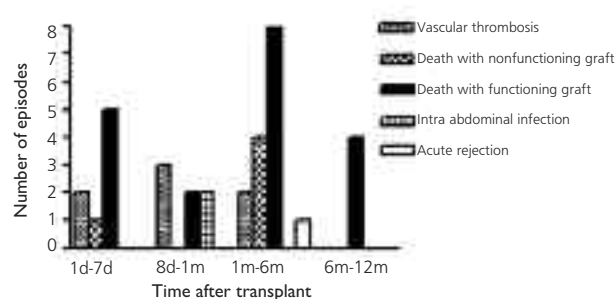


Figure 2. Causes of pancreas allograft losses during the first year after transplant, expressed as days (d) and months (m).

the iliac vessels.

Risk factors for pancreatic allograft loss during the first year included IAI ($P < .0001$; OR, 12.83; 95% CI, 3.16 to 52.16); vascular thrombosis ($P = .002$; OR, 40.55, 95% CI, 3.85-426.64); acute kidney rejection ($P = .027$; OR, 3.06; 95% CI, 1.13-8.26); donor serum sodium exceeding 155 mEq/L ($P = .02$; OR, 3.27; 95% CI, 1.2-8.92); and donor dopamine doses greater than 7.6 $\mu\text{g/kg/min}$ ($P = .046$; OR, 2.85; 95% CI, 1.01-7.98). Logistic regression to identify risk factors for IAI yielded only occurrence of DGF as the main factor ($P = .027$; OR, 3.56; 95% CI, 1.16-10.63).

Peritoneal dialysis was the only risk factor associated with acute pancreatitis ($P = .035$; OR, 3.2; 95% CI, 1.08-8.9). Among patients with acute pancreatitis, 50% developed IAI. On the other hand, no risk factor was identified as promoting pancreatic vascular thrombosis.

Discussion

In this study, 1-year patient and pancreatic survival rates after simultaneous pancreatic-renal transplant performed at a single center were analyzed. We

found that DGF involving the renal allograft, and also IAI, had important impacts on either patient or pancreatic allograft survival.

The main cause of pancreas allograft loss was patient death with a previously functioning graft, followed by technical failure and immunologic loss. By multivariate analysis, IAI, vascular thrombosis, acute kidney rejection, and deceased donor factors such as serum sodium and dopamine dose contributed to pancreatic allograft loss.

Surgical complications after simultaneous pancreatic-renal transplant occur in a third of cases at individual research centers, although data from United Network for Organ Sharing reported decreasing rates, as low as 6%, since 1988 (2, 12, 13). Occurrence of IAI, noted in 12% to 27.5% of pancreatic transplants, had a negative impact on pancreatic allograft survival, as in our patients (12-15). In some other reports, IAI had a negative impact on patient survival (12), but not in all (13). The main risk factor for IAI in our patients was DGF of the renal allograft, which could be explained partly by excessive immunosuppression in patients with dialysis or impaired renal allograft function after transplant, and also ischemia-reperfusion injury of the pancreatic allograft, as also can occur in the renal allograft.

We did not find a correlation between IAI and other reported risk factors, such as donor age greater than 45 to 50 years, cardio- or cerebrovascular donor death, donor or recipient BMI exceeding 30 kg/m², and preservation time exceeding 24 hours (12, 14, 16-18). In Brazil, the age limit for deceased donors in pancreatic transplant is 45 years, although 1 Brazilian donor was 46 years old. Indeed, brain death in our patients was attributed to trauma in two-thirds, while mean BMI (donor or recipient) differed considerably from other reports; this could explain lack of association with IAI.

However, donor serum sodium and dopamine dose at time of donation, even if not directly affecting the incidence of IAI and vascular thrombosis, influenced pancreatic allograft survival. To our knowledge, this is the first report of association between donor serum sodium and pancreatic loss. The mechanism may involve direct damage to pancreatic allograft cells by hypernatremia. In liver transplants, for example, hypernatremia may adversely affect short-term outcome (19). In addition, dopamine may have worsened pancreatic

hypoperfusion in the deceased donors, contributing to pancreatic cellular damage.

The association of acute kidney rejection with pancreatic allograft loss can be explained by effects of immunosuppressive therapy and possibly also by simultaneous acute pancreatic allograft rejection, although the latter possibility should be considered with caution since we did not perform pancreatic biopsies in most patients.

Acute pancreatitis, occurring in some 14% of our patients with enteric drainage as in previous reports (16), usually occurs in the immediate postoperative period and it is related to sepsis, ischemia-reperfusion injury, surgical trauma, or immunosuppressive therapy. Although self-limited, pancreatitis can carry complications such as peripancreatic fluid collections or areas of necrosis, which can worsen infection and require drainage. In this study, the only risk factor associated with acute pancreatitis was peritoneal dialysis. Disagreement prevails as to whether peritoneal dialysis can predispose to infection after pancreatic transplant, and possible mechanisms require further characterization (20-22). Incidence of thrombosis among our patients (5.3%) was in agreement with previous studies (23). We could not identify any risk factors for either venous or arterial thrombosis. Thrombosis has been attributed to ischemia-reperfusion injury, surgical trauma, postoperative hypoperfusion, kinking of the pancreatic vessels, and endothelial injury to the pancreatic vasculature secondary to acute rejection. Risk factors described elsewhere include older donors, cardio- and cerebrovascular causes of brain death, left-sided pancreas implantation, and arterial reconstructions other than a Y-graft (23).

In conclusion, delayed kidney allograft function and IAI had an important impact on both patient and pancreatic allograft survival. Donor factors such as dopamine dose and serum sodium, as well as acute rejection and technical failure, affected pancreatic allograft outcome after simultaneous pancreatic-renal transplant. However, death with a functioning graft was the main category of pancreas graft loss. New strategies that should be implemented include optimization of donor care, changing focus from pancreas allograft salvage to preserving the patient's in cases occurrences of IAI, and minimization of ischemia-reperfusion injury by use of anti-thymocyte globulin and pulsatile perfusion machines.

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