

Urinary Tract Diversion (UTD) in Clinical Pancreas Transplantation

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THE USUAL GUIDE to pancreas rejection is the level of blood sugar, but this is probably a very late indicator of rejection. A better indicator of rejection might therefore lead to earlier treatment of rejection and better graft survival. The aim of this study is to monitor the pancreatic graft in a group of ten patients using the urinary tract as a method of handling the pancreatic graft secretion. In nine patients a simultaneous kidney transplant was performed.

MATERIALS AND METHODS

Combined cadaveric renal and pancreatic transplantations with both of the organs provided by the same donor were performed in eight men and two women who suffered from juvenile diabetes of long standing (14 to 32 years), their age range 29 to 54 years. All had end-stage uremia due to diabetic nephropathy. One patient was a non-uremic non-kidney transplant diabetic patient with mild renal disease; the whole organ with the Ampulla of Vater was anastomosed to the bladder, pancreaticocystostomy (WOPCys). The pancreatic graft consisted of the body and tail in four patients and pancreatico-pyelostomy (PPy) for exocrine diversion. In five patients the whole pancreas without the duodenum, but preserving the sphincter of Oddi, were anastomosed to a divided ureter, pancreatico-ureterostomy (WOPUr). Both techniques have been published elsewhere.^{1,2} The simultaneously transplanted kidney anastomosed to iliac vessels was placed extraperitoneally. Immunosuppression consisted of azathioprine 2.5 mg/kg/d in the group of PPy patients and cyclosporine 12 mg/kg/d in the WOPUr and WOPCys patients. In the group of patients with simultaneous kidney transplants, the increase in the serum creatinine level, morphological (echography), and functional studies (isotope) were used as an early determinant of rejection initiating immunosuppressive therapy. Rejection was treated with 0.25 to 1 g doses of methylprednisolone given

intravenously (IV) over several days. In all patients urine amylase (U/24 h) was measured daily until the patient was discharged and monthly thereafter. Only in three patients urine lipase (U/24 h) was also measured. Renal function was monitored at frequent intervals.

RESULTS

Twenty-four hours after transplantation, graft function was excellent both in maintaining the blood glucose level and in secreting large amounts of amylase into the urine (UA), ranging between 1,028 and 21,683 U/24 h. One patient died 72 hours after transplantation with a myocardial infarction with both allografts functioning (Table 1). Another patient had an irreversible acute graft rejection three days after transplantation. Before rejection episodes all patients had elevated levels of UA. In seven of eight patients, a significant drop of UA was observed in the day of rejection. Serum glucose was normal at all times. A gradual increase of the urinary amylase occurred after antirejection therapy and could be the result of healing of an ischemic injury. A progressive increase in higher levels were reached after graft stabilization. One patient is currently with the kidney and pancreas functioning for more than 2 years after transplantation. Another patient (case 5), after reaching graft stabilization, suffered crisis of abdominal pain at 30 weeks after transplantation. Abdominal echography showed graft pancreatic pseudocyst. At this time UA decreased from $36,918 \pm 15,727$ to 12,375 U/24 h. Through a retroperitoneal approach a pancreatic pseudocyst involving the distal pancreas was removed. Two weeks later, this patient died of sepsis of unknown origin with both kidney (creatinine 1.2 mg/dL) and pancreas (UA 6600 U/24 h and normal blood glucose) functioning.

Two patients (cases 7 and 8) had two

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0041-1345/86/1805-0054\$03.00/0

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IV) over several days. In all patients urine (4 h) was measured daily until the patient died and monthly thereafter. Only in three patients lipase (U/24 h) was also measured. Renal function monitored at frequent intervals.

RESULTS

four hours after transplantation, renal function was excellent both in maintaining normal glucose level and in secreting significant amounts of amylase into the urine (UA), between 1,028 and 21,683 U/24 h. Patient AM died 72 hours after transplantation with myocardial infarction with both kidneys nonfunctioning (Table 1). Another patient had an irreversible acute graft rejection 10 weeks after transplantation. Before this episode all patients had elevated UA. In seven of eight patients, a drop of UA was observed in the immediate postoperative period. Serum glucose was normal at the time of rejection. A gradual increase of the urinary amylase occurred after antirejection therapy and was the result of healing of an acute pancreatitis. A progressive increase in UA were reached after graft stabilization. Patient AM is currently with the kidneys nonfunctioning for more than 2 years after transplantation. Another patient after reaching graft stabilization, had an episode of abdominal pain at 30 weeks after transplantation. Abdominal echography showed a pancreatic pseudocyst. At this time UA increased from 36,918 ± 15,727 to 66,000 U/24 h. Through a retroperitoneal approach a pancreatic pseudocyst involving the pancreas was removed. Two weeks later the patient died of sepsis of unknown origin with both kidney (creatinine 1.2 mg/dl) and pancreas (UA 6600 U/24 h and lipase 6600 U/24 h) functioning.

Patients AM and R.R. (cases 7 and 8) had two

Table 1. Urine Amylase (U/24 hours)

Cases	Technic	Before Rejection	Rejection	Graft Stabilization	Graft Functioning (wk)
P.A.2	K+PPy	2,132 ± 1,028	158	—	1*
R.S.3	K+PPy	3,852 ± 267	2611	14,224 ± 4,555	23†
C.C.4	K+PPy	3,375 ± 1,634	973	86,665 ± 20,927	136‡
S.LL.5	K+PPy	28,124 ± 9,109	5026	36,918 ± 15,727	36§
A.M.7	K+WOPUr	1,224 ± 144	1 st 1,050 2 nd 8,208	34,716 ± 22,978 123,939 ± 83,665	33
R.R.8	K+WOPUr	27,792 ± 20,735	1 st 19,106 2 nd 324	— 7,327 ± 3,845	28
E.O.9	K+WOPUr	26,767 ± 21,858	18,971	—	3¶
A.C.10	WOPCys	17,945 ± 11,328	2,566	—	5§

Abbreviation: K, kidney.

*Graft vascular occlusion.

†Graft failure related to patient's failure to continue the immunosuppressive drugs.

‡Currently functioning.

§Died of sepsis with kidney and pancreas functioning.

||Graft failure consequence of immunological rejection.

¶Reoperation (partial pancreatic necrosis and ascitis); died of septic shock.

episodes of rejection and during this period UA dropped abruptly. Although the treatment of rejection succeeded to increase UA to a significant level, all of them eventually had graft failure, for immunologic reasons.

Patient AM (case 7) had graft stabilization four weeks until 29 weeks after transplantation with a mean of UA 1,293,939 ± 83,665 U/24 h. He was readmitted to the hospital 30 weeks after transplantation with abdominal pain and distension and rebound tenderness. Serum amylase and lipase were clearly elevated 1,940 U (NV < 200) and 507 U (NV < 70), respectively. An exploratory laparotomy was indicated and the only clinical finding was an enlarged pancreatic graft with dark discoloration in the tail of the pancreas. Blood glucose remained normal and stable in spite of a significant decrease of UA (39,927 ± 23,307) and renal function was also normal. However, four weeks later the patient suffered of rejected episodes of abdominal pain and elevations of serum pancreatic enzymes and hyperglycemia was detected requiring the resumption of insulin (20 to 30 U). This clinical

picture led to indicate pancreas transplantectomy at 46 weeks after transplantation.

DISCUSSION

Urinary tract diversion^{1,2,3} has the advantage of allowing easy monitoring of graft function (exocrine pancreas). PPy is a feasible alternative technique for the management of exocrine pancreatic secretion only in patients with end-stage renal failure. The disadvantage of this method is that nephrectomy has to be performed in order to use the pelvis as a drainage conduit. The pancreas can be placed in a paratopic position. WOPCys is our technique of choice in non-uremic patients. In patients with simultaneous kidney transplant we prefer WOPUr avoiding the bladder, which is frequently contaminated.

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