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The role of early baseline computed tomography in the interpretation of morphological changes after kidney-pancreas transplantation

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Abstract. In a prospective study, 17 early baseline computed tomography (CT) scans were obtained 2 or 3 days after simultaneous kidney-pancreas transplantation. Morphological changes and their relevance to the early detection of graft rejection and complications were evaluated. The pancreatic grafts were enlarged and showed signs of mild pancreatitis. Serial scans obtained during the first renal graft rejection episode were compared with the baseline CT scans ($n = 7$). They showed a significant increase in pancreatic graft size in the case of biopsy-proven severe renal graft rejection ($P = 0.008$). Normally functioning pancreatic allografts showed a 15%–40% decrease in size 1–6 months after transplantation. We conclude that the morphological changes observed early after transplantation are compatible with mild pancreatitis, which may contribute to the development of pancreatic graft thrombosis. There is an increase in the number of morphological changes during severe rejection, yet enlarged pancreatic grafts appear to recover from transplantation-related damage and regain their normal size without signs of atrophy.

Key words: Pancreas transplantation, computed tomography – Kidney-pancreas transplantation, computed to mography – Computed tomography, kidney-pancreas transplantation – Pancreatitis, computed tomography

Introduction

Transplantation of the pancreas for the treatment of type I diabetes mellitus is an increasingly accepted therapeutic option for preventing or delaying complications associated with this disease and for improving the quality of life. The results of pancreas transplantation have improved impressively during the last 5 years, especially those of simultaneous kidney-pancreas transplantation [22, 23]. These improvements are due to better surgical techniques and more effective immunosuppressive treatment. However, there is a high complication rate in the early postoperative period during which graft thrombosis, rejection, and pancreatitis with the development of ascites, pseudocyts, and abscesses may occur. Graft thrombosis and rejection are the most common causes of graft loss [20, 23]. To evaluate morphological changes in the graft and their relevance to the early detection of graft rejection and complications, radiologic assessment frequently becomes necessary. The value of computed tomography (CT), ultrasound, scintigraphy, and magnetic resonance imaging has already been established [5, 7, 10, 12, 14, 16, 17, 25, 27]. These modalities are sensitive in detecting parenchymal abnormalities and changes in graft size and margination. Yet even when these techniques are used, early detection of graft rejection remains difficult [11, 26].

Sequential radiologic examinations, starting with an early baseline study, may be helpful in better understanding the relevance of changes in size and appearance of the allograft. However, early post-transplantation baseline CT scans are rarely obtained. Without such data on donor organs, it is not possible to use size and morphology as indicators of graft dysfunction or complications [14, 27].

The purpose of this prospective study was to analyze the changes in size and appearance of the pancreatic graft in the early postoperative period and to determine the value of serial CT scans in the assessment of complications, including rejection. A total of 87 abdominal and pelvic CT examinations in 17 patients are described and correlated with clinical and laboratory data.

Patients and methods

Between 1987 and 1990, 22 diabetic patients (16 male, 6 female) with end-stage renal failure received ABO-compatible grafts. In each case both organs came from the same cadaveric donor and were transplanted simultaneously. At the time of transplantation the

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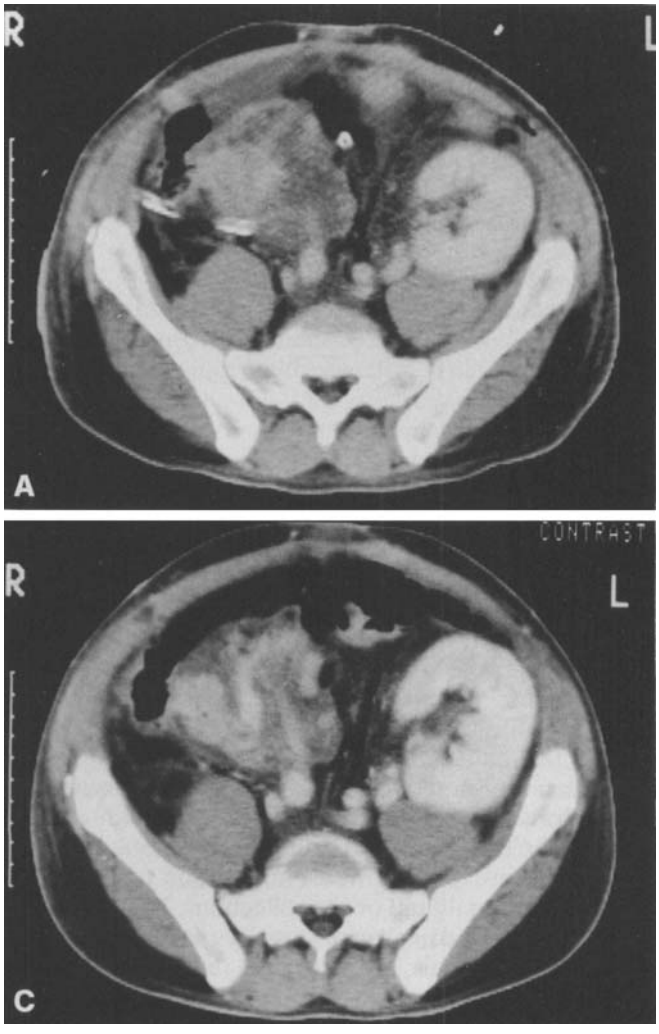


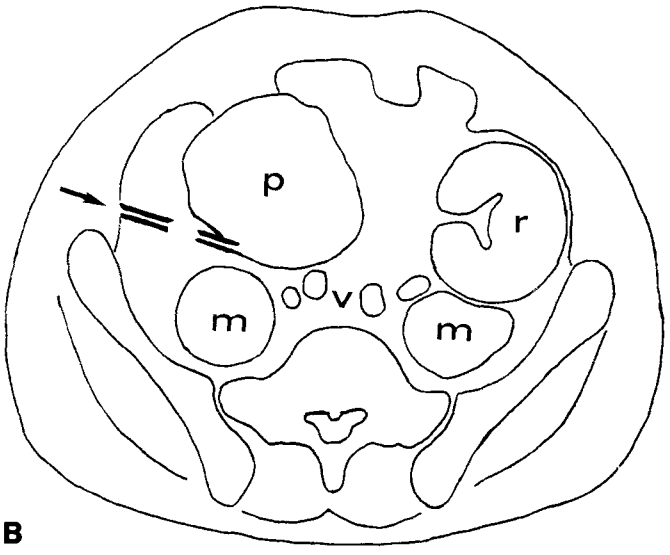


Fig. 1. a, b Contrast-enhanced baseline CT scan performed on the 2nd postoperative day. **c** CT scan performed during a kidney biopsy-proven first rejection episode (day 8) demonstrating enlargement of the pancreatic graft with parenchymal inhomogeneity and streaky infiltration of the pancreatic fat. The follow-up CT scan shows an increase in renal and pancreatic graft size indicating simultaneous rejection. *Arrow* indicates drain. *p*, pancreatic graft; *r*, renal graft; *m*, psoas muscle; *v*, iliac vessels

mean recipient age was 36.5 years (range 23–50 years). Mean donor age was 31 years (range 13–57 years).

The first 5 grafts were preserved with Euro-Collins (EC) solution and the remaining 17 grafts with University of Wisconsin (UW) solution. Mean cold ischemia time was 9 h 30 min (range 3 h 20 min–15 h). The mean warm ischemia time for the pancreatic graft was 24 min (range 18–42 min). The pancreatic graft was placed intraperitoneally in an upright position and vascular anastomoses were done in end-to-side fashion to the iliac vessels. The exocrine drainage was directed to the urinary bladder via a periampullary segment of donor duodenum. The transplanted kidney was placed retroperitoneally in the contralateral iliac fossa.

Table 1. Computed tomography findings in 17 patients 2–3 days after simultaneous kidney-pancreas transplantation

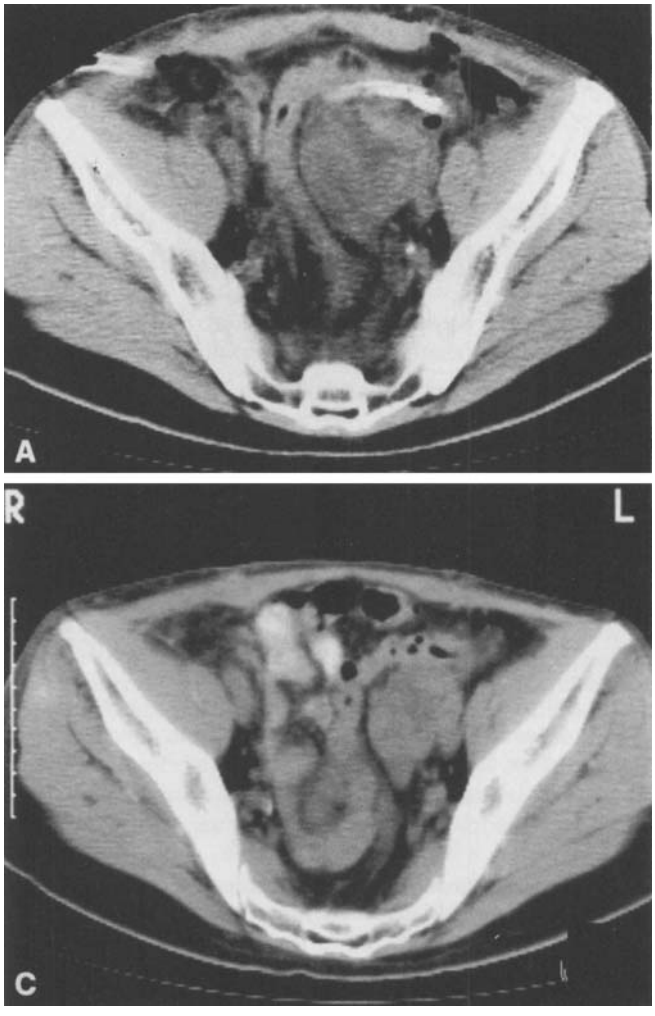
Pancreatic aspects	Mean size 6.2 cm	
	Parenchymal inhomogeneity	60%
	Contour irregularities	82%
Peripancreatic abnormalities	Small fluid collections	65%
	Inflammatory changes in peripancreatic fat	94%
Renal aspects	Mean size 6.3 cm	
	Contour irregularities	12%
Perirenal abnormalities	Small fluid collections	29%
Miscellaneous	Ascites/pleural effusion	35%
	Intestinal distention	18%
	Intraperitoneal air collections	100%

In the 1st month after transplantation 62 CT scans were obtained. Intravenous contrast material (100 ml Télébrix 350, Laboratoire Guerbet, France) was administered during 32 examinations for evaluation of pancreatic enhancement. Bowel opacification was used whenever the patient was able to accommodate the fluid intake (2% Télébrix 350, 500 ml 1 h before scanning and 250 ml 15 min before scanning). A Tomoscan 350 scanner (Philips) was used to obtain contiguous slices with a thickness of 9 mm.

To allow meaningful interpreation of CT findings in the case of rejection or complications, baseline CT scans were performed. In 17 cases, a baseline CT scan was obtained on the 2nd or 3rd day after transplantation. Five patients were not availiable for baseline CT scanning because early reoperation for urinary leakage, graft thrombosis, or hemorrhage precluded such an investigation. Successive scans were performed when indicated, such as for possible peripancreatic fluid collections or abscesses, rejection, or hemorrhage.

CT findings were evaluated both qualitatively and quantitatively. For qualitative analysis, baseline and subsequent scans were subject to blind review by two radiologists according to the prognostic signs in acute pancreatitis described by Balthazar et al. [2, 3]. The scans were evaluated for graft size, parenchymal attenuation, contour irregularities, peripancreatic and perirenal abnormalities, and abnormal fluid and gas collections. For quantitative analysis the size of the pancreatic head was measured in anteroposterior direction and perpendicular to this direction. The size of the pancreas was defined as the mean of these two measurements. Successive measurements of the same pancreas were performed on the same level. The size of the kidney was determined in a similar fashion.

Rejection of the pancreatic graft was diagnosed if 24-h urine amylase levels showed a decrease of at least 25% compared to those of the previous day. Pancreatic graft biopsies were not performed. For the transplanted kidney, the diagnosis of acute rejection was based on an elevation of serum creatinine and confirmed by renal al-



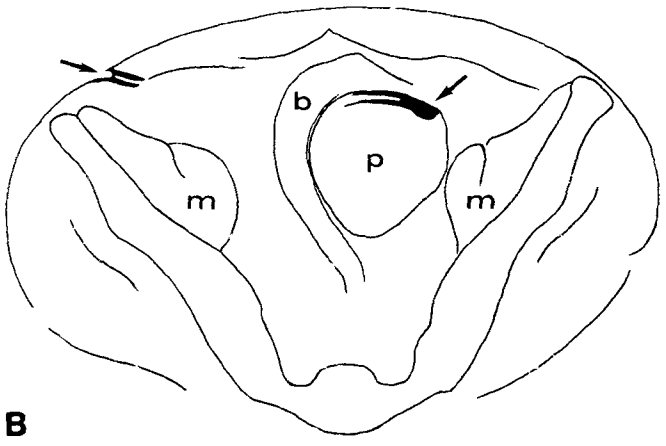


Fig. 2.a, b Early baseline CT scan. **c** CT scan performed 27 days after transplantation in the absence of complications. Note enlargement of the pancreatic graft with parenchymal inhomogeneity in the baseline scan and marked decrease in pancreatic graft size with decreased parenchymal inhomogeneity in the follow-up scan. Note different field of view. *Arrows* indicate drains. *p*, pancreatic graft; *m*, ilio-psoas muscle; *b*, bowel loop

lograft biopsy findings. Rejection was graded as mild, moderate, or severe depending on the intensity of vasculitis and mononuclear cell infiltration.

In 13 cases a CT scan of both grafts was obtained during the first or subsequent biopsy-proven kidney rejection episodes and compared with the baseline scan for the characteristics already described. Comparison was also made with CT scans that were performed 1–6 months after transplantation. Changes in size of the transplanted kidney and pancreas were analyzed statistically using Student's *t*-test with paired samples.

Results

Findings on early postoperative baseline CT scans

In 17 patients a baseline CT scan was performed 2–3 days postoperatively to assess the appearance of a normally functioning graft (Figs. 1, 2). The pancreas showed an inhomogeneous parenchyma in most cases. Intravenous contrast was administered during seven baseline examinations, six of which showed parenchymal inhomogeneity. In all postoperative cases peripancreatic abnormalities were observed that generally consisted of small fluid collections and minimal streaky infiltration of the peripancreatic fat. In most cases the peritransplant fluid collections were presented as a fluid density halo around the graft. More extensive fluid collections were noted only exceptionally. The region of the duodenal segment was identified in all cases and showed a normal soft tissue density without abnormal fluid or gas collections. The pancreatic head was enlarged in all cases, its size ranging from 4.2 to 7.7 cm (mean 6.2 cm). The size of the renal graft appeared normal in all cases, ranging from 5.2 to 7.6 cm (mean 6.3 cm). The CT findings are summarized in Table 1.

At the time of baseline CT sanning, the mean serum amylase was 690 U/l (range 167–4428 U/l; normal range 70–300 U/l). Mean fasting blood glucose was 5.9 mmol/l (range 4.0–9.4 mmol/l) and mean serum creatinine was 251 μmol/l (range 99–1237 μmol/l).

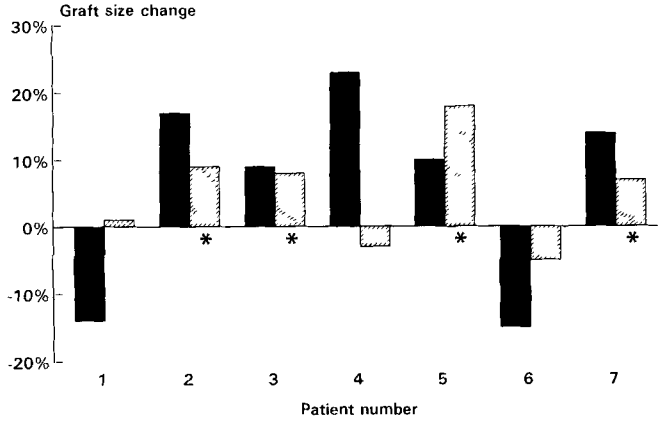


Fig. 3. Illustration of the changes in pancreatic (■) and renal (▨) graft size in seven patients during the first rejection episode of the kidney when compared to the graft size in the baseline situation. Note significant increases in graft size during histologically severe renal graft rejection, which are marked with an asterisk

Frist rejection episodes in the early postoperative period

All patients experienced one or more rejection episodes in the first few weeks after transplantation. The first episode started between day 7 and day 12. In seven cases of a biopsy-proven first kidney rejection period, CT scans were available. In four of these patients, histological examination showed severe renal graft rejection. In these cases there was a significant increase in renal graft size (8%–18%; $P = 0.014$) and in pancreatic graft size (9%–17%; $P = 0.008$) when compared to the baseline situation. In the remaining three patients, renal biopsies showed histological evidence of mild rejection. These renal grafts were not enlarged while two pancreatic grafts had decreased in size by 15% and one was enlarged by 23%. This latter graft was lost on day 41; pathological evaluation of the pancreatic graft showed histopathological changes characteristic of severe pancreatitis, indicating that the enlargement of the pancreas in the early postoperative phase was probably caused by mild pancreatitis. The changes in graft size during the first rejection episodes are summarized in Fig. 3. An example is shown in Fig. 1.

The pancreatic parenchymal inhomogeneity and peripancreatic abnormalities observed during the first rejection episode were less extensive than the baseline CT findings. Abnormal findings on serial scans obtained during the second or even the third rejection episodes tended to persist for weeks. Renal graft size during these rejection episodes had increased only in cases of severe rejection.

Changes in graft size during a longer follow-up period

In spite of several rejection episodes, pancreatic grafts tended to be smaller than they were at baseline (Fig. 2). In the absence of complications, eight pancreatic transplants with a normal endocrine function showed a 15%–40% decrease in size 1–6 months after transplantation ($P = 0.001$) as compared to early postoperative baseline CT scans. There was no statistically significant change in renal graft size during the same period. The graft size changes are shown in Fig. 4. Six pancreatic grafts subsequently had normal function. In two patients the pancreatic graft was lost due to rejection.

An example of the changes in renal and pancreatic graft size in a single patient from the 2nd day until more than a year after transplantation is shown in Fig. 5. During the first rejection episode, which started on day 8, there was an increase in renal and pancreatic graft size. During antirejection treatment, the transplanted kidney and pancreas decreased in size. The renal graft returned to its normal size, as measured at baseline, while the pancreatic graft tended to show a slow and progressive decrease in size to normal measurements within 3 months' time.

Complications and incidental findings

Seven out of 22 pancreatic grafts failed. The causes of failure were thrombosis ($n = 3$), rejection ($n = 3$), and pancreatitis ($n = 1$). Three patients lost their renal and

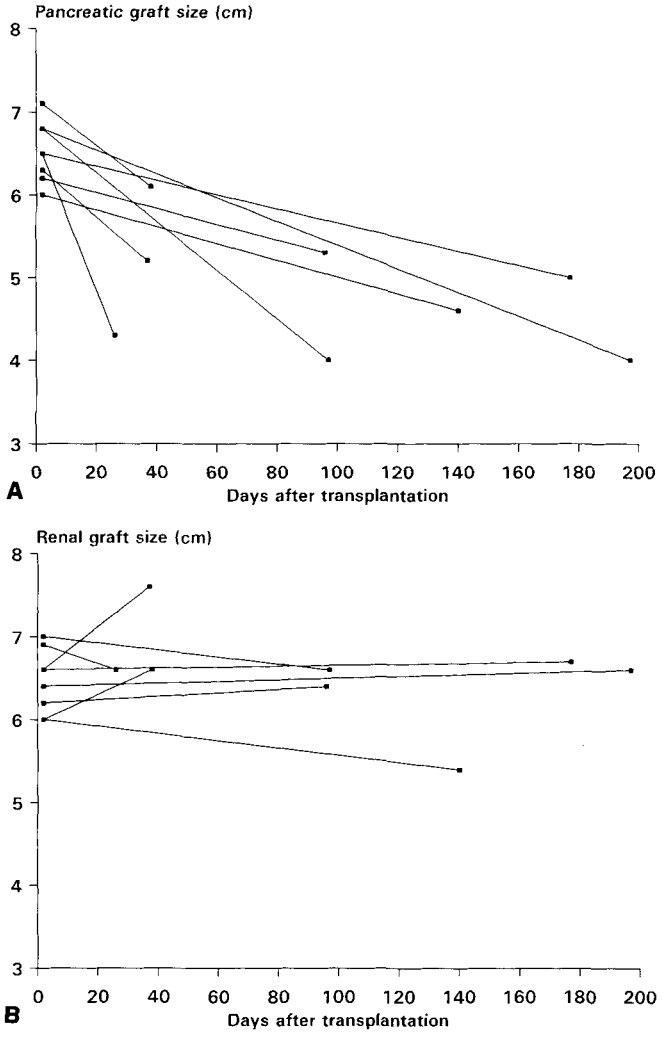


Fig. 4. a, b. Changes in **a** pancreatic and **b** renal graft size during a prolonged follow-up period in the absence of complications. Note significant ($P = 0.001$) decrease in pancreatic graft size over time. Changes in renal graft size are not statistically significant

pancreatic grafts almost simultaneously, two due to rejection and one due to infection. One transplanted kidney was rejected solitarily. None of the patients died.

In one patient a pseudocyst in the tail of the pancreatic graft was detected and percutaneous needle aspiration under CT guidance was performed to obtain material for microbiological analysis. The material was colonized with *Candida albicans*. A week after aspiration surgical drainage was required.

A leaking duodenal segment was identified in one case using follow-up CT by demonstrating abnormal gas collections in and around the segment. In this patient fluid collections developed especially around the liver, the renal graft, and the tail of the pancreatic graft. After administration of intravenous contrast material, the pancreatic graft showed an improved visualization of parenchymal inhomogeneity, which had increased when compared to the baseline CT scan. Surgical intervention confirmed leakage of the duodenal segment, probably due to ischemia or rejection. In the subsequent period an abscess developed in the pancreatic head, which was percutaneously drained.

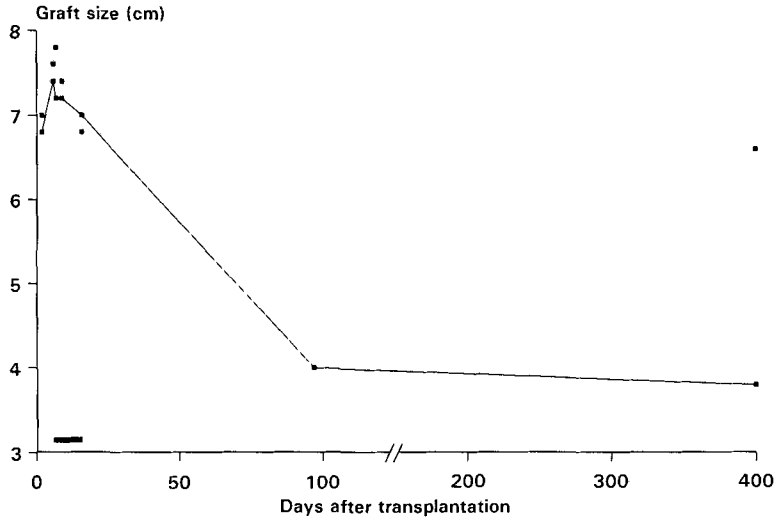


Fig. 5. Changes in pancreatic (—■—) and renal (----■----) graft size in one patient for a period of 400 days showing a simultaneous increase in the size of both organs during a reversible biopsy-proven rejection episode *(bar)*. Note the normalization of pancreatic graft size

Rejection and pancreatitis were manifested as nonspecific pancreatic graft enlargement with less distinct gland margins and inflammatory changes in the peripancreatic fat.

Discussion

The first objective of this study was to describe and analyze the early CT findings following simultaneous kidney-pancreas transplantation and to assess the clinical usefulness of CT for the monitoring of rejection or complications. Early postoperative baseline CT studies enabled us to interpret changes in size and other aspects of the graft as they occur in the post-transplantation period.

Under normal conditions, after correcting for donor age, the anteroposterior diameter of the pancreatic head ranges from 2.4 to 3.3 cm [8]. Shortly after transplantation, the pancreatic graft was enlarged, due to edema probably resulting from reperfusion. This early increase in size contrasted with the virtual absence of postoperative changes in renal graft size. These findings support the tendency for the pancreatic graft to develop early graft thrombosis. The pancreas is a low-flow organ, and edema will tend to further reduce pancreatic blood flow to a critical level of perfusion.

The normal CT appearance of the pancreatic graft at different stages after transplantation has been described by other authors as homogeneous and well marginated, with a parenchymal density similar to that of the native gland [14, 17]. In these studies, however, asymptomatic graft recipients were not studied, and routine CT scanning was not performed in the early postoperative phase. On the basis of magnetic resonance imaging performed at different intervals within 4 weeks after transplantation, the transplanted pancreas has been described as enlarged and inhomogeneous with surrounding fluid collections [27]. The present study showed similar findings when CT scanning was carried out on the 2nd or 3rd day after transplantation. Although intra-abdominal fluid has often been observed after pancreatic transplantation [10, 12], the high frequency of 64 % seen in this study had not previously been observed. These fluid collections have a high amylase content (over 100 000 U/l) and spontaneous resolution of these collections normally occurs within 2–4 weeks after transplantation. Pancreatic inhomogeneity and peripancreatic inflammation on CT scans of pancreatic grafts are similar in appearance to pancreatitis in the native gland [14]. The elevation of serum amylase levels (mean 690 U/l), the focal or diffuse enlargement of the gland with intrinsic pancreatic abnormalities, and the inflammatory changes in the peripancreatic fat are characteristics of mild edematous pancreatitis. This postoperative pancreatitis may not be accompanied by abdominal pain because of graft denervation and analgesic therapy administered early after operation. Early post-transplantation pancreatitis can be initiated during organ donor care, graft preservation, and surgical handling. This illustrates the importance of optimal donor care, organ selection, and graft implantation.

Routine baseline CT scans provided us with detailed information on early but nonspecific morphological changes in the pancreatic graft. The early findings have had no influence on patient management but have increased our awareness of the degree to which postoperative pancreatic edema is critical with respect to pancreatic graft perfusion and thrombosis [18].

The usefulness of CT scanning in the detection of rejection has been questioned [17, 26], and the detection of pancreatic graft rejection is still a major clinical problem. In renal transplantation patients, the combination of increased serum creatinine levels, a rise in temperature, and painful swelling of the graft are sensitive parameters for monitoring graft rejection. Rejection can easily be proven by biopsy of the renal graft. In contrast, in the case of pancreatic graft rejection, no noninvasive test is conclusive for early diagnosis. Data on urinary amylase, serum amylase, and serum glucose levels are of limited value. Changes in amylase levels are not specific for the detection of a change in function, e.g., of rejection [15, 24]. Hyperglycemia occurs late in the course of graft destruction, when most of the endocrine tissue has been destroyed, and can

therefore not be used as a parameter for monitoring graft rejection [9, 19].

As long as the renal graft is rejected before or simultaneously with the pancreatic graft, renal graft function can serve as an indicator of pancreatic graft rejection. Thus, changes in graft morphology observed on CT scans do not increase the accuracy of the rejection diagnosis. The two grafts may, however be rejected independently [13, 21], so that early detection of isolated pancreatic rejection is essential. Percutaneous large-gauge core biopsy of the transplanted pancreas may be safely performed with CT guidance [4], and changes in pancreatic graft size may be helpful in timing such a biopsy procedure.

All of our patients had an early rejection episode, starting in the 2nd week after transplantation. Because baseline CT scans had previously been recorded, it was possible to relate changes in graft size and appearance to rejection. Others have reported that during rejection the transplanted pancreas is usually slightly enlarged [17]; in some cases such enlargement may be absent [27]. In our study, histopathological examination of four renal biopsies showed severe rejection and both the renal and pancreatic grafts were enlarged (Fig. 3, cases 2, 3, 5, and 7). It is conceivable that both grafts were being rejected simultaneously. A reduction in the size of the pancreas was observed during histologically mild rejection of the kidney (Fig. 3, cases 1 and 6). These findings indicate that rejection of the pancreas may occur at a later stage or may be milder than that of the renal graft. This observation is supported by the suggestion that the effects of immune activation in the pancreatic graft appear more slowly than in the transplanted kidney [1]. One pancreatic graft was enlarged as a result of severe and persistent pancreatitis (Fig. 3, case 4). In this patient a rise in serum amylase and urine amylase was observed. Histopathological examination after transplantectomy confirmed the diagnosis of severe pancreatitis. The diagnosis of isolated pancreatic graft rejection was not made. This would have required percutaneous pancreatic biopsies, which were not performed on our patients.

The second objective of our study was to assess changes in pancreatic graft size during a longer follow-up period in order to document long-term effects of early pancreatic enlargement on graft function and to address the question of long-term graft atrophy after pancreatic denervation and exocrine deviation.

Measuring graft size during subsequent rejection periods was of limited value because pancreatic grafts tended to decrease in size during the first few months after transplantation. During acute rejection two different processes appear to counteract each other. On the one hand, recovery from pancreatitis as a result of the transplantation procedure leads to a decrease in pancreatic size and, on the other hand, graft rejection with cellular infiltration and edema may lead to an increase in graft size.

Changes in pancreatic graft size over a longer period of time have occasionally been described. However, these reports mostly concern nonfunctioning grafts with a reduced size due to atrophy [14], although severely atrophic glands with normal endocrine function are also described [27]. Whether the enlarged gland returns to its normal size within a few months following transplantation seems to depend on intervening complications. In the present study most grafts returned to their normal size permanently and had a normal endocrine function. Despite graft denervation and exocrine deviation, there were no signs of pancreatic atrophy.

It is generally accepted that CT scanning can play an important role in the evaluation of complications after a combined kidney-pancreas transplantation, especially in detecting fluid collections or abscesses, but that it is less significant in evaluating suspected graft rejection [6, 10, 12, 17]. Nevertheless, the present study demonstrates that an early baseline CT examination performed within a few days after transplantation may be helpful in assessing short-term changes in pancreatic size that may indicate acute rejection; however, it plays a less important role in the detection of rejection at a later stage. Furthermore, this study shows that normally functioning pancreatic transplants tend to decrease in size and return to normal within a few months with no signs of pancreatic atrophy at that stage. Finally, CT scanning is well suited for the detection of clinically important complications after combined kidney-pancreas transplantation, especially when early baseline scans are available for comparison.

References

1. Allen RDM, Wilson TG, Grierson JM, Greenberg ML, Earl MJ, Nankivell BJ, Pearl TA, Chapman JR (1991) Percutaneous biopsy of bladder-drained pancreas transplants. Transplantation 51: 1213–1216
2. Balthazar EJ, Ranson JHC, Naidich DP, Megibow AJ, Caccavale R, Cooper MM (1985) Acute pancreatitis: prognostic value of CT. Radiology 156: 767–772
3. Balthazar EJ, Robinson DL, Megibow AJ, Ranson JHC (1990) Acute pancreatitis: value of CT in establishing prognosis. Radiology 174: 331–336
4. Bernardino M, Fernandez M, Neylan J, Hertzler G, Whelchel J, Olson R (1990) Pancreatic transplants: CT-guided biopsy. Radiology 177: 709–711
5. Catafau AM, Lomena FJ, Ricart MJ, Pons F, Piera C, Pavia J, Moragas M, Garcia A, Herranz R, Andreu J, Setoain J (1989) Indium-111-labeled platelets in monitoring human pancreatic transplants. J Nucl Med 30: 1470–1475
6. Crass JR, Feinberg SB, Sutherland DER, Loken M, Gedgaudas EG (1982) Radiology of the human segmental pancreatic transplant. Gastrointest Radiol 7: 153–158
7. Freeny PC (1989) Classification of pancreatitis. Radiol Clin North Am 27: 1–3
8. Heuck A, Maubach PA, Reiser M, Feuerbach S, Allgayer B, Lukas P, Kahn T (1987) Age-related morphology of the normal pancreas on computed tomography. Gastrointest Radiol 12: 18–22
9. Kyriakides GK, Rabinovitch A, Olson L, Mintz D, Rapaport FT, Miller J (1981) Intraperitoneal segmental pancreatic allografts with unligated duct in dogs: the role of histocompatibility for long-term survival. Transplant Proc 13: 810–811
10. Letourneau JG, Maile CW, Sutherland DER, Feinberg SB (1987) Ultrasound and computed tomography in the evaluation of pancreatic transplantation. Radiol Clin North Am 25: 345–355
11. Low A, Kuni CC, Letourneau JG (1990) Pancreas transplant imaging: an overview. Am J Roentgenol 155: 13–21
12. Maile CW, Crass JR, Frick MP, Feinberg SB, Goldberg ME, Sutherland DER (1985) CT of pancreas transplantation. Invest Radiol 20: 609–612

13. Margreiter R, Klima G, Bösmüller C, Königsrainer A, Schmid T, Steiner E (1989) Rejection of kidney and pancreas after pancreas-kidney transplantation. Diabetes 38 [Suppl 1]: 79–81
14. Moulton JS, Munda R, Weiss MA, Lubbers DJ (1989) Pancreatic transplants: CT with clinical and pathologic correlation. Radiology 172: 21–26
15. Munn SR, Engen DE, Barr D, Carpenter HA, Perkins JD (1990) Differential diagnosis of hypoamylasuria in pancreas allograft recipients with urinary exocrine drainage. Transplantation 49: 359–362
16. Nobrega J, Halvorsen RA, Letourneau JG, Snover D, Sutherland DER (1990) Cystic central necrosis of transplanted pancreas. Gastrointest Radiol 15: 202–204
17. Patel B, Markivee CR, Mahanta B, Vas W, George E, Garvin P (1988) Pancreatic transplantation: scintigraphy, US, and CT. Radiology 167: 685–687
18. Schaapherder AFM, Oosterhout ECAA van, Bode PJ, Woude FJ van der, Lemkes HHPJ, Gooszen HG (1993) Pancreatic graft survival after arterial graft thrombosis in simultaneous renal-pancreatic transplantation. Clin Transplant 7: 37–42
19. Schulak JA, Drevyanko TF (1991) Experimental pancreas allograft rejection: correlation between histologic and functional rejection and the efficacy of antirejection therapy. Surgery 98: 330–336
20. Snider JF, Hunter DW, Kuni CC, Castaneda Zuniga WR, Letourneau JG (1991) Pancreatic transplantation: radiologic evaluation of vascular complications. Radiology 178: 749–753
21. Sollinger HW, Stratta RJ, D'Alessandro AM, Kalayoglu M, Pirsch JD, Belzer FO (1988) Experience with simultaneous pancreas-kidney transplantation. Ann Surg 208: 475–482
22. Sutherland DER, Moudry-Munns KC (1990) International Pancreas transplantation registry analysis. Transplant Proc 22: 571–574
23. Sutherland DER, Chow SY, Moudry-Munns KC (1989) International pancreas transplant registry report – 1988. Clin Transplant 3: 129–149
24. Tom WW, Munda R, First MR, Alexander JW (1987) Physiologic consequences of pancreatic allograft exocrine drainage into the urinary tract. Transplant Proc 19: 2339–2342
25. Vahey TN, Glazer GM, Francis IR, Li K, Dafoe DC, Aisen AM, Smid DM (1988) MR diagnosis of pancreatic transplant rejection. Am J Roentgenol 150: 557–560
26. Yuh WTC, Wiese JA, Abu-Yousef MM, Rezai K, Sato Y, Berbaum KS, Kao SCS, Hunsicker LG, Corry RJ (1988) Pancreatic transplant imaging. Radiology 167: 679–683
27. Yuh WTC, Hunsicker LG, Nghiem DD, Sato Y, Smith JL, Wright FH, Corry RJ (1989) Pancreatic transplants: evaluation with MR imaging. Radiology 170: 171–177