Abdominal Imaging

Published online: 19 June 2010

Abdom Imaging (2011) 36:206–214
DOI: 10.1007/s00261-010-9632-6

Complications following pancreatic transplantations: imaging features

S. Y. Liong,[1] R. E. Dixon,[2] N. Chalmers,[1] A. Tavakoli,[3] T. Augustine,[3] S. O'Shea[1]

[1]Department of Clinical Radiology, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL, UK
[2]Department of Clinical Radiology, Royal Preston Hospital, Sharoe Green Lane North, Fulwood, Preston PR2 9HT, UK
[3]Department of Transplant Surgery, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL, UK

Abstract

Whole organ vascularized pancreatic transplant is a recognized treatment for diabetes and is increasingly being performed worldwide. The procedure itself is complex and is associated with significant mortality and morbidity. Despite improvements in surgical techniques, postoperative complications of pancreatic transplantation are still common and include graft rejection, pancreatitis, peripancreatic fluid collections, exocrine leaks, vascular thrombosis, and hemorrhage. In this pictorial essay, we review clinical presentation and imaging features of these complications. We also briefly discuss technique and complications of islet cell transplants.

Key words: Pancreas—Transplant—Complications—Computed tomography—Magnetic resonance—Ultrasound

The first whole organ pancreas transplant was performed in Minnesota in 1966 by Kelly and colleagues [1]. Since then, advancements in surgical techniques and immunosuppressive regimens have resulted in improved graft survival rates. As of 2004, more than 23000 pancreatic transplantations were reported to the International Pancreas Transplant Registry [2]. Within the United States alone, 13719 pancreas transplants were reported to the Scientific Registry of Transplant Recipients between 1998 and 2007 [3].

Most recipients are patients with type 1 diabetes but a small subset (7.7%) have type 2 disease [4]. Patients are considered for a pancreas transplant if they have or are at high risk of secondary complications of diabetes (e.g., nephropathy, retinopathy, neuropathy), have disabling or life-threatening hypoglycemic unawareness, or are likely to develop these and are judged to be fit enough to survive the operation [5].

The goal of pancreas transplantation is to safely restore normoglycemia, rendering the diabetic patient free of insulin injections. Studies have reported improved long-term survival in type 1 diabetic patients following pancreatic transplantation compared with patients with transplanted kidney alone [6, 7] or on hemodialysis [7], with beneficial effects on diabetic nephropathy, retinopathy, neuropathy, and coronary artery disease [5].

However, it is important to remember that pancreatic transplantation is a major operation associated with significant mortality and morbidity. Long-term advantages of transplantation have to be balanced against the potential complications associated with the surgical procedure itself and the long-term requirement for immunosuppression.

Transplant procedure and surgical anatomy

Knowledge of surgical anatomy is key to the interpretation of images in the post-transplant patient. There are three main types of pancreas transplantation.

- *Simultaneous pancreas and kidney transplant* (*SPK*)—pancreas and kidney (usually from the same deceased donor) are transplanted simultaneously. This is the most commonly performed operation, accounting for 66% of pancreatic transplants in the United States [3, 4].
- *Pancreas-after-kidney transplant* (*PAK*)—donor pancreas transplanted after a previous successful donor kidney transplant. This is the second most commonly performed mode of pancreatic transplant, accounting for 25% of transplants in the United States [3, 4].
- *Pancreas transplant alone* (*PTA*)—donor pancreas transplanted only, usually performed in type 1 diabetic patients with frequent severe hypoglycemic episodes, but with adequate kidney function.

Correspondence to: S. Y. Liong; *email:* sue_liong@yahoo.co.uk

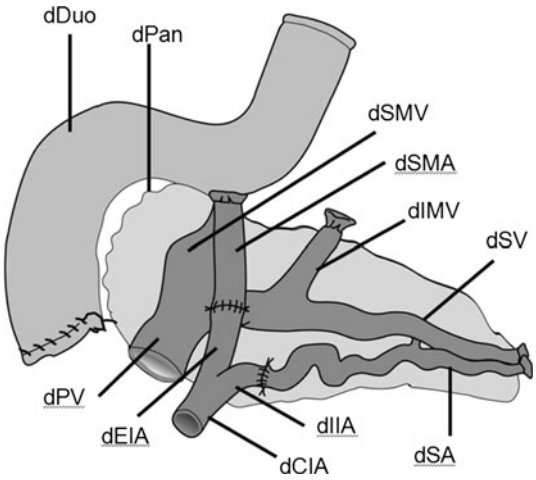

Fig. 1. Pancreas, duodenum, and vessels procured from the donor—pancreas (dPan), c-loop of duodenum (dDuo), splenic vein (dSV), inferior mesenteric vein (dIMV), superior mesenteric vein (dSMV), portal vein (dPV), superior mesenteric artery (dSMA), splenic artery (dSA), and Y-graft comprising common iliac artery (dCIA), external iliac artery (dEIA), internal iliac artery (dIIA).

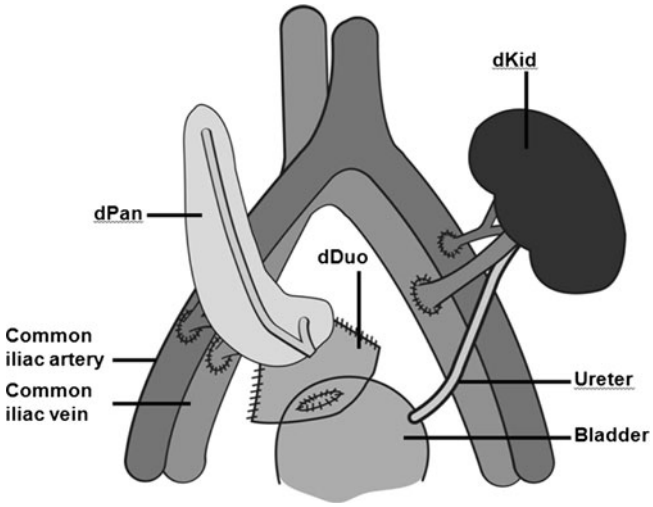

Fig. 2. Bladder-drainage of donor pancreatic exocrine secretions—donor pancreas (dPan), duodenum (dDuo), kidney (dKid). Note arterial and venous anastomoses of the transplanted organs to the common iliac artery and veins.

Organ procurement

The donor pancreas, C-loop of the duodenum (Fig. 1), and spleen are retrieved en bloc from the donor. The splenic and superior mesenteric arteries are marked for bench reconstruction. The spleen is subsequently removed at bench or recipient operation.

At some centers, the donor's common iliac artery bifurcation is also procured to function as a "Y-graft"—the donor external iliac artery is anastomosed end-to-end to the donor superior mesenteric artery, and the donor internal iliac artery end-to-end to the donor splenic artery (Fig. 1). The portal vein is used for venous drainage of pancreas.

Transplant procedure

A midline incision is made for intraperitoneal placement of the procured organs. The donor pancreas and kidney are usually sited within the right and left iliac fossae, respectively (Figs. 2, 3, 4).

There are three anastomoses to consider and they are given below.

1. Arterial supply usually through end-to-side anastomosis between the donor common iliac Y-graft and recipient common iliac (Fig. 2) or external iliac arteries.
2. Venous drainage may be
   - Systemic (performed in approximately 80% of SPK operations [4])—through anastomosis of donor portal vein with recipient inferior vena cava, common, or external iliac veins (Fig. 2),

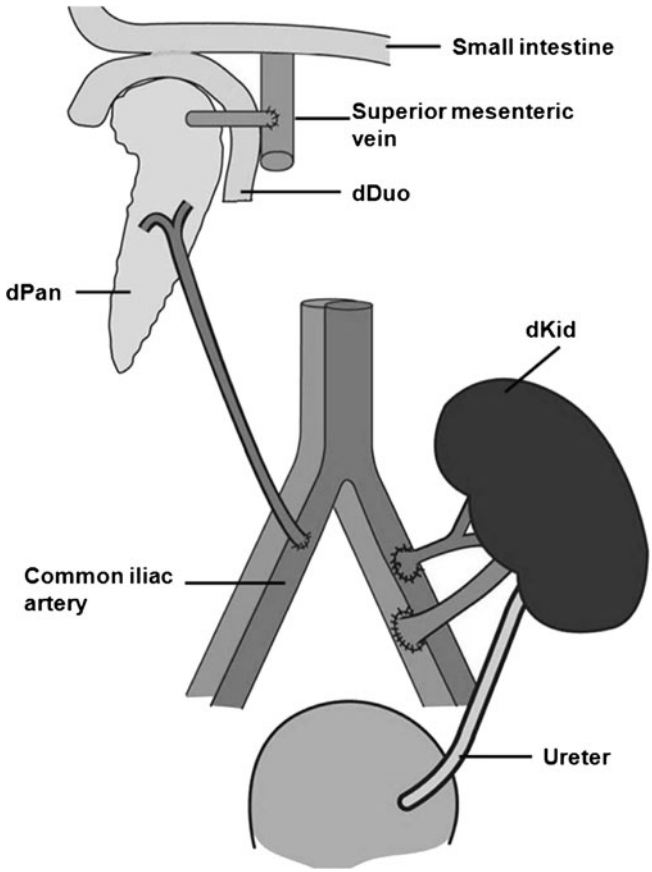

Fig. 3. Enteric-drainage of donor pancreatic exocrine secretions—donor pancreas (dPan), duodenum (dDuo), kidney (dKid). Note venous drainage through anastomosis of donor portal vein to recipient superior mesenteric vein.

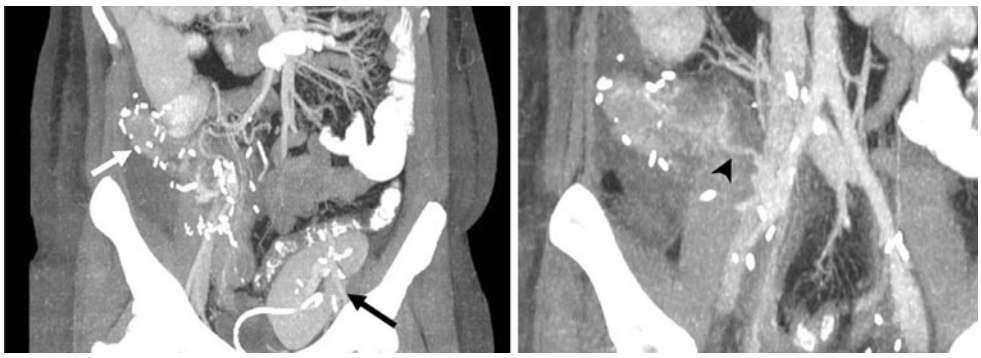

Fig. 4. Coronal oblique maximum intensity projection images in a patient post-simultaneous pancreas-kidney transplantation depict the donor pancreas (*white arrow*) and kidney (*black arrow*) sited within the right and left iliac fossae, respectively. The anastomosis between donor portal vein and recipient common iliac vein can be seen (*black arrowhead*).

- Portal-through anastomosis of donor portal vein with recipient portal or superior mesenteric vein (Fig. 3).

3. Pancreatic duct drainage via donor duodenum C-loop anastomosis to
   - Small bowel (accounting for approximately 80% of SPK operations [4], Fig. 3),
   - Bladder (Fig. 2).

While there are no significant differences in patient, kidney, and pancreas survival between bladder- and enteric-drained patients [6]; exocrine bladder drainage has been associated with higher incidences of urinary tract infections [6, 8], resulting in a considerable conversion rate (of up to 50% [6]) to enteric drainage, necessitating a second operation.

Imaging the post-transplant patient

Ultrasound in conjunction with Doppler imaging has the advantage of being readily available, cheap, portable, and free from ionizing radiation. However, ultrasound is operator dependent and visualization of the intraperitoneal pancreas can be impossible in the presence of overlying gas-filled loops of bowel [9].

Cross-sectional imaging with computed tomography (CT) and magnetic resonance (MR) imaging enables evaluation of the abdomen including the transplanted graft and vasculature. However, with CT, the use of iodinated contrast media is of concern in the setting of impaired renal graft function. Kidney transplant patients account for 27% of patients with nephrogenic systemic fibrosis (NSF) [10]. Therefore, the use of MR-based intravenous contrast agents should also be used with caution in post-SPK transplant patients.

Fluoroscopy, catheter angiography, and nuclear medicine studies may also be employed to aid in the assessment of selected post-transplant patients. Their roles will be discussed later in this review.

Complications

Complications following pancreas transplantation can broadly be divided into four subsets.

- Parenchymal
  - Rejection
  - Pancreatitis
- Exocrine leaks and fistulation
- Vascular
  - Thrombosis
  - Hemorrhage
  - Pseudoaneurysm and arteriovenous fistula
- Peripancreatic
  - Lymphocoele
  - Abscess
  - Hematoma
  - Pseudocyst
  - Urinoma

Parenchymal complications

Graft pancreatitis

In the immediate post-operative period, pancreatitis occurs to some degree in all patients due to hypoxia, organ handling, and re-perfusion injury. This is usually mild and self-limiting, but a small subset of patients may experience severe pancreatitis following surgery with resultant threat to graft survival. Pancreatitis occurring weeks after surgery may be secondary to reflux of exocrine secretions (“reflux pancreatitis”).

Patients with pancreatitis present with tenderness over graft site, pyrexia, and elevated amylase. As in native pancreatitis, extension of the inflammatory process around adjacent vessels can predispose to vascular thrombosis and pseudoaneurysm formation.

In general, sonographic appearances are non-specific and can also be seen in graft rejection. Sonographic findings of heterogenous parenchymal echotexture [11] and donor pancreatic duct dilatation [12] (Fig. 5) have been reported in patients with graft rejection and reflux pancreatitis.

Computed tomography is useful for demonstrating extent and severity of graft pancreatitis (Fig. 6). CT findings are similar to those of native pancreatitis—peripancreatic fat stranding, peripancreatic collections, abnormal

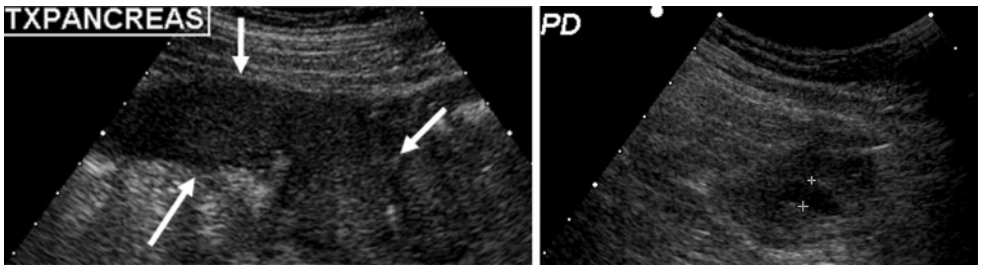

Fig. 5. Longitudinal (**A**) and axial (**B**) image of a patient post-PAK with elevated amylase depict heterogenous parenchymal echotexture (*white arrows*) and a dilated pancreatic duct (PD) measuring 6.5 mm in diameter (**B**, callipers).

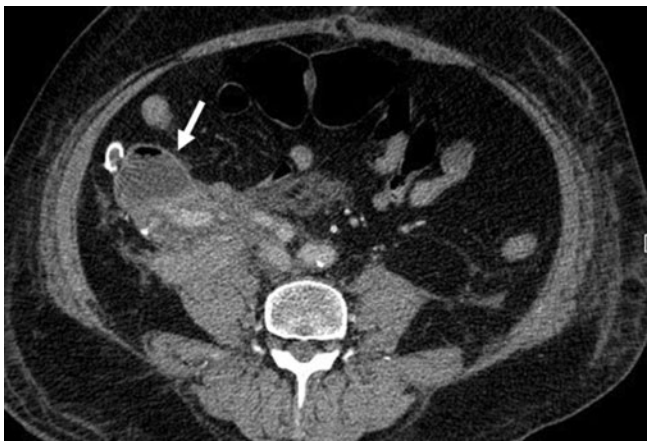

Fig. 6. Axial CT image in a patient with post-transplant pancreatitis showing heterogenous pancreatic enhancement, mesenteric stranding, and a gas- and fluid-containing collection (*white arrow*).

parenchymal enhancement, pseudocyst formation, and free fluid may be evident [13].

Graft rejection

Acute graft rejection has been reported to complicate up to 10.6% of pancreatic transplants [14]. It is thought to be secondary to an alloimmune arteritis causing small vessel occlusion, which can in turn progress to more proximal larger vessel occlusion [15].

Patients with graft rejection may present with low-grade fever, unexplained leukocytosis, and tenderness over graft site. Alterations in C-peptide and insulin levels may not be present in early acute rejection.

On ultrasound, the most common abnormality in patients with acute rejection is pancreatic enlargement with a sensitivity and specificity of 58% and 100%, respectively [16]. CT findings are non-specific and biopsies are usually required to ascertain diagnosis of acute rejection. Dynamic contrast enhanced MR has been shown to be highly sensitive for acute rejection, but less specific due to some overlap of appearances between normal and rejection cases [17].

Chronic rejection can occur following multiple undiagnosed or incompletely treated episodes of acute rejection, culminating in a small atrophic graft. On MR imaging, the atrophic graft has decreased T1- and T2-weighted signal intensity secondary to fibrosis and diminutive feeding vessels, but demonstrates normal enhancement [15].

Exocrine leak

Exocrine leak occurs in up to 10% of transplant patients following enteric drainage [18] and remains a significant cause of graft loss. Pancreatic exocrine secretion may incite strong inflammatory intraperitoneal reactions, with resultant formation of phelgmons or infected collections. Subsequent fistula formation may ensue if left unrecognized or untreated.

In the early post-operative period, early leaks are usually due to ischemia or related to anastomotic techniques. In the later post-operative period, leaks may be secondary to infection, rejection, or ischemia of the duodenal staple line. Patients generally present with abdominal pain and signs of sepsis with elevated white cell count and inflammatory markers. It is important to note that as a result of immunosuppressive therapy, some transplant patients may not display overt signs of infection or leak, and a high index of suspicion is critical to timely diagnosis and treatment.

Treatment usually takes the form of surgical repair and while clinical suspicion may be sufficient to mandate treatment, imaging can often provide confirmatory evidence in equivocal cases. CT with intraluminal contrast (Fig. 7) and fluoroscopy (Fig. 8) are useful imaging techniques in ascertaining the presence of and defining extent of leaks. Intraluminal contrast increases the conspicuity of intraabdominal collections on CT and the presence of extraluminal contrast extravasation confirms the presence of enteric leakage.

Vascular complications

Thrombosis

Graft thrombosis remains the most common nonimmunologic cause of early graft loss, accounting for pancreas transplant failure in 12–13% of patients [19, 20]. Thrombosis can be venous or arterial, with the former being more common.

Multiple risk factors have been implicated in the development of graft thrombosis. These include low blood flow within the pancreas graft, cold ischemia time

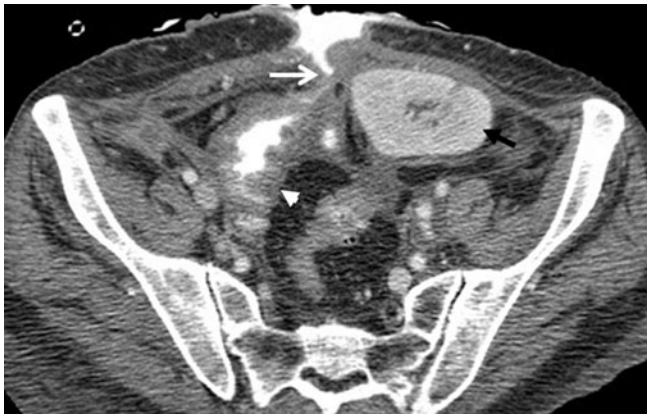

Fig. 7. Axial CT with intravenous and enteral contrast in a patient post-SPK depict thickening of pelvic small bowel loop (*white arrowhead*) and an enterocutaneous fistula (*white arrow*). Note the transplant kidney sited within the left iliac fossa (*black arrow*).

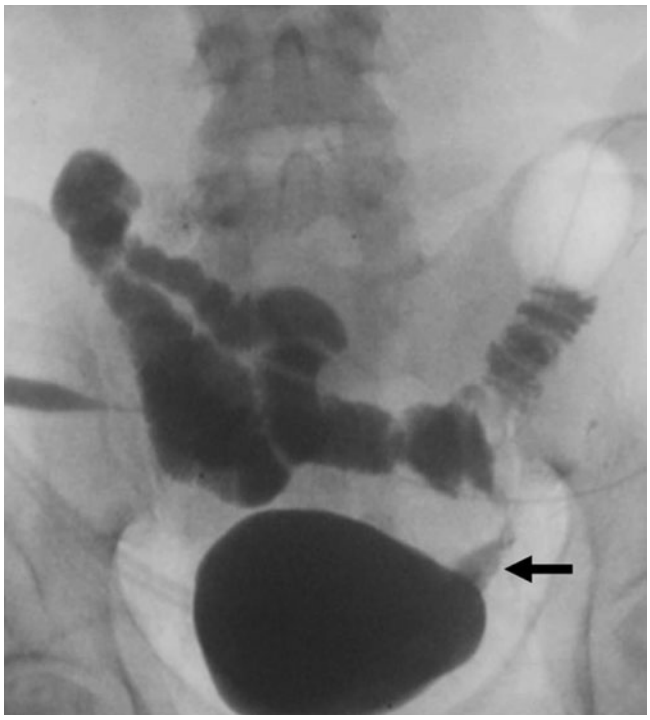

Fig. 8. Fluoroscopic image in a patient following enteric conversion of previous bladder-drained transplant pancreas shows a fistula between the bladder and adjacent small bowel loop (*black arrow*).

in excess of 12 h, advanced donor age, cerebrovascular cause of donor death, left-sided implantation into recipient, graft pancreatitis [19] and use of an interpositioned vascular graft [6, 19].

The majority of graft thromboses occur early after transplant and should be suspected in the setting of graft tenderness, hyperglycemia, elevated serum amylase, or

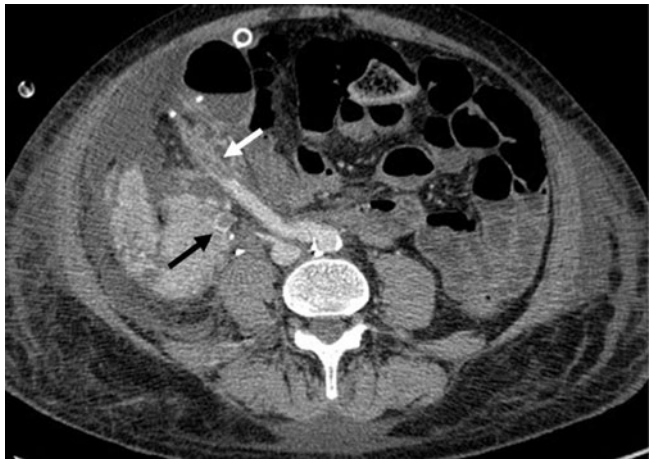

Fig. 9. Axial CT image in a patient following SPK depicting a non-occlusive thrombus within the donor portal vein (*black arrow*) and donor superior splenic artery (*white arrow*). Also note generalized anasarca suggesting low-flow states and possibly predisposing to thromboses.

lipase. In patients with bladder drainage, there may be a decrease in urinary amylase.

On ultrasound, reversal of diastolic flow in pancreatic transplant arteries has been reported to be highly specific for detection of graft venous thrombosis [21]. While there may be an overlap of resistive indices between normal and diseased allografts, resistive indices of 1.0 or greater and the absence of venous flow, in combination, are believed to be highly sensitive and specific for the diagnosis of pancreatic graft venous thrombosis [21].

Computed tomography (Fig. 9) and conventional angiography may depict thrombus as a filling defect but their use is limited by the requirement for intravenous iodinated contrast, which may be contraindicated in the setting of recovering renal insufficiency following SPK transplants.

Small studies of MR angiography have shown good accuracy in the diagnosis of graft thrombosis [22, 23], depicting thrombi as filling voids within the vessels. Nonenhancement or heterogeneous enhancement of graft parenchyma have also corresponded to glandular necrosis at pancreatectomy [23].

Vascular stumps may be present on both the venous and arterial components of the transplant. It is not uncommon to identify stump thrombi within these low-flow stumps. Short-segment stump thrombi occurring within peripheral segments of vessels that do not contribute arterial supply or venous drainage to the pancreatic graft are often incidental findings and are usually not treated [15].

Pseudothrombosis is an important pitfall to consider. This phenomenon describes an apparent venous filling defect due to slower flow of blood through the pancreatic graft, failing to opacify the right external iliac vein [24]. If pseudothrombosis is suspected, a delayed CT through

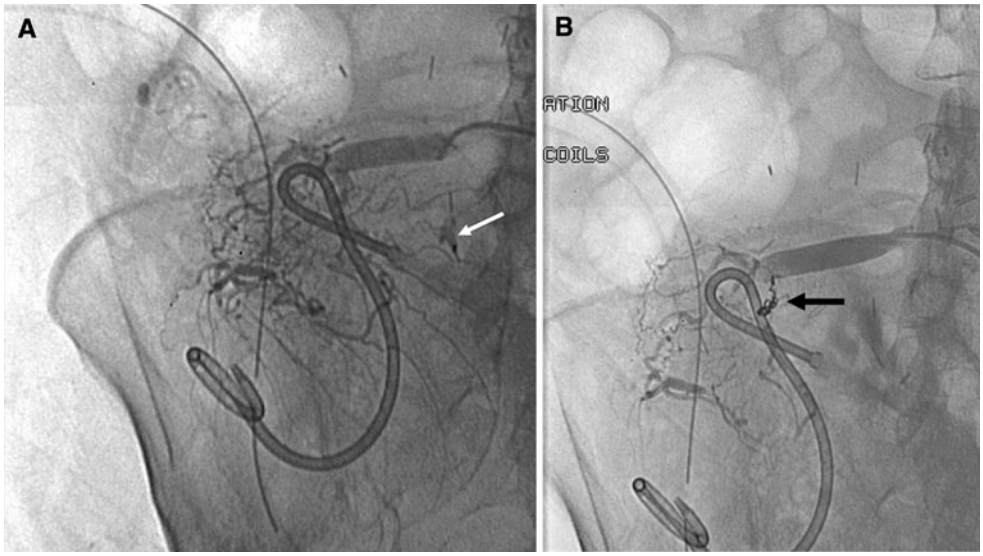

Fig. 10. Catheter angiography reveal hemorrhage from the donor superior mesenteric artery (**A**) which was successfully coiled (**B**, *black arrow*). There is a duodenal drain in situ.

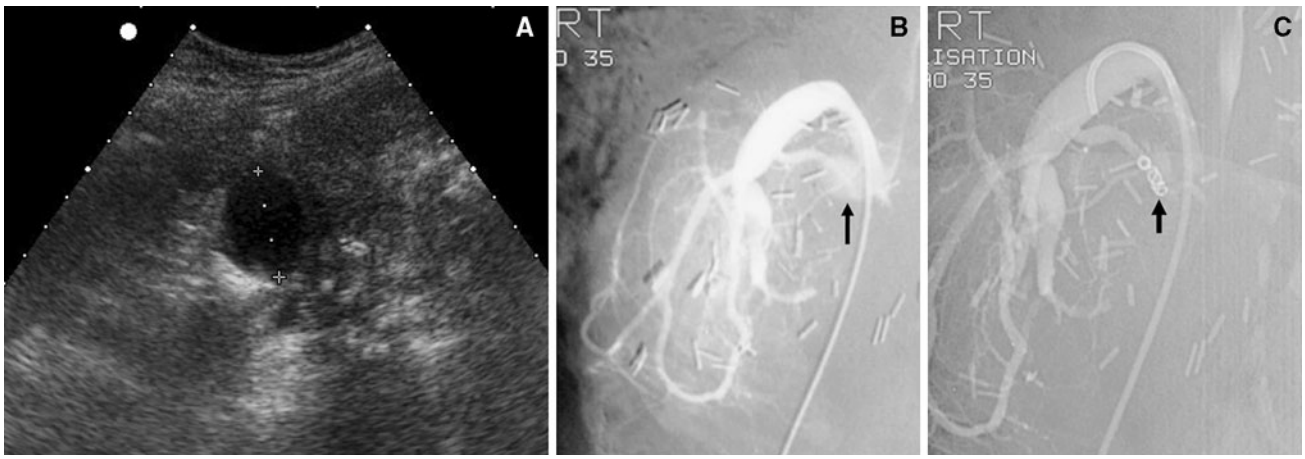

Fig. 11. Ultrasound (**A**) of a patient post-SPK depicting a focal hypoechoic lesion (callipers) which demonstrated flow on Doppler imaging. A pseudoaneurysm was suspected and this was confirmed with catheter angiography (**B**, *black arrow*). The pseudoaneurysm was successfully treated with coils (**C**, *black arrow*).

the region of abnormality or Doppler ultrasound can be performed for confirmation.

Complete arterial or venous thrombosis are usually not salvageable conditions and often necessitate exploration and pancreatectomy. In selected cases of partial thrombosis, percutaneous thrombolysis or thrombectomy have roles in the treatment of patients [20].

Hemorrhage

In the early post-operative period, bleeding is usually due to surgical factors [25] and should be suspected in the context of decreasing hematocrit levels. Ischemia or ulcers (usually CMV induced) of the duodenal segment are also important causes to consider [26]. Hematuria in bladder-drained patients can be assessed using cystoscopy, but gastrointestinal bleeds in enteric-drained patients are more difficult to evaluate. Following standard upper and lower endoscopy, small bowel follow through [26] or cross-sectional imaging may be used to evaluate for small bowel pathology such as ulceration. Tc-99m-labeled red blood cell studies have also been employed to identify site of hemorrhage [27]. While CT angiography may be used to detect the site of bleeding, catheter angiography (Fig. 10) affords the option of embolization, potentially sparing the patient from graft pancreatectomy.

Pseudoaneurysms [28–31] of the donor arteries are relatively rare and may be the result of infection [30] (mycotic aneurysms). When pseudoaneurysms rupture, arteriovenous [32, 33] and arterioenteric [30, 34] fistulae may ensue.

Sonographically (Fig. 11A) pseudoaneurysms appear as well-defined hypo- or anechoic lesions with turbulent

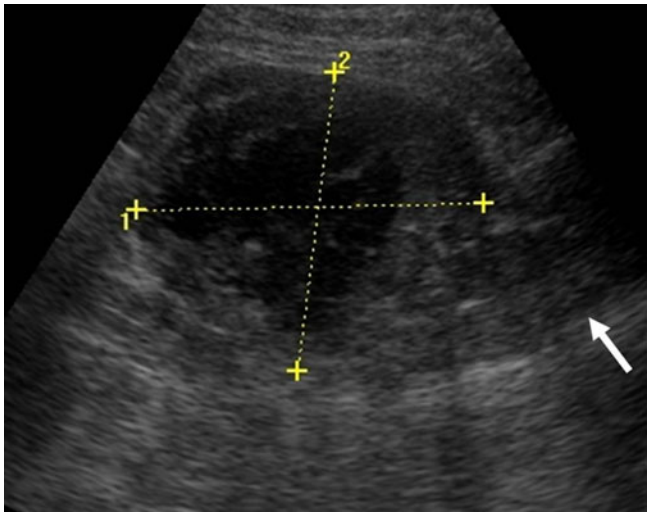

Fig. 12. Sonographic image showing a thick walled, predominantly hypoechoic collection (callipers) seen adjacent to the transplanted pancreas (*white arrow*).

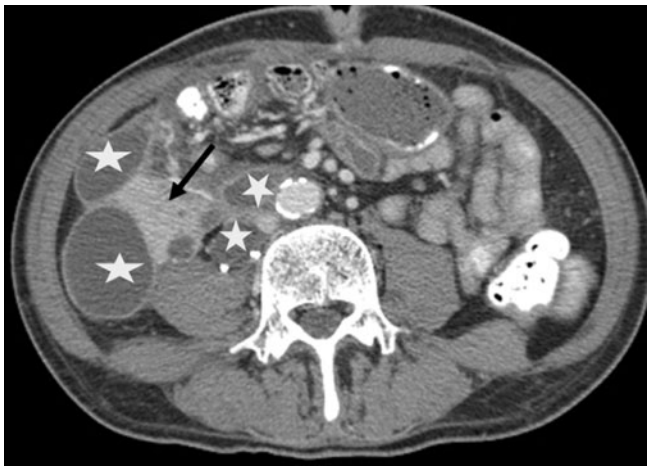

Fig. 13. Axial CT depicting multiple collections (*white stars*) around a normally enhancing transplanted pancreas (*black arrow*).

internal flow on Doppler interrogation. Catheter angiography (Fig. 11B, C) affords both diagnostic and therapeutic capabilities. Coil embolizations of pseudonaneurysms have been performed with good results [31–33].

Peripancreatic collections

Peri-pancreatic-transplant fluid collections are frequently seen post-operatively and are associated with increased morbidity, mortality, and graft loss [35]. Fluid collections include urinoma, seroma, lymphocele, abscess, hematoma, and pseudocyst.

Ultrasound (Fig. 12) is often used for the detection of peripancreatic fluid collections, which appear as focal anechoic or heterogenous lesions. CT (Fig. 13) and MR

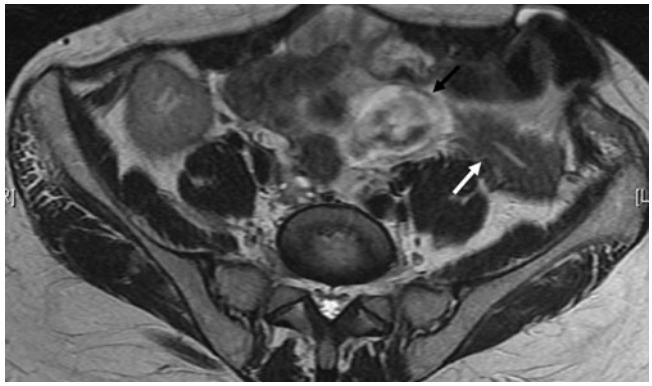

Fig. 14. T2-weighted axial MR image showing a heterogenous, predominantly high intensity collection sited within the left iliac fossa (*black arrow*), adjacent to the transplanted graft (*white arrow*). The transplanted kidney is sited within the right iliac fossa.

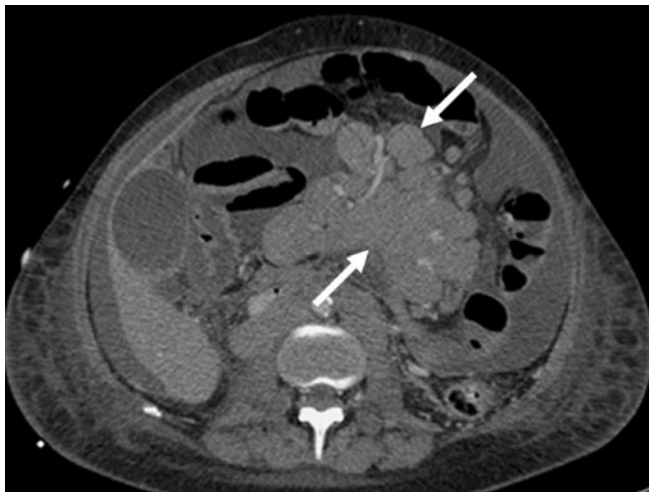

Fig. 15. Axial CT in a patient with PTLD depicting ascites and conglomerate lymph nodal mass (*white arrow*) encasing the mesenteric vessels.

(Fig. 14) may be used to define the extent of the collection. To ascertain the cause of fluid collections, ultrasound and CT-guided aspirations are sometimes performed to determine fluid composition. Percutaneous drainage of infected collections or pseudocysts may help to avoid surgery in some cases [35].

Other considerations

Post-transplant lymphoproliferative disorder (PTLD) is a significant cause of morbidity and has been reported in 2.4–6.1% of patients following pancreatic transplant [36, 37]. CT features include lymph nodal enlargement, extranodal low-density foci within the liver, spleen, kidney, and thickening of the gallbladder or bowel walls [38]. The pancreatic allograft itself can be diffusely enlarged, an

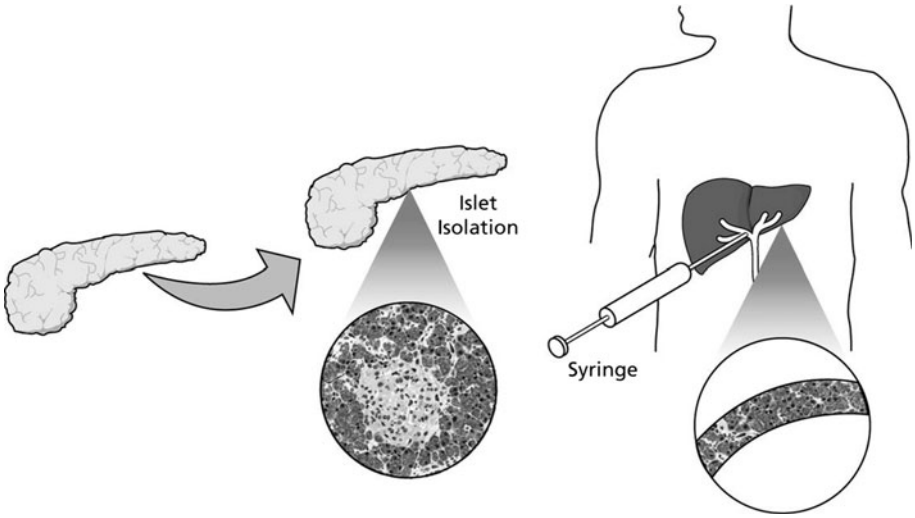

Fig. 16. Islet cell transplant. Islet cells are isolated from the donor pancreas. Following processing, the pancreata is injected into the portal vein under fluoroscopic and/or sonographic guidance.

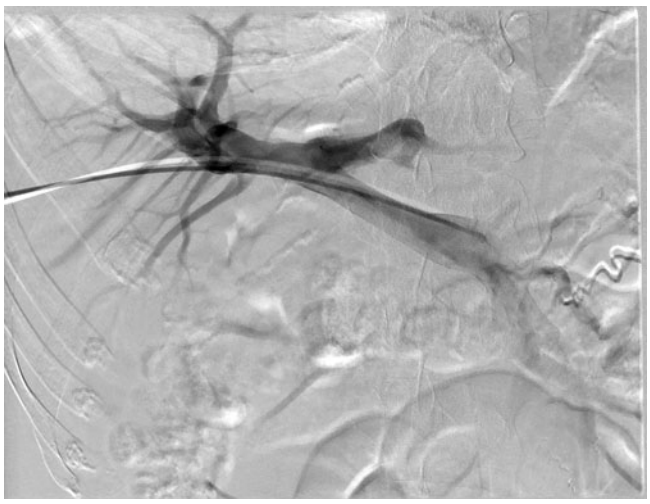

Fig. 17. Fluoroscopic image obtained at time of pancreatic islet cell transplant depicting catheter sites within the main portal vein.

appearance that may be indistinguishable from acute pancreatitis or transplant rejection. However, failure of response to immunosuppressive therapy and the presence of intraallograft or extraallograft focal masses should suggest the diagnosis of PTLD [37] (Fig. 15).

Bowel obstruction following transplant can occur secondary to adhesions or an internal hernia through a defect created at the time or intraperitoneal placement of the pancreatic allograft [39].

Islet cell transplant

Islet cell transplant is an alternative means of restoring endogenous insulin secretion. Overall, 70% of all islet-alone recipients achieved insulin independence [40] compared with 85% of post-SPK patients [3]. Like whole organ transplantation, there are three main types of islet cell transplantation procedures:

– simultaneous islet-kidney transplantation (SIK),
– islet after kidney transplantation (IAK),
– islet transplantation alone (ITA).

Islet cell transplantation requires initial isolation and processing of donor pancreata. Islet cells can either be from donor (*allo-transplantation*) or from patient (*auto transplantation*).

Transplantation technique involves cannulating the portal vein under fluoroscopic and/or ultrasound guidance and injecting the processed pancreata into the portal vein [41] (Figs. 16, 17). Complications that can occur following islet cell transplantation include bleeding, portal vein thrombosis [41, 42], and hypercholesterolemia [42].

Conclusion

Pancreatic transplant remains a viable treatment for diabetes but is associated with a complex spectrum of postsurgical complications. Prompt diagnosis of complications is crucial for graft survival and prevention of graft pancreatectomy. As clinical presentations of post-surgical complications are often non-specific and symptoms may be masked by immunosuppressive therapy, imaging is often employed to aid in the assessment of patients. Clear understanding of surgical anatomy and imaging features of complications are key to the image interpretation of patients.

Acknowledgments. The authors would like to thank Dr Gerry Murphy, Consultant Radiologist at Manchester Royal Infirmary for angiographic images.

References

1. Kelly WD, Lillehei RC, Merkel FK, Idezuki Y, Goetz FC (1967) Allotransplantation of the pancreas and duodenum along with the kidney in diabetic nephropathy. Surgery 61:827–837

2. International Pancreas Transplant Registry. www.med.umn.edu/iptr. Accessed Feb 2010
3. Scientific Registry of Transplant Recipients. www.ustransplant.org. Accessed Feb 2010
4. Gruessner AC, Sutherland DE (2005) Pancreas transplant outcomes for United States (US) and non-US cases as reported to the United Network for Organ Sharing (UNOS) and the International Pancreas Transplant Registry (IPTR) as of June 2004. Clin Transplant 19:433–455
5. White SA, Shaw JA, Sutherland DE (2009) Pancreas transplantation. Lancet 373:1808–1817
6. Sollinger HW, Odorico JS, Becker YT, D'Alessandro AM, Pirsch JD (2009) One thousand simultaneous pancreas-kidney transplants at a single centre with 22-year follow-up. Ann Surg 250:618–630
7. Reddy KS, Stablein D, Taranto S, et al. (2003) Long-term survival following simultaneous kidney-pancreas transplantation versus kidney transplantation alone in patients with type 1 diabetes mellitus and renal failure. Am J Kidney Dis 41:464–470
8. Pirsch JD, Odorico JS, D'Alessandro AM, et al. (1998) Posttransplant infection in enteric versus bladder-drained simultaneous pancreas-kidney transplant recipients. Transplantation 66:1746–1750
9. Nikolaidis P, Amin RS, Hwang CM, et al. (2003) Role of sonography in pancreatic transplant. Radiographics 23:939–949
10. Prince MR, Zhang HL, Roditi GH, Leiner T, Kucharczyk W (2009) Risk factors for NSF: a literature review. J Magn Reson Imaging 30:1298–1308
11. Low RA, Kuni CC, Letourneau JG (1990) Pancreas transplant imaging: an overview. AJR 155:13–21
12. Ciancio G, Montalvo B, Roth D, Miller J, Burke GW (2000) Allograft pancreatic duct dilatation following bladder drained simultaneous pancreas-kidney transplantation: clinical significance. J Pancreas 1:4–12
13. Dachman AH, Newmark GM, Thistlethwaite JR, et al. (1998) Imaging of pancreatic transplantation using portal venous and enteric exocrine drainage. AJR 171:157–163
14. Larson TS, Bohorquez H, Rea DJ, et al. (2004) Pancreas-after-kidney transplantation: an increasingly attractive alternative to simultaneous pancreas-kidney transplantation. Transplantation 27:838–843
15. Dobos N, Roberts DA, Insko EK, et al. (2005) Contrast-enhanced MR angiography for evaluation of vascular complications of the pancreatic transplant. Radiographics 25:687–695
16. Wong JJ, Krebs TL, Klassen DK, et al. (1996) Sonographic evaluation of acute pancreatic transplant rejection: morphology-Doppler analysis versus guided percutaneous biopsy. AJR 166:803–807
17. Krebs TL, Daly B, Wong JJ, Carroll K, Bartlett S (1999) Acute pancreatic transplant rejection: evaluation with dynamic contrast-enhanced MR imaging compared with histopathologic analysis. Radiology 210:437–442
18. Goodman J, Becker YT (2009) Pancreas surgical complications. Curr Opin Organ Transplant 14:85–89
19. Troppmann C, Gruessner AC, Benedetti E, et al. (1996) Vascular graft thrombosis after pancreatic transplantation: univariate and multivariate operative and nonoperative risk factor analysis. J Am Coll Surg 182:316
20. Gilabert R, Fernandez-Cruz L, Real MI, et al. (2002) Treatment and outcome of pancreatic venous graft thrombosis after kidney-pancreas transplantation. Br J Surg 89:355–360
21. Foshager MC, Hedlund LJ, Troppmann C, Benedetti E, Gruessner RW (1997) Venous thrombosis of pancreatic transplants: diagnosis by duplex sonography. AJR 169:1269–1273
22. Hagspiel KD, Nandalur K, Pruett TL, et al. (2007) Evaluation of vascular complications of pancreas transplantation with high-spatial-resolution contrast-enhanced MR angiography. Radiology 242:590–599
23. Eubank WB, Schmiedl UP, Levy AE, Marsh CL (2000) Venous thrombosis and occlusion after pancreas transplantation: evaluation with breath-hold gadolinium-enhanced three-dimensional MR imaging. AJR 175:381–385
24. Gupta R, Rottenberg G, Taylor J (2002) Pseudothrombosis of the iliac vein in patients following combined kidney and pancreas transplantation. BJR 75:692–694
25. Marsh CL, Forge P (1994) The diagnosis and management of urologic complications in non-renal transplant recipients. Semin Urol 12:233–236
26. Barone GW, Webb JW, Hudec WA (1998) The enteric drained pancreas transplant: another potential source of gastrointestinal bleeding. Am J Gastroenterol 93:1369–1371
27. Bingener-Casey J, Esterl RM, Kenneth WW, et al. (2002) Tc-99m-labeled red blood cell scanning localizes anastamotic hemorrhage between the distal ileum and duodenal stump of an enteric-drained pancreas transplant. Clin Nucl Med 27:205–206
28. Green BT, Tuttle-Newhall J, Suhocki P, Smith SR, O'Connor JB (2004) Massive gastrointestinal hemorrhage due to rupture of a donor pancreatic artery pseudoaneurysm in a pancreas transplant patient. Clin Transplant 18:108–111
29. Verni P, Leone JP, DeRoover A (2001) Pseudoaneurysm of the Y-graft/iliac artery anastomosis following pancreas transplantation: a case report and review of the literature. Clin Transplant 15:72–76
30. Tzakis AG, Carroll PB, Gordon RD, et al. (1989) Arterial mycotic aneurysm and rupture. A potentially fatal complication of pancreas transplantation in diabetes mellitus. Arch Surg 124:660–661
31. Dalla Valle R, Capocasale E, Mazzoni MP, et al. (2005) Embolization of a ruptured pseudoaneurysm with massive hemorrhage following pancreas transplantation: a case report. Transplant Proc 37:2275–2277
32. Angle JF, Matsumoto AH, McGraw JK, et al. (1999) Percutaneous embolization of a high-flow pancreatic transplant arteriovenous fistula. Cardiovasc Intervent Radiol 22:147–149
33. Barth MM, Khwaja K, Faintuch S, Rabkin D (2008) Transarterial and transvenous embolotherapy of arteriovenous fistulas in the transplanted pancreas. J Vasc Interv Radiol 198:1231–1235
34. McBeth D, Stern SA (2003) Lower gastrointestinal hemorrhage from an arterioenteric fistula in a pancreatorenal transplant patient. Ann Emerg Med 42:587–591
35. Patel BK, Garvin PJ, Aridge DL, Chenoweth JL, Markivee CR (1991) Fluid collections developing after pancreatic transplantation: radiologic evaluation and intervention. Radiology 181:215–220
36. Issa N, Amer H, Dean PG, et al. (2009) Posttransplant lymphoproliferative disorder following pancreas transplantation. Am J Transplant 9:1894–1902
37. Meador TL, Krebs TL, Wong JJ, et al. (2000) Imaging features of posttransplantation lymphoproliferative disorder in pancreas transplant recipients. AJR 174:121–124
38. Pickhardt PJ, Siegel MJ (1999) Posttransplantation lymphoproliferative disorder of the abdomen: CT evaluation in 15 patients. Radiology 213:73–78
39. Lall CG, Sandrasegaran K, Maglinte DT, Fridell JA (2006) Bowel complications seen on CT after pancreas transplantation with enteric drainage. AJR 187:1288–1295
40. Collaborative Islet Transplant Registry (CITR) 2009 report. www.citregistry.org
41. Venturini M, Angeli E, Maffi P, et al. (2005) Technique, complications and therapeutic efficacy of percutaneous transplantation of human pancreatic islet cells in type 1 diabetes: the role of ultrasound. Radiology 234:617–624
42. Ryan EA, Lakey JR, Paty BW, et al. (2002) Successful islet transplantation: continued insulin reserve provides long-term glycemic control. Diabetes 51:2148–2157