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Early and Late Presentations of Graft Arterial Pseudoaneurysm Following Pancreatic Transplantation

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Abstract
Background Graft pseudoaneurysm (PSA) following pancreatic transplantation (PT) is a rarely reported complication that has significant morbidity and mortality. Few case reports and small series of this complication exist.
Methods Retrospective review of files of 106 patients who underwent PT at the Tel-Aviv Sourasky Medical center between 1995 and 2010. Accessible asymptomatic patients ($n = 35$) were referred for graft PSA screening using ultrasound-Doppler.
Results Eight patients developed graft PSA (8 %). All had early posttransplant sepsis. PSA incidence among patients who had perioperative sepsis is 13 %. Three patients developed early postoperative PSA, presenting as massive abdominal bleeding requiring urgent laparotomy and graft resection. Five patients were diagnosed with late-onset graft PSA between 3 months and 11 years posttransplant: clinical presentations were massive gastrointestinal bleeding ($n = 2$), acute renal failure ($n = 1$), and asymptomatic finding on screening ultrasound-Doppler ($n = 2$, 6 % of screened patients).
Conclusions PSA following PT occurs in 8 % of patients. Perioperative infection is a risk factor. Early PSAs present as massive intra-abdominal bleeding. PSA may develop years posttransplant, may be asymptomatic, but late rupture is possible and presents as gastrointestinal bleeding. We recommend screening of patients at risk with ultrasound Doppler for early detection and treatment of asymptomatic PSAs.

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Introduction

Combined kidney and pancreas transplantation is used increasingly for the treatment of end-stage renal disease (ESRD) resulting from insulin-dependent diabetes mellitus (DM). Graft and patient survival have significantly improved because of changes in patient selection (donor and recipient), and advancement in surgical techniques, postoperative management, and immunosuppressive regimens. Early vascular complications following pancreatic transplantation (PT) are not rare, and represent the most common cause of early graft failure [1–4]. The most common vascular complication is graft thrombosis, either venous or arterial. The formation of a pseudoaneurysm (PSA) following PT is a rarely reported complication that can present as massive abdominal bleeding due to free rupture of the PSA, as GI bleeding due to erosion of the PSA to the GI tract, or as an asymptomatic contained PSA. Most of the reported post-PT PSAs have been diagnosed in the early postoperative period, usually presenting as massive abdominal bleeding [5–7]. Late development of PSAs has only rarely been reported [8–11]. Reported treatment

options for post-PT PSAs include graft resection, open vascular repair, and endovascular repair.

We retrospectively reviewed data of patients who underwent PT at our institution and were diagnosed with graft PSA. We also performed ultrasound (US)-Doppler studies on all accessible patients that had undergone PT at our institution, in search of asymptomatic PSAs of the transplanted pancreas. We report on the prevalence, the clinical presentation, and management of early versus late graft PSA following PT.

Methods

We retrospectively reviewed the medical files of all patients who underwent PT with or without simultaneous kidney transplantation at the Tel-Aviv Medical Center between January 1, 1995 and June 1, 2010. The medical in-patient and outpatient records were reviewed for graft PSA presenting in either the early or late postoperative interval. We evaluated demographics, as well as perioperative, clinical, laboratory, and imaging data that we extracted from their charts. All available patients consented to undergo screening for the presence of pancreatic PSA by means of US-Doppler studies at our institution.

Donor and recipient selection and surgery

Transplant recipients had type 1 DM with severe kidney dysfunction (creatinine clearance <30 ml/min). The pancreas was recovered from young and healthy donors, according to selection criteria suggested by Stratta [12]. Simultaneous kidney and pancreas transplantation (SPK) was intraperitoneal, with the pancreas placement in the right iliac fossa. Arterial inflow was via a Y-graft to the splenic and the superior mesenteric arteries. Venous drainage was systemic to the iliac vessels. Exocrine drainage was enteric via a Roux-en-Y reconstruction for procedures performed before 2002, and via side-to-side hand-sewn duodenoenterostomy thereafter. All patients underwent routine US-Doppler studies on postoperative days (PODs) 1–3. Ultrasonography (US) is not routinely performed after the patient is discharged from the hospital unless there is clinical reason to suspect vascular, infectious, or inflammatory pathology, nor is regular US surveillance routinely performed beyond the perioperative interval. Biopsy of the transplanted pancreas is rarely performed at our institution. The vast majority of pancreas transplantations are performed as SPK, and a kidney biopsy is taken whenever there is clinical suspicion of rejection.

Immunosuppression regimen

Induction of immunosuppression included an anti-T-lymphocyte globulin, tacrolimus or cyclosporine, mycophenolate mofetil, and steroids. Maintenance immunosuppression was based on triple therapy, using tacrolimus or cyclosporine, mycophenolate mofetil, and steroids. Cytomegalovirus (CMV) preemptive treatment was given to all patients.

Clinical course

We assessed the clinical parameters in patients with arterial graft PSAs. These parameters included transplant operative and postoperative course and complications, episodes of rejection, episodes of active CMV infection, findings on postoperative imaging, time from transplant to diagnosis of PSA, clinical presentation of the PSA, management of the PSA, and clinical outcome following treatment. An infectious complication was assumed when clinical signs, laboratory parameters, and/or a positive culture led to the initiation of antibiotic treatment.

Follow-up

All patients who were accessible for follow-up were contacted directly. They underwent US-Doppler studies at our institution by a single US specialist experienced in performing US-Doppler studies on transplanted patients (A.K.). Patients with sonographic suspicion of PSA underwent CT angiography (CTA) for confirmation and treatment planning. Patients with confirmed PSA were offered diagnostic angiography and endovascular treatment.

Results

A total of 106 patients underwent PT at our institution between 1995 and 2010. Overall, eight patients (8 %) were diagnosed with graft arterial PSA. The main clinical characteristics of the eight patients with PSAs are outlined in Table 1. The patients' mean age at the time of transplantation was 38 years, and 38 % were males.

Pattern of occurrence of pseudoaneurysms

Four patients were diagnosed as having early post-PT PSA (up to 3 months posttransplant): two had early PSA formation that was diagnosed 15 and 30 days posttransplant, one (patient #3) had graft thrombosis 11 days posttransplant

Table 1 Patients with pseudoaneurysm (PSA) after pancreatic transplantation

Variable	Patient #1	Patient #2	Patient #3	Patient #4	Patient #5	Patient #6	Patient #7	Patient #8
Postop, infection	Abscess	Fever responded to Ab Tx	Abscess	Abscess	Leak, reoperation	Abscess (Fig. 1)	Fever responded to Ab Tx	Candidemia
Postop. rejection	–	Cellular	–	–	Cellular + humoral	–	–	–
early reoperation	–	–	Graft resection d/t thrombosis, POD 11	–	–	–	–	Bleeding, POD 1
Graft function at PSA pres.	Normal	Normal	Normal renal	Normal	Failure	Normal	Normal	Normal
Interval to pres.	15 days	30 days	30 days	3 months	2 years	4 years	9 years	11 years
Pres. symptom	Bleeding, shock	Bleeding, shock	Bleeding, shock	Acute renal failure	Abdominal pain, Fever followed by massive GI bleeding	Massive GI bleeding	Asymptomatic	Asymptomatic
Rupture	At pres.	At pres.	At pres.	–	4 months after pres.	At pres.	–	–
Therapy	Graft resection	Graft resection EA bypass	Resection of retained vascular anastomosis, EA bypass	Graft resection EA bypass	Graft resection EA bypass	Graft resection	Endovascular	Follow-up: refused endovascular treatment
Outcome	Normal renal function	Normal renal function	Normal renal function	Normal pancreas function	Death	Normal renal function	Normal pancreas and renal function	Normal pancreas and renal function; PSA enlargement

Postop. postoperative, *pres*. presenting/presentation, *EA* extra-anatomic, *GI* gastrointestinal, *Fcn* function, *Ab* antibiotic, *Tx* treatment

followed by massive abdominal bleeding from a ruptured PSA 3 weeks later, and one (patient #4) had acute pain accompanied by acute renal failure 3 months posttransplant. The remaining four patients had late-onset PSA formation, which was diagnosed between 2 and 11 years posttransplant.

Posttransplant complications

Posttransplant infectious complications in patients with pseudoaneurysms

All eight patients had infectious complications during the immediate postoperative period following the transplantation. The clinical presentation of the sepsis included fever without an apparent cause that was treated with empiric antibiotics ($n = 2$), peripancreatic abscess that was drained percutaneously ($n = 4$; Fig. 1), leak from the transplanted duodenum that was treated surgically ($n = 1$), and systemic sepsis with candidemia that responded to prolonged antibiotic treatment with amphotericin B ($n = 1$). After review of files of pancreatic transplant patients, we found record of postoperative infectious complications in 60 cases (56 %). These complications are summarized in Table 2. The incidence of PSA following PT in patients who developed postoperative infectious complication was 13 % (8/60) and zero in patients who did not develop postoperative sepsis ($P = 0.008$). Five of 25 patients (20 %) with peripancreatic abscess or leaks developed symptomatic PSAs.

Clinical presentation of the PSA

Early presentation of PSA (up to 3 months posttransplant; $n = 4$)

General presentation

Two of the four patients with early graft PSA presented with massive intra-abdominal hemorrhage and profound shock that required immediate laparotomy for hemostasis (Fig. 1). One patient (patient #3) experienced acute pancreatic graft thrombosis on POD 11 and underwent

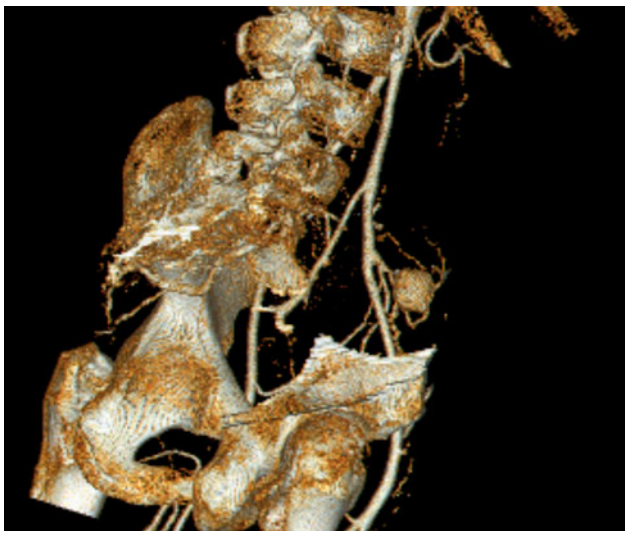

Fig. 1 Computerized tomographic angiogram of patient #2 who had early postoperative ruptured pseudoaneurysm

Table 2 Summary of posttransplant septic complications

Complication	Percentage of patients
No postoperative septic complication	43
Intra-abdominal collection	22
Wound infection	9
Fever	9
Urinary tract infection	9
Urinary leak	2
Enteric leak	3
Bacteremia (unknown origin)	1
Candidemia (unknown origin)	2

pancreatic graft resection after which she developed signs of systemic infection accompanied by abdominal collections and purulent discharge from the abdominal drain. On POD 30, she had sudden onset of hemodynamic shock accompanied with new bloody output through the abdominal drain. The fourth patient (patient #4) presented with acute onset lower abdominal pain and renal failure that required immediate laparotomy and renal transplant resection.

Posttransplant complications in patients with PSAs

Three patients developed fever and peripancreatic collections in the early postoperative interval that required CT-guided drainage and antibiotic treatment. The fourth patient developed fever without a clear source and had sterile blood and urine cultures. He responded to empiric antibiotic treatment.

Other complications in patients with PSAs

One patient (patient #3) experienced acute pancreatic graft thrombosis on POD 11 and underwent pancreatic graft resection. Another patient (patient #2) was diagnosed with acute cellular rejection (a kidney biopsy was performed); treatment consisting of high-dose steroids yielded a good response.

Management and outcome of PSAs

All four patients underwent immediate laparotomy during which a ruptured PSA of the arterial graft was diagnosed, and they all subsequently underwent graft resection. Three patients underwent resections that included a graft-iliac anastomosis and required construction of extra-anatomical femorofemoral bypass. The fourth patient underwent resection of the graft with preservation of the graft-iliac anastomosis. All four patients had an unremarkable postoperative course and retained normal function of their remaining grafts.

Late presentation of PSA (>3 months posttransplant; $n = 5$)

General presentation

Four patients had late-onset PSA that occurred between 3 months to 11 years posttransplant (Fig. 2). Their clinical presentations included acute massive gastrointestinal (GI) bleeding ($n = 1$), abdominal pain and fever with signs of subacute graft inflammation, followed 4 months later by

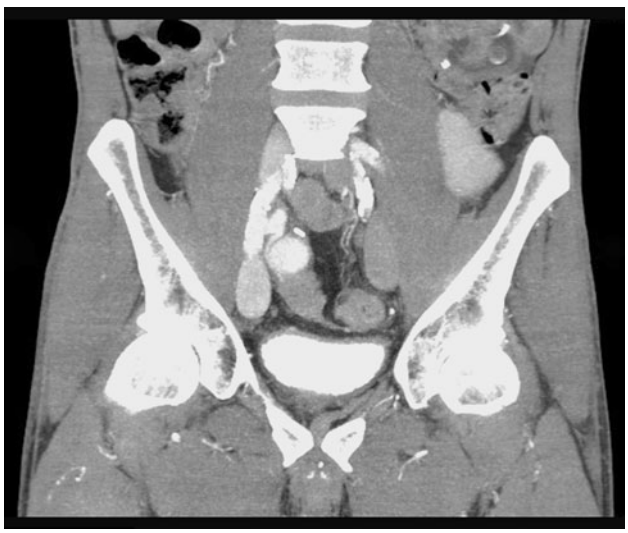

Fig. 2 Computerized tomographic angiogram of patient #7 who had an asymptomatic late pseudoaneurysm

acute massive GI bleeding ($n = 1$), and asymptomatic PSA diagnosed on screening duplex studies ($n = 2$).

Posttransplant complications in patients with PSAs

One patient (patient #5), who developed a postoperative enteric leak with signs of sepsis and enteric fluid in the drains, underwent a relaparotomy and primary suture repair of the graft-enteric anastomosis. Another patient (patient #6) developed fever and peripancreatic collections in the early postoperative interval that required CT-guided drainage and antibiotic treatment (Fig. 3). Patient #6 developed fever postoperatively without a clear source, with sterile blood and urine cultures. He responded to empiric antibiotic treatment. The fourth patient (patient #8) developed prolonged postoperative fever with candida growth in blood cultures and systemic signs of candidiasis. That patient responded to a prolonged course of Amphotericin B.

Other complications in patients with PSAs

One of the patients (patient #8) experienced massive bleeding on POD 1 that originated from the arterial renal artery-iliac anastomosis. He underwent relaparotomy and primary repair with good outcome. Another patient (patient #5) developed combined cellular and acute humoral rejection (confirmed on kidney graft biopsy). That patient was treated with high-dose steroids, plasmapheresis, and intravenous immunoglobulin with partial response and eventually underwent the loss of graft functions as a result of rejection at 1 year posttransplant.

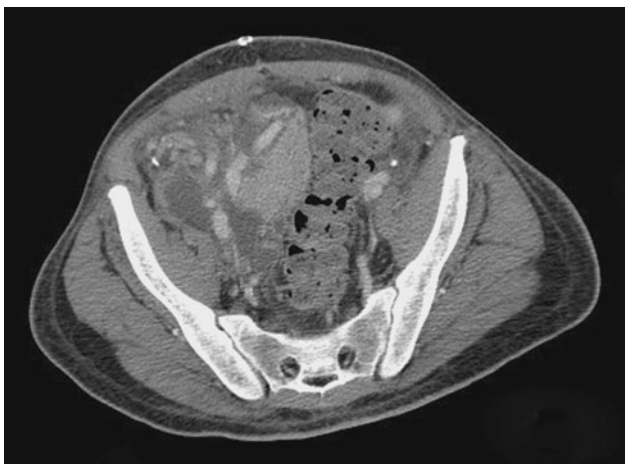

Fig. 3 Computerized tomographic scan of patient #6: a peripancreatic abscess is visible several days after simultaneous kidney and pancreas transplantation. This patient developed acute rupture of a pseudoaneurysm 4 years later

Management and outcomes of PSAs

Two patients underwent immediate laparotomy during which ruptured PSA of the arterial graft was diagnosed, requiring graft resection. One patient underwent resections that included a graft-iliac anastomosis and required construction of extra-anatomical femorofemoral bypass, and the other patient underwent resection of the graft with preservation of the graft-iliac anastomosis. The postoperative course of one patient (patient #5) was complicated by sepsis and multiorgan failure followed by death. The other patient had an unremarkable postoperative course and retained normal function of the remaining graft.

Two patients were diagnosed on duplex studies as having asymptomatic PSA. They both were offered endovascular treatment. One of them (patient #8) refused further treatment and is being followed regularly by duplex studies, which have shown gradual enlargement of the PSA. That patient is asymptomatic and has normal graft function. The second one (patient #7) underwent angiography, which demonstrated a large PSA distal to the Y-graft bifurcation, followed by successful endovascular occlusion of the PSA and demonstration of flow in the proximal graft artery. That patient has retained normal graft function, and lack of flow in the PSA was demonstrated on several follow-up duplex studies.

US-Doppler screening of asymptomatic transplant patients

Thirty-five patients underwent screening US-Doppler studies at the Tel-Aviv Medical Center by a single ultrasound specialist experienced in performing US-Doppler studies on transplanted patients (A.K.). Four patients were diagnosed with suspected PSA of the arterial pancreatic graft. They underwent CTA, which ruled out PSA in two of them and demonstrated a PSA in the other two (6 % of all screened patients). The subsequent management of these patients is described above.

Discussion

Simultaneous pancreatic and kidney (SPK) transplantation is currently the preferred therapeutic approach for patients with type 1 DM and ESRD. This treatment has proved to be efficacious in normalizing blood sugar levels, prolonging survival, and improving quality of life [13]. PT has a higher rate of surgical complications compared with other solid organ transplantations [14]. Despite the significant advancements made in patient and donor selection, surgical technique, and immunosuppressive treatment, early complications still

account for a relatively high rate of reoperations and are the leading cause of early graft loss [15]. Whereas postoperative hemorrhage and vascular thrombosis are the most frequently described vascular complications following PT, there are only a few reports on the development of PSA following PT [5–11].

A number of causes may be responsible for the formation of PSAs following PT. These include congenital anomalies, back-table injuries, technical failure involving the vascular anastomoses, and iatrogenic injury during pancreas biopsy. However, septic complications, and accompanying leakage of pancreatic fluid, appear to be the most significant predisposing factor for PSA formation. There are a few reports of PSA formation in patients undergoing nontransplant related pancreatic resections. In these cases, PSA usually develops following postoperative septic complications and pancreatic fistula formation [16–18]. Despite the improvement in the results of SPK transplant in recent years, infectious complications continue to be a significant cause of morbidity and mortality. Posttransplant infectious complication rates of up to 90 % have been reported, with the highest incidence during the first 90 days after the surgery [19–24]. In our series, the early perioperative infectious complication rate was 56 %. The rate of intra-abdominal collection was 22%, and 3 % of the patients had anastomotic leaks that required relaparotomy. Previous series on outcome of pancreas transplantation report intra-abdominal infections that required percutaneous drainage or laparotomy in 16–27 % of the patients [19–24]. There are a number of reports of post-PT PSAs from the 1980s and 1990s that occurred after an infectious complication [10, 25]. The relative paucity of such reports from recent years may be related to the reduced incidence of posttransplant infectious complications in recent years. The eight patients in our report were operated by three different experienced transplant surgeons; therefore, a recurrent technical error is less likely. All eight patients in our report had a perioperative infectious complication, which probably contributed to the development of PSA. The occurrence of perioperative infectious complications seems to be the most significant risk factor for the future development of either early or late graft PSA. In our report, PT patients who had postoperative infectious complications had an alarming 13 % chance of developing graft PSA compared with zero in patients without postoperative infectious complications. The incidence of symptomatic PSAs among patients who had postoperative peripancreatic abscess or anastomotic leak was an even higher 20 % (5/25 patients). We, therefore, believe that surveillance of PT patients with postoperative sepsis using US-Doppler may be indicated.

The timing of PSA presentation relative to the transplantation has important clinical and prognostic implications. In our series, the four patients who presented in the early postoperative interval (up to 3 months posttransplant) had acute and sudden free rupture of the PSA to the abdominal cavity. These patients presented with signs of severe systemic shock which necessitated emergent operations and pancreatic graft removal. In contrast, only one patient with late-onset PSA (between 2 and 11 years after transplantation) presented initially with massive lower GI bleeding. Interestingly, whereas early PSAs ruptured to the peritoneal cavity, late PSA ruptures were intraluminal, and presented as massive GI bleeding. Graft PSA rupture should be included in the differential diagnosis of PT patients with GI bleeding, even many years posttransplant. Because some of these patients are initially treated in nontransplant institutions, it should be emphasized that in PT patients with massive GI bleeding who undergo emergent angiography visualization of the iliac vessels should always be performed in addition to the visceral vessels. For the more stable PT patient with GI bleeding, CT angiography should be performed in the initial workup to rule out the possibility of graft PSA.

Management of posttransplant PSA is complex and largely depends on the clinical scenario and the timing of presentation relative to the transplantation. PSAs that present as free rupture should be treated with immediate laparotomy and graft resection. There are no clear-cut answers in the literature to the question of whether the iliac artery Y-graft anastomosis should be resected (with an extra-anatomic bypass for limb perfusion). We performed this procedure in four of our patients. The other two patients who presented with free rupture underwent graft artery resection distal to the iliac Y-graft anastomosis, and both had an unremarkable postoperative course. However, we recommend that resection of the iliac-graft anastomosis be performed and that an extra-anatomic bypass be completed for limb perfusion when an infectious etiology is suspected.

The optimal treatment of asymptomatic late-onset graft PSA is controversial. The natural history of these lesions and risk of rupture are unknown, and active intervention may jeopardize graft function and limb vasculature. However, two patients in our series with late-onset PSA that eventually ruptured and another patient with documented enlargement of the PSA on follow-up US-Doppler studies (the patient that refused endovascular treatment) serve as examples that some of these lesions may grow and eventually rupture if left untreated. Nevertheless, regular follow-up with interval US-Doppler studies may be a valid option for consideration in asymptomatic patients with small PSAs, with progressive enlargement of the PSA as being an indication for intervention.

Treatment options for patients with a nonruptured PSA of the pancreatic graft include endovascular and surgical procedures. Although there are several reports of

successful surgical treatment of pancreatic PSAs with preservation of graft function, we believe the preferred initial approach in most patients should be endovascular, thereby optimizing the chances for graft salvage [6, 9].

There are several recent reports on endovascular treatment of PSAs following PT. Tan et al. [5] described successful deployment of a covered stent over a leaking PSA in the iliac artery. Paduch et al. [7] reported achieving a good result after treating a peripheral arterial PSA by angio-embolization. Green et al. [8] reported selective angio-embolization of a ruptured PSA in a patient who presented with massive GI bleeding, in which several episodes of rebleeding necessitated graft pancreatectomy. Fujita et al. [11] reported initial coil embolization of an asymptomatic 1.9-cm PSA resulting in a leak and massive bleeding 3 weeks later, which was successfully treated by endovascular covered stent deployment. One patient in our series was treated by endovascular techniques. Patient #7, who had an asymptomatic massive PSA, was successfully treated by deployment of a vascular occluder in the feeding vessel, with preservation of the proximal pancreatic artery. We believe that endovascular therapy is a valid therapeutic option for stable patients with PSAs following PT.

Conclusions

The results of this series demonstrated that post-PT graft PSA occurs in at least 8 % of patients. All of the studied patients had a history of a perioperative infectious complication. The incidence of PSA in PT patients that had postoperative septic complication is 13 %. Early postoperative PSAs presents as massive intraperitoneal bleeding, for which the preferred treatment is emergent laparotomy with graft and vascular resection and possible extra-anatomic vascular bypass. Patients may develop PSAs years after PT, and although a long asymptomatic course may be possible, continued enlargement and eventual intraluminal rupture can occur, presenting as massive GI bleeding. For early detection and preventive endovascular treatment of asymptomatic lesions, we recommend periodic screening with US-Doppler of PT patients, especially for those who had postoperative septic complications.

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