ABSTRACT

Graft thrombosis is the most common cause of early graft loss after pancreas transplantation. The grafted pancreas is difficult to salvage after complete thrombosis, especially arterial thrombosis, and graft pancreatectomy is required. We describe a patient presenting with a functioning pancreas graft with thromboses of the splenic artery (SA) and superior mesenteric artery (SMA) after simultaneous pancreas-kidney transplantation (SPK). A 37-year-old woman with a 20-year history of type 1 diabetes mellitus underwent SPK. The pancreaticoduodenal graft was implanted in the right iliac fossa with enteric drainage. A Carrel patch was anastomosed to the recipient’s right common iliac artery, and the graft gastroduodenal artery was anastomosed to the common hepatic artery using an arterial I-graft. The donor portal vein was anastomosed to the recipient’s inferior vena cava. Four days after surgery, graft thromboses were detected by Doppler ultrasound without increases in the serum amylase and blood glucose levels. Contrast enhanced computed tomography revealed thromboses in the SA, splenic vein and SMA. Selective angiography showed that blood flow was interrupted in the SA and SMA. However, pancreatic graft perfusion was maintained by the I-graft in the head of the pancreas and the transverse pancreatic artery in the body and tail of the pancreas. We performed percutaneous direct thrombolysis and adjuvant thrombolytic therapy. However, we had to stop the thrombolytic therapy because of gastrointestinal hemorrhage. Thereafter, the postoperative course was uneventful and the pancreas graft was functioning with a fasting blood glucose level of 75 mg/dL, HbA1c of 5.1%, and serum C-peptide level of 1.9 ng/mL at 30 months post-transplantation.
increases in the serum amylase and blood glucose levels. Contrast enhanced computed tomography (CE-CT) revealed thromboses in the SA, SV, and SMA (Fig 1). After written informed consent was obtained from the patient, we attempted interventional therapy. Arterial access was achieved via the right femoral artery. Selective angiography via the Carrel patch revealed that blood flow was interrupted in the SA and SMA to the graft due to the graft thromboses. Graft thrombosis was also observed in the SV. However, pancreatic graft perfusion was maintained by the I-graft in the head of the pancreas and the transverse pancreatic artery from the dorsal pancreatic artery in the body and tail of the pancreas (Fig 2). We immediately performed percutaneous direct thrombolysis with the administration of urokinase via the SA and SMA (60,000 IU in each artery) to prevent progression of graft thromboses. This procedure resulted in significant decrease of the thromboses. However, splenic venous congestion was still present. Therefore, we administered adjuvant thrombolytic therapy by continuously infusing urokinase (240,000 IU per day) via the graft’s SA along with systemic heparinization. However, we stopped the thrombolytic and the anticoagulant therapies the next day because of gastrointestinal hemorrhage. Thereafter, the postoperative course was uneventful and the pancreas graft was functioning well. The patient was discharged 52 days after the SPK. CE-CT at discharge revealed that the pancreas graft was well enhanced whereas graft thromboses existed in the distal SA and SMA.

The graft pancreas is functioning well. The patient had a fasting blood glucose level of 75 mg/dL, HbA1c of 5.1%, and serum C-peptide level of 1.9 ng/mL at 30 months post-transplantation.

Discussion

To our knowledge, this is the first report to describe a functioning graft pancreas with thrombotic occlusion of the SA and SMA after SPK. In the present case, pancreas graft perfusion was maintained via the I-graft in the head of the pancreas and the transverse pancreatic artery in the body and tail of the pancreas with SA and SMA thromboses. Because the SA and SMA are two major arteries of the pancreatic graft, when thromboses of these two arteries occur, graft pancreatectomy is usually required. Margreiter et al [3] showed that entire perfusion of the pancreatic graft and sufficient graft function are sustained after thrombotic occlusion of one branch of the Y-graft (SA or SMA) by a complex system of intra-parenchymal anastomoses. On the other hand, Akhtar et al [4] reported a patient with thrombotic occlusion of the SMA who required revascularization of the GDA on re-laparotomy.

Simultaneous procurement of the pancreas and liver necessitates division of the vessels supplying both organs. The most common technique for arterial reconstruction in pancreas transplantations involves a donor Y-graft of the common iliac artery with its external and internal iliac branches [5–7]. GDA arterial reconstruction using an interposed donor iliac artery between the stumps of the CHA and GDA is common with a good outcome in our country because of the high rate of marginal donors [8]. However, the efficacy of GDA reconstruction still remains unclear.

Graft thrombosis occurs secondary to donor hypoperfusion, poor preservation, technical issues, immunologic issues, sepsis, coagulopathy, or other issues [9]. Pancreas graft thrombosis has been attributed to the change in the hemodynamics of blood flow from high to low flow after ligation of the distal SA, SV, SMA, and superior mesenteric vein. This would create a change in the hemodynamic state of blood flow in these large vessels from a high-flow into a low-flow state [9,10]. Considering the higher occurrence rate of graft thrombosis after pancreas transplantation compared with other organ transplantations, this hemodynamic change may greatly contribute to the development of graft thrombosis in patients who undergo pancreas transplantation.

In the present case, graft perfusion in the head of the pancreas was maintained via the I-graft. It is still unknown whether arterial perfusion in the pancreas head was salvaged due to the GDA reconstruction using I-graft after SMA thrombosis. Because thromboses in this case developed in the peripheral vessels and not in proximal vessels (at the site of vascular anastomosis), a low-flow state in the SMA and SA might have existed. Thus, it is speculated that rather than as a consequence of the SMA and SA thromboses, the graft arterial flow might have been more dominant through the I-graft and GDA than through the SMA in the head of the pancreas and more dominant through the transverse pancreatic artery than through the SA in the body and tail of the pancreas. As a result, SMA and SA thrombotic occlusions might have occurred.

Various procedures for salvaging compromised graft veins and arteries, such as surgical thrombectomy and early pancreas transplantation, have been reported [9,11,12]. Recently, several authors

![Fig 1. Coronal view of the contrast-enhanced abdominal CT 4 days after transplantation.](image)
have reported the use of alternative interventional therapies, such as pharmacomechanical thrombolysis [13], transarterial thrombolysis [14] and catheter-directed thrombolysis combined with balloon thrombectomy [15,16]. In the present case, we were concerned about progression of SA and SMA thromboses as well as SV thrombosis. We performed percutaneous direct thrombolysis and adjunct thrombolytic therapy even though the definitive indication of interventional therapies has not been established. Because artery thrombolysis requires large amounts of urokinase, it should be noted that the risk of iatrogenic hemorrhage is increased. As the patient was asymptomatic with normal glycemic control and had normal serum amylase and lipase levels, and arterial graft perfusion was maintained, another option might have been to monitor the patient closely with or without systemic anticoagulation therapy [3,17].

In conclusion, it may not be necessary to perform immediate intensive intervention for asymptomatic graft arterial thromboses in patients with sufficient perfusion of the entire pancreas. Evaluation of the long-term graft function will be needed.

REFERENCES