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LETTER TO THE EDITOR

A rare case of functional pancreas graft with newly developed collateral venous drainage after complete portal thrombosis

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Portal venous thrombosis has been reported as one of the major surgical complications after pancreas transplantation [1,2]. It is usually detected too late to rescue the graft, and graft pancreatectomy remains the most common option after graft thrombosis [3]. Recently, we experienced a rare case in which a pancreas graft remained functional through spontaneous development of collateral venous drainage within a short period after complete portal thrombosis.

The patient was a 38-year-old male subject with end-stage renal disease caused by type 1 diabetes. Eight months after living kidney transplantation, he received a pancreas transplant from a 53-year-old male subject who had been diagnosed as brain-dead because of subarachnoid hemorrhage with at least 25 min of cardiopulmonary arrest. The pancreas was preserved in University of Wisconsin solution, and the total ischemic time was 9 h 18 min. Pancreatic arterial reconstruction was performed with a donor iliac artery bifurcation Y-graft anastomosed to the splenic and superior mesenteric artery. The graft gastroduodenal artery was anastomosed to the donor iliac Y-graft in an end-to-side manner using an interposition graft as performed routinely in our center. Arterial revascularization of the allograft was carried out by anastomosing the Y-graft to the iliac artery of the recipient. Venous anastomosis was performed between the portal vein of the graft and the iliac vein of the recipient without portal venous extension. The exocrine secretion was drained to the proximal jejunum. The patient's initial course was uneventful, with immediate pancreatic function. The patient had been prophylactically anticoagulated with low-dose heparin (5000–8000 IU/day) since postoperative day (POD) 2. Partial thrombus in the graft splenic vein was detected by routine ultrasonography (US) and enhanced computed tomography (eCT) (Fig. 1a and b)

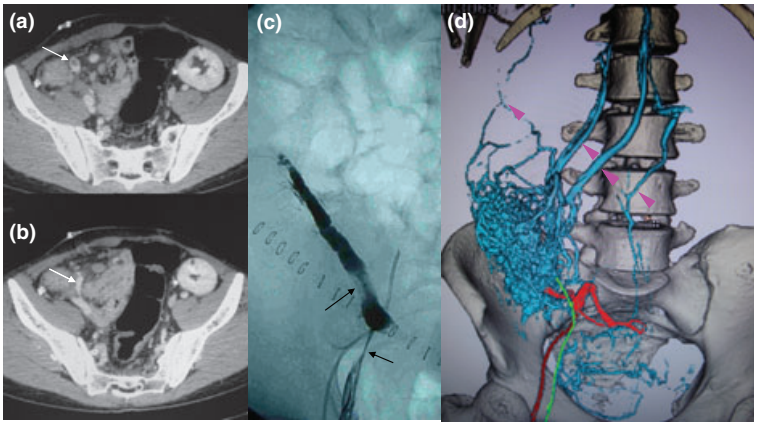

Figure 1 (a) Findings of enhanced computed tomography (eCT) on postoperative day (POD) 6. Partial thrombus in graft splenic vein (arrow) was detected. (b) Findings of enhanced computed tomography (eCT) on postoperative day (POD) 6. Patency of portal trunks (arrow) was preserved without thrombus formation. (c) Angiography for pancreatic graft on POD 14. Contrast medium was infused via the catheter (arrow) placed in splenic vein of the pancreatic graft after successful mechanical thrombectomy. (d) Findings of 3-D enhanced computed tomography (eCT) on POD 15. Contrast medium was infused via the arterial catheter placed in the Y-graft of the pancreatic graft. Red line showed the arterial catheter placed in the pancreatic graft through the right femoral artery. Red line also indicates the arterial Y-graft, graft splenic artery and superior mesenteric artery. Yellow-green line shows venous catheter placed in the graft splenic vein through the right femoral vein. Blue line indicates the venous flow of the graft including the mesenteric vein (pink arrow) of the recipient.

on POD 6, followed by percutaneous intra-arterial infusion of urokinase (120 000 units) through the catheter, which was inserted from the right femoral artery and advanced into Y-graft on POD 7. The patient was free of extrinsic insulin, with stable secretion of intrinsic insulin from the graft. Nevertheless, on POD 11, US and eCT showed complete portal vein thrombosis with a preserved arterial supply. Immediately, continuous transarterial infusion of urokinase (240 000 units/day) was started for thrombolysis by the same approach described above and continued for 9 days. Slight elevation of serum amylase and lipase level suggested post-transplant pancreatitis. On POD 14, because of persistent complete portal vein thrombus, venous thrombolysis and mechanical venous thrombectomy were added with placement of an inferior vena cava filter [4]. Even after successful mechanical venous thrombectomy (Fig. 1c), arterial flow did not return to the graft portal vein but to the recipient portal system via newly developed collateral vessels (Fig. 1d). On POD 19, continuous transarterial infusion of urokinase was discontinued because the situation had not changed. Systemic i.v. heparin was continued until POD 21, followed by anticoagulation with oral aspirin and warfarin. He was discharged from hospital on POD 55 without extrinsic insulin. A glucose tolerance test showed sufficient release of intrinsic insulin from the graft.

Because the reported risk factor for thrombosis includes older donor age, cardiovascular cause of donor death, longer ischemic time and graft pancreatitis [5], the present case was highly susceptible to the graft thrombosis after pancreas transplantation. When the thrombus was detected, it was not complete but partial as seen in cross-sectional eCT as a floating mass in the center of venous lumen with preserved circumferential flow. Therefore, we chose arterial thrombolysis first but not surgical procedure [6]. We then opted for mechanical venous thrombectomy and direct infusion of urokinase into the graft portal vein. Although this procedure successfully removed the portal thrombus, blood flow in the pancreas parenchyma was returned not to the graft portal vein, but unexpectedly to the recipient portal system via newly developed collateral vessels.

In cases of chronic vein occlusion, collateral vessels sometimes develop as well as neovascularization [7]. However, in the setting of organ transplantation, there has been no report of collateral vessels developing between a transplanted organ and the vasculature of the recipient within a short period.

There are two possible mechanisms by which collateral vessels could have developed between the transplanted pancreas and the recipient's portal system in such a short period: one is arteriovenous fistula formation and the other is venovenous fistula formation. Although there is another possibility that venous flow returned to the recipient's portal system via the grafted pancreas head and duodenum where the venous system was not occluded, this seems unlikely because angiographic findings in the early phase suggested direct graft blood flow into the recipient's mesenteric vein from the grafted pancreas tail. And it is more likely that post-transplant pancreatitis caused a venovenous fistula in the tail of the graft. Although details of the mechanism responsible for establishment of collaterals between the pancreas graft and portal system of the recipient are unclear, our experience in this case suggests that follow-up of vascular patency by duplex Doppler ultrasonography, prophylactic anticoagulation and interventional radiologic techniques are essential strategies for management of thrombosis after pancreas transplantation.

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