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Commentary

The Impact o Pancreas Transplantation on Cardiovascular Mortality

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DESCRIPTION

The uremic state is rife with multiple physiologic derangements, all of which contribute to the wide range of vascular disease manifestations seen in end-stage renal disease. Hyperphosphatemia, hyperparathyroidism, hyperhomocysteinemia, increased insulin resistance, and other factors interact to form a unique pathophysiology and epidemiology of vascular disease in renal disease. Despite improvements in the uremic milieu in patients receiving a functional kidney transplant, cardiovascular disease continues to be a factor. Cardiovascular disease kills more grafts than immunologic loss, neoplasm, or infection combined.

In the well-controlled insulin-dependent diabetic, a similar paradox exists. Although exogenous insulin typically results in an anti-atherogenic lipid profile [Low Density Lipoprotein (LDL), elevated High Density Lipoprotein (HDL), and rapid triglyceride clearance], insulin-dependent diabetics have higher rates of coronary artery disease than the general population. A chronic hyperinsulinemic state results from the replacement of feedback-regulated pancreatic beta cells with an intensive regimen of insulin injections. Excess insulin, which is already known to be atherogenic due to its ability to act as a vascular smooth muscle growth factor, causes a negative change in lipoprotein surface composition.

Pancreas transplantation was conceived of and eventually developed with the hope that restoring euglycemia would halt or even reverse the clinical sequelae of long-standing type diabetes. After pancreas transplantation, there is pathologic evidence of early diabetic nephrology reversal in native kidneys. Diabetes nephropathy recurrence in a functioning kidney-pancreas transplant is uncommon. Unfortunately, the addition of a pancreas to a kidney transplant remains debatable.

An increase in risk factors and a worsening of the cardiovascular risk profile following pancreas transplantation. Similar to the well-controlled insulin-dependent diabetic, kidney-pancreas transplants with systemic (endocrine) venous drainage result in euglycemia at the expense of hyperinsulinemia. Insulin resistance caused by the immunosuppressive agents, as well as hyperinsulinemia, contribute to insulin levels spiralling out of control. Obesity, hypertension, and arteriosclerosis have all been linked to hyperinsulinemia. Pioneered the portal-enteric pancreas transplant, in which exocrine secretions were deposited enterically rather than through systemic venous drainage of endocrine hormones. Portal drainage allows for euglycemia without hyperinsulinemia due to the first-pass effect. Previous complications of kidney-pancreas transplants, such as metabolic acidosis and dehydration, were resolved with the perfection of this technique, demonstrating that the portal-enteric method of pancreas transplantation can be performed with comparable graft and patient survival rates to the systemic-bladder technique.

Although it has been demonstrated that strict glycemic control can slow down secondary complications of diabetes like neuropathy and retinopathy, the impact of euglycemia on cardiac function is still debatable.

At 24 months after transplantation, the normalisation of diastolic and systolic cardiac function was examined in kidney-pancreas transplants as compared to kidney transplants alone. Unfortunately, it was not possible to draw a connection between these findings and a decline in cardiovascular mortality. The data in this issue of Kidney International are the first to show that kidney/pancreas transplants significantly reduce cardiovascular mortality when compared to kidney alone or patients who are still on the waiting list, despite the discussion of the potential relationship between hyperinsulinemia and coronary artery disease (syndrome X) that was mentioned earlier.

With obvious confounders controlled, this study, with its relatively large sample size, are the first to show that the cardiovascular mortality of a pancreas transplant patient (even by the systemic-bladder method) is reduced despite the theoretical concerns and known complications of the procedure. The importance of these conclusions cannot be understated and should serve as the nidus for a large prospective randomized trial comparing systemic versus portal drainage versus kidney alone and resulting rates of cardiovascular mortality.

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