

Adipokines and glucose abnormalities post-renal transplantation

Maria P. Martinez Cantarin

Thomas Jefferson University Hospital, USA

Adipose tissue produces pro-inflammatory and anti-inflammatory adipocytokines and their metabolism is altered in kidney disease. Adiponectin, the main adipose tissue transcript, has antidiabetic properties, and patients with obesity, diabetes, and insulin resistance have low plasma adiponectin levels. However, although kidney disease is associated with insulin resistance, adiponectin is elevated in end-stage renal disease. The cause of this adiponectin elevation in renal disease is unclear although the decrease in renal clearance of the molecule has been postulated as levels of adiponectin inversely correlate with glomerular filtration rate. On the other hand, we have shown high plasma levels of inflammatory adipocytokines including adiponectin in kidney disease patients with increased adiponectin mRNA and protein expression in adipose tissue. This suggests impaired renal clearance may not be the only mechanism by which adiponectin levels are elevated in kidney disease. The driver of increased adiponectin production in kidney disease is unclear and also it is not known if adiponectin is functionally active in patients with kidney disease. Our data also suggests that adiponectin resistance in kidney disease could be the cause of the increased hormone production. Transplant-Associated Hyperglycemia (TAH) develops at relatively high rates following renal transplantation and is associated with great morbidity and mortality. After a kidney transplant plasma adipokine levels decrease but they are still higher than the plasma levels seen in patients without kidney disease. Lower pre-transplant adiponectin levels have been associated with the development of new onset diabetes after transplantation. Further mechanistic studies describing the functionality of adiponectin in kidney disease and after renal transplantation may help to unravel the pathogenesis of transplant associated hyperglycemia.

Biography

Maria P. Martinez Cantarin obtained her medical degree from the Complutense University in Madrid, Spain. She completed her clinical training at Thomas Jefferson University in Philadelphia including Internal Medicine Residency, Nephrology, Transplant Nephrology and Clinical Pharmacology Fellowships. She also obtained a MS degree in Pharmacology from Thomas Jefferson University. She is an Assistant Professor of Medicine in the Division of Nephrology and is the Director of Diabetes Research in ESRD and Transplant at Thomas Jefferson University Hospital.

maria.martinezcantarin@jefferson.edu