Coronary artery intimal thickening in the transplanted heart: An In Vivo Intracoronary Ultrasound Study of Immunologic and Metabolic Risk Factors

Transplantation: January 15th, 1996 - Volume 61 - Issue 1 - p 46-53

Authors

Rickenbacher, Peter R.; Kemna, Mariska S.; Pinto, Fausto J.; Hunt, Sharon A.; Alderman, Edwin L.; Schroeder, John S.; Stinson, Edward B.; Popp, Richard L.Chen, Ida; Reaven, Gerald; Valantine, Hannah A.

Abstract

This study examined the hypothesis that immunologic factors are the major correlates of coronary artery intimal thickening and luminal stenosis. The study population included 116 adult heart transplant recipients with a mean age of 44.7 \pm 12.0 years (89 men and 27 women) undergoing annual coronary angiography and intracoronary ultrasound 3.4 \pm 2.7 (range, 1.0-14.6) years after transplantation. Mean intimal thickness was obtained from several distinct sites along the left anterior descending and/or left circumflex coronary artery by intracoronary ultrasound. Coronary artery stenosis defined by angiography was classified as mild (<30% luminal stenosis), moderate (\geq 30-70% luminal stenosis), or severe (>70% luminal stenosis or diffuse pruning of distal vessels). Prevalence of any transplant coronary artery disease (TxCAD) was 85% by intracoronary ultrasound and 15% by angiography. By multiple regression analysis, only average fasting plasma triglyceride level (P<0.006) and average weight(P<0.007) were significantly correlated with severity of intimal thickening (R=0.54, P<0.0001). Donor age(P<0.006) and average fasting plasma triglyceride level (P<0.009) were significantly correlated with stenosis by angiography.

Correlation of multiple immunologic and metabolic factors with intimal thickness by univariate analysis suggests a multifactorial etiology for TxCAD. Among the multiple univariate correlates of TxCAD, higher fasting plasma triglyceride levels and body weight are the only independent correlates of TxCAD. The absence of acute rejection as an independent predictor of intimal thickening suggests that mechanisms beyond those mediating typical cellular rejection should be targeted for advancing our understanding of TxCAD.