Title	Association between serum concentrations of soluble lectin-like oxidized low density lipoprotein receptor-1 and serum amyloid A in type 2 diabetes
Author(s)	Lee, CH; Shiu, SWM; Wong, Y; Tan, K
Citation	The 10th International Diabetes Federation Western Pacific Region Congress and the 6th AASD Scientific Meeting, Singapore, 21-24 November 2014. In Diabetes Research and Clinical Practice, 2014, v. 106 suppl. 1, p. S56
Issued Date	2014
URL	http://hdl.handle.net/10722/211521
Rights	NOTICE: this is the author's version of a work that was accepted for publication in Diabetes Research and Clinical Practice. Changes resulting from the publishing process, such as peer review, editing, corrections, structural formatting, and other quality control mechanisms may not be reflected in this document. Changes may have been made to this work since it was submitted for publication. A definitive version was subsequently published in Diabetes Research and Clinical Practice, vol. 106, suppl. 1, November 2014. DOI: 10.1016/S0168-8227(14)70315-8

ASSOCIATION BETWEEN SERUM CONCENTRATIONS OF SOLUBLE LECTIN-LIKE OXIDIZED LOW DENSITY LIPOPROTEIN RECEPTOR-1 AND SERUM AMYLOID A IN TYPE 2 DIABETES

C.H. Lee, S. Shiu, Y. Wong, K. Tan

Medicine, University of Hong Kong, Hong Kong, Hong Kong

BACKGROUND: Lectin-like oxidized low density lipoprotein receptor-1 (LOX-1) mediates the uptake of oxidized LDL in endothelial cells and macrophages, and has been implicated in the pathogenesis of vascular inflammation and atherosclerosis. The extracellular domain of LOX-1 can be proteolytically cleaved and released as a soluble form (sLOX-1). It has been suggested that circulating level of sLOX-1 reflects LOX-1 expression and can act as a biomarker. LOX-1 expression is inducible and recent experimental data have shown that serum amyloid A (SAA), an acute phase protein, can induce LOX-1 expression in macrophages in vitro. Since type 2 diabetes is associated with subclinical inflammation, we have evaluated whether serum sLOX-1 level is increased in patients with type 2 diabetes and its relationship to SAA. METHOD: 300 patients with type 2 diabetes and 150 agematched non-diabetic controls were recruited. Subjects with a history of any recent acute illness were excluded. Serum sLOX-1 and SAA were measured by ELISA. RESULT: Type 2 diabetic patients had higher serum sLOX-1 than controls [median 114.7 ng/ml (interquartile range 89.0–132.2) vs 102.6 (83.8–120.3) respectively, $p < 0.01$] and the differences remained significant after adjusting for gender, body mass index and smoking. SAA was significantly increased in diabetic subjects than controls [129.7 ng/ml (85.8–179.3) vs 119.3 (81.6–147.9) respectively, $p < 0.05$] even in the absence of any acute illness. Serum log(sLOX-1) correlated significantly with log(SAA) ($r = 0.51$, $p < 0.001$) and weakly with log(eGFR) ($r = -0.11$, $p = 0.05$) in diabetic patients but not in non-diabetic controls. On linear regression analysis, log(SAA) remained an independent determinant of sLOX-1 even after adjusting for age, gender, body mass index, HbA1c, eGFR and smoking in subjects with diabetes. CONCLUSION: Elevated sLOX-1 concentration in type 2 diabetes suggests that LOX-1 is upregulated and we have found a significant relationship between circulating sLOX-1 and SAA levels. Our results suggest that SAA may potentially play a role in inducing LOX-1 expression in diabetes and contribute to the development of atherosclerosis.