ASSOCIATION OF LIPOPROTEIN ASSOCIATED PHOSPHOLIPASE A2 WITH DIABETIC RETHINOPATHY

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Objectives:

Diabetic rethinopathy is an important microvascular complication in diabetic patients. The association between hyperlipidemia and diabetic rethinopathy remains elusive. Lipoprotein associated phospholipase A2 (Lp-PLA2) is Ca independent serin kinase that hydrolyses oxidized LDL and forms lysilphoshotidylcholine and free fatty acid. The aim of the study was to determine serum levels of Lp-PLA2, traditional lipids, apolipoproteins in patients with diabetic rethinopathy and to evaluate correlation of these parameters with disease severity.

Methods:

A total of 67 diabetic patients were divided into 3 groups (nonproliferative diabetic rethinopathy, proliferative rethinopathy, no rethinopathy) based on fundoscopic examination and matched to 15 healthy and nondiabetic people. Blood samples for lipids, apolipoproteins, Lp-PLA2 were drawn.

Table1: Comparision of biochemical variabilities between groups

	PDR	NPDR	Diabetic controls	Healthy controls	p
Total cholesterol, mg/dl (mean±SD)	212.5±47.1	213.6±60	201.2±38.1	224.6±33	0.67[a] 0.77[b]
LDL mg/dl (mean±SD)	136±34.1	122.7±43.22	113.4±32.4	134.6±31.5	0.24[a] 0.18[b]
HDL mg/dl (mean±SD)	42.5±6.4	46.8±11.6	47±12.3	61.1±14.5	**<0.001[a]** 0.22[b] **0.001[c]** **<0.001[e]**
TG mg/dl (median) (min-max)	185 (52-351)	180.5 (73-923)	190.5 (92-506)	120 (76-305)	0.14[a], 0.89[b]
ApoB mg/l (mean±SD)	1116.6±251.2	1084.5±280.9	1033.2±256.7	982.6±238	0.40
ApoA1 mg/l (mean ±SD)	1402.2±213.7	1459±268.4	1442.9±284.6	1460.7±274.1	0.83
Lipo (a) mg/l (median) (min-maks)	93 (63-685)	154.5 (93-1060)	126 (93-1140)	133 (93-438)	0.07[a], **0.03[b]** **<0.05[f]**
Lp-PLA2 ng/ml (mean±SD)	234.9±68.9	223.7±82	250.5±80.1	195.7±66.7	0.25[a], 0.66[c] 0.36[e] 0.95[f]

TG: Trigliserid, Apo B: Apolipoprotein B, Apo A1: Apolipoprotein A1, Lipo (a): Lipoprotein (a), Lp-PLA2: Lipoprotein associated phospholipase A2, PDR: Proliferative diabetic rethinopathy, NPDR: Nonproliferative diabetic rethinopathy

[a]Comparision of four groups, [b]Comparision of NPDR, PDR, diabetic controls [c]Comparision of NPDR and healthy controls, [e]Comparision of PDR and healthy controls, [f]Comparision of NPDR and PDR

Results:

No significant difference was observed in terms of total cholesterol, TG, LDL-C levels between groups. However HDL-C levels were similar in proliferative diabetic rethinopathy, nonproliferative rethinopathy patients and diabetic controls (42.5±6.4, 46.8±12.3) and were lower than healthy controls (61.1±14.5, $p<0.005$). No statistically significant difference was shown with respect to apolipoprotein A1, apolipoprotein B in all comparisions. Serum levels of Lp-PLA2 was similar in all groups (Table1).

Conclusions:

Serum levels of lipids, apolipoproteins and Lp-PLA2 were not associated with diabetic rethinopathy. Although Lp-PLA2 were found to be associated with macrovascular complications of diabetes in most studies, the role of this molecule in microvascular complications wasn't known. The larger sample sized studies may clarify the relationship between Lp-PLA2 and rethinopathy.

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391--EP
Diabetes (to include obesity, pathophysiology & epidemiology)
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DOI: 10.3252/pso.eu.17ece.2015
Poster presented at: ECE2015 16–20 May 2015 DUBLIN, IRELAND 17th EUROPEAN CONGRESS OF ENDOCRINOLOGY