Perubahan ekspresi lipoprotein associated phospholipase A2 dan lysophophatidylcholine dalam perkembangan awal aterosklerosis: studi in vivo pada tikus model diabetes melitus tipe 2 dan dislipidemia = Expression changes of lipoprotein associated phospholipase A2 and lysophosphatidylcholine in early development of atherosclerosis in vivo studies of the rodent model of type 2

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Abstrak

ABSTRAK
Aterosklerosis merupakan inflamasi akibat OxLDL yang berhubungan dengan lipoprotein associated phospholipase A2 LpPLA2 , sehingga menarik mengetahui lebih jauh peran LpPLA2 dalam patogenesis awal aterosklerosis, Penelitian eksperimental ini menggunakan metode post-test with control group secara invivo 50 ekor tikus Sprague Dawley SD yang dikelompokkan dalam kelompok normal, dislipidemia DL , DM tipe 2 DMT2 dan DL serta DMT2 yang diberikan Darapladib DP dengan dosis 30 mg/kg berat badan/hari. Terdiri dari 2 serial waktu perlakuan yaitu 8 dan 16 minggu. Uji statistik menunjukkan kondisi DL, ekspresi protein Lp-PLA2 di jaringan aorta signifikan dengan peningkatan jumlah sel busa. Pada kondisi DMT2, ekspresi relatif mRNA Lp-PLA2 darah signifikan dengan peningkatan jumlah sel busa. Pemberian DP menekan ekspresi Lp-PLA2 dan LisoPC di jaringan aorta tetapi DP tidak menekan ekspresi mRNA Lp-PLA2 jaringan aorta dan darah baik pada kondisi DMT2 maupun DL. DP mampu menekan inflamasi baik di jaringan aorta maupun plasma kondisi DL maupun DMT2. Ekspresi protein Lp-PLA2 jaringan aorta sesuai dengan perubahan kadar LisoPC jaringan aorta. Namun, profil ekspresi mRNA Lp-PLA2 tidak sesuai dengan profil perubahan kadar LisoPC. Protein Lp-PLA2 tidak dapat menggambarkan ekspresi Lp-PLA2 di aorta. Terdapat perbedaan jalur patomekanisme Lp-PLA2 dalam mengaktivasi respons inflamasi diantara kondisi DMT2 dan DL. Keywords : Aterosklerosis, Dislipidemia, DM Tipe 2, LP-PLA2, LisoPC
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ABSTRACT
Athrosclerosis in an inflamation caused by OxLDL which has corellation with lipoprotein associated phospholipase A2 LpPLA2 . Then, it is interesting to do a deeper exploration about LpPLA2 rsquo s role in atherosclerosis patogenesis. These experimental reseacrh use an invivo post test with control group in 50 Sprague Dawley rats SD that will be grouped in a normal, dyslipidemia DL , type 2 diabetes DMT2 or DL and DMT2 group with Darapladib DP administration 30 mg kg body weight daily, each group consisted of 2 serials treatment time, which are 8 weeks and 16 weeks treatment groups. Statistics result showed that in DL condition Lp PLA2 protein expression in aortic tissue correlate significantly with the increase of foam cells, while in DMT2 condition mRNA Lp PLA2 blood expression correlate significantly with the increase of foam cells. DP decreases Lp PLA2 protein expression and LysoPC in aortic tissue, but DP failed to decrease blood and aorta tissue mRNA Lp PLA2 expression both in DL and DMT2 condition. DP is able to decrease inflammation marker both in aortic tissue and plasma both in DMT2 and DL condition. The pattern of Lp PLA2 protein expression in aorta is similar to LysoPC level. However, mRNA Lp PLA2 expression pattern

is different from lisoPC level pattern. mRNA Lp PLA2 and Lp PLA2 protein expressions in aorta is different from the blood. Therefore, Lp PLA2 expression in blood does not represent the expression of Lp PLA2 in the aorta. There is a different pattern of Lp PLA2 pathomechanism in activating inflammation response between DMT2 and DL conditions. Keywords Atherosclerosis, Dyslipidemia, type 2 DM, LP PLA2, LisoPC