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Elevated Lipoprotein(A) Impairs Platelet Radiolabeling Yield

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Abstract

Objectives: Platelet radiolabeling in clinical routine usually results in labeling efficiencies (LE) above 80%. A variety of risk factors and clinical conditions are known to impair platelet labeling yield, among them elevated triglycerides and low-density lipoproteins. The potential influence of lipoprotein(a) (Lp(a)), an atherogenic lipoprotein particle containing a kringle subunit, which is widely found in the proteins of fibrinolysis pathway, has never been studied. Normal Lp(a) levels range below 30 mg/dl. The exact prevalence of elevated Lp(a) is unknown, most likely ranging below 10%. Even more rare is an isolated elevation despite an otherwise normal lipoprotein profile.

Methods: We examined the role of isolated elevated Lp(a) (> 50 mg/dl, ranging up to 440 mg/dl) compared to patients with normal lipid profile. Platelets were radiolabeled with in-111-oxine at 37 °C for 5 minutes using ISORBE-consensus methodology.

Results: The findings indicate that already at levels below 100 mg/dl Lp(a) decreases LE. LE assessment after cross-incubation of hyper-Lp(a) platelets with normal Lp(a) plasma and vice versa reveals that platelets rather than the plasmatic environment are responsible for the deterioration of labeling yield. This behavior already has been reported for elevated low-density lipoproteins. Apparently, the quantitative influence of LDL and Lp(a)/mg is comparable. Plotting the sum of LDL and Lp(a) versus LE reveals a clear significant negative correlation.

Conclusion: As extremely elevated Lp(a), particularly above 150 mg/dl, may significantly impair labeling results. We therefore recommend to include extremely elevated Lp(a) into the list of parameters, which should be known before performing radiolabeling of human platelets.

Key words: Platelet labeling, lipoprotein(a), labeling efficiency

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Investigation of 99mTc Labeled Clioquinol Derivative on Amyloid Plaque Specifity by Using Animal Model of Alzheimer's Disease

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Abstract

Objective: Alzheimer's disease (AD), which comes up an important problem due to the aging population, has two hallmarks defined as amyloid plaques and neurofibrillary tangles. Despite the fact that AD affects a wide audience adversely, definitive diagnosis of AD still requires postmortem examination of the brain through histological staining of these hallmarks. Nowadays, these two hallmarks are playing crucial role in researches for diagnosis and treatment of AD. In addition, deficiency of AD diagnosis agents and limitation of assessing the blood-brain barrier (BBB) are main reasons of the current researches focusing on brain agents with plaque or tangle imaging potential. Metal-protein attenuating compounds (MPACs) such as clioquinol and cloxyquin have a great increasing promise on amyloid plaques. Yurt Kilcar et al. radiolabeled Bioquin-Hexa methyl propylene amine oxime (HMPAO) with 99mTc (99mTc-Bioquin-HMPAO) and evaluated the biodistribution on healthy male Balb/C mice (unpublished data). In current study, it is aimed to investigate amyloid plaque specifity of the 99mTc-Bioquin-HMPAO by using biodistribution studies on animal model of AD.

Methods: Bioquin-HMPAO was labeled with 99mTc according to previous study. In accordance with the objectives of the study, animal model of AD created on healthy male Sprague Dawley rats with intrahippocampal stereotaxic injection of amyloid beta 1-42 (Aβ1-42). Intrahippocampal administration was applied to the CA1 area of hippocampus bilaterally (Aβ1-42 to the left side and PBS to the right side as control). Additionally, healthy male Sprague Dawley rats were utilized as naïve group. Twenty days after surgery, biodistribution studies of 99mTc-Bioquin-HMPAO on animal model of AD and control group