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LETTER TO THE EDITOR

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Should dual antiplatelet treatment be guided by lipoprotein(a) concentration?

Jacek Kubica

Department of Cardiology and Internal Medicine, Collegium Medicum, Nicolaus Copernicus University, Bydgoszcz, Poland

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Lipoprotein(a) (Lp(a)) is a low-density lipoprotein-like molecule, composed of the apolipoprotein (a) (apo(a)), which is attached to the apolipoprotein B-100 by a single disulfide bond [1]. It is recognized as an independent risk factor for cardiovascular events. Lp(a) favors initiation of atherogenesis by modulating recruitment of inflammatory cells in the vessel wall, increases atherosclerotic plaque vulnerability by provoking local inflammation, and adversely affects discrete key points in primary and secondary hemostasis as well as in fibrinolysis [1]. Due to the high degree of homology between apo(a) and plasminogen, Lp(a) potentiates thrombosis through inhibiting plasminogen activation and fibrin degradation, and promoting endothelial plasminogen activator inhibitor expression, tissue factor pathway inhibitor activity. The role of Lp(a) in platelet activation and aggregation is a matter of debate, as the results of in vitro experimental studies are inconsistent and in-depth clinical studies are lacking.

Recently Cui et al. [2] reported secondary analysis of a single-center, prospective registry demonstrating that extended (> 1 year) dual antiplatelet therapy (DAPT) was associated with lower risk of ischemic cardiovascular events in patients with acute coronary syndrome (ACS) treated with percutaneous coronary intervention (PCI) with elevated Lp(a) levels, but not in individuals with normal Lp(a) level. The extended DAPT was not associated with increased risk of clinically relevant bleeding and did not differ between the two groups with different Lp(a) levels. This study is the first one evaluating the effect of Lp(a) concentrations on the clinical outcomes of extended DAPT in ACS patients after PCI [2]. The present study has several limitations, including the specific Asian ethnicity of the patients, but it provides a strong rationale for more complex assessment of lipid parameters including Lp(a) in all currently ongoing and planned clinical trials aimed at assessing DAPT modification, especially de-escalation and prolonged treatment [3–7]. To understand the mechanism of influence of these factors on platelet reactivity and on the efficacy of antiplatelet drugs, multilevel pharmacodynamic and pharmacokinetic studies are necessary [8–10].

Conflict of interest: None declared

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Address for correspondence: Prof. Jacek Kubica, Department of Cardiology and Internal Medicine, Collegium Medicum, Nicolaus Copernicus University, ul. M. Skłodowskiej-Curie 9, 85–094 Bydgoszcz, Poland, tel: +48 52 585 40 23, e-mail: jkubica@cm.umk.pl

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