LETTER TO THE EDITOR

Lipoprotein(a) in familial hypercholesterolemia: Tips from family history

Dear Editor,

We read with interest the paper by Brandão et al. entitled "Lipoprotein(a) as a key target in combined therapeutic approaches for cardiovascular disease" highlighting, in the era of new lipid-lowering drugs, the value of Lipoprotein(a) [Lp(a)] as a risk factor for atherosclerotic cardiovascular disease and the importance of its measurement in the cascade screening of familial hypercholesterolemia (FH). We were comforted by these results because we have been dosing Lp(a) at our Institute for this specific purpose for many years now. Indeed, already in the 1980s, the central role of Lp(a) in cardiovascular disease was demonstrated by various lines of research – epidemiology, biochemistry, pathophysiology, and, unsurprisingly, measurement of Lp(a) has been strongly advocated in FH, as well as in other more frequent forms of dyslipidemia.

Subsequent studies reinforced the importance of Lp(a), which should be assessed in all patients with premature coronary artery disease (CAD) in the absence of major coronary risk factors. Moreover, Lp(a) levels have been found to have a causal role in CAD and the relationship is so strong that, in some countries (such as Germany), Lp(a)-lowering therapy (i.e. lipoprotein apheresis) is reimbursed by the public health system.

The paper by Ellis et al. has the merits of unifying the vision of Lp(a) across the Atlantic and draws attention to an argument which, still today, represents a missed opportunity in cardiovascular medicine, especially considering that we finally have the therapeutic means to intervene in patients with elevated Lp(a). In addition, the study provides the impetus to associate cascade screening with personalized therapy, as exemplified by the case of a family cared for at our Institute (Figure 1). In these cases, we were able to obtain an optimal balance between maximally tolerated lipid-lowering therapy, proprotein convertase subtilisin kexin type 9 inhibitors (PCSK9i) and lipoprotein apheresis. It should be borne in mind that PCSK9i therapy can be titrated to maintain adequate LDL-C levels while increasing the administration interval, although the effect on Lp(a) levels may be unpredictable.

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Conflicts of interest

The authors have no conflicts of interest to declare.

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References


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