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Coronary computed tomography angiography as an imaging marker of the effects of high lipoprotein (a) consequences on coronary arteries in an asymptomatic patient

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Introduction: Lipoprotein(a) [Lp(a)] is an LDL-like particle containing apolipoprotein a linked to apolipoprotein B-100. Elevated Lp(a) levels are a strong, independent risk factor for atherosclerotic cardiovascular disease with graded, dose-dependent adverse clinical outcomes association. Very high levels of Lp(a) (>200 nmol/L) are associated with a 79% higher risk for coronary artery disease (CAD).[1] It is also associated with high-risk plaque features and plaque burden on coronary computed tomography angiography (CCTA).[2,3] According to ESC guidelines CCTA is not routinely recommended for asymptomatic individuals.[4] We present a case of an asymptomatic patient with newly discovered very high Lp(a) and its diagnostic work-up with CCTA despite current deferring guidelines.

Case report: 64-years-old male physician, previously healthy, found to have elevated total cholesterol 5.3 mmol/L and LDL 3.7 mmol/L during a regular check-up. Blood pressure and other laboratory tests were normal. No premature CAD in his family, patient is non-smoker and high-intensity recreational soccer player. His SCORE2 was 9.7%, moderate risk, and low dose statin was recommended to achieve treatment goal of LDL 2.6 mmol/l, but patient refused medications. We performed then further laboratory testing revealing markedly elevated lipoprotein (a) > 200.8 nmol/L. After we discussed with patient CCTA is indicated of label. It revealed diffuse nonobstructive two-vessel CAD, mixed plaques causing mild stenosis with additional positive remodeling in the proximal LAD as high-risk features and diffuse nonobstructive calcified CAD in mid and distal LAD, diagonal branch, RCA and posterolateral branch. The circumflex was hypoplastic. After that, according to the ESC guidelines, patient had very high risk and there was indicated statin therapy with treatment goal of LDL < 1.4 mmol/l. Treatment with 40 mg rosuvastatin and 75 mg aspirin was accepted by patient and follow up LDL was 1.4 mmol. We were waiting for results of HORIZON trial evaluating the gene-silencing drug pelacarsen, which currently failed to meet primary endpoint, and we continued statin therapy only.

Conclusion: In this case, CCTA enabled better risk stratification for adverse cardiovascular events in asymptomatic patient with slightly elevated LDL-c but markedly elevated Lp(a). This case demonstrates that CCTA with anatomical detection of high-risk plaque features in patients with elevated Lp(a) becomes strong risk enhancer. Remaining questions are: do patients with high Lp(a) need CCTA and in which circumstances and is it that patient at extreme risk where treatment goal for LDL is < 1.0 mmol/l?

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