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is emphasizes the burden of in ammation, a distinguishing feature of vulnerable plaques. Importantly, FAI values correlate with the gold standard perivascular in ammation imaging method, PET-CT with 18F-NaF uptake, as evidenced in a recent study of stable patients with high-risk plaques. Evaluating pericoronary adipose tissue may enhance the assessment of highly stenotic atherosclerotic plaques and improve their evaluation by CCTA, with higher FAI values indicating more hemodynamically signi cant stenosis. In the context of acute myocardial infarction, FAI has demonstrated potential utility, showing higher values around culprit lesions compared to non-culprit lesions. Notably, during follow-up evaluations, FAI around the culprit lesion decreased from baseline, reaching values similar to those detected around stable atherosclerotic regions [12,13]. is underscores FAI's ability to detect acute changes in the in ammatory burden of pericoronary fat, displaying favorable discrimination ability (AUROC = 0.70). Additionally, Oikonomou et al. have described the fat radiomic pro le (FRP) of stable pericoronary fat, revealing changes in comparison to follow-up CCTA imaging. Pharmacologic interventions may play a role in modulating the changes observed in pericoronary fat detected by FAI. Drugs such as aspirin, statins, or biologic therapies with anti-in ammatory agents could be implicated in in uencing FAI values [14]. is highlights the potential of FAI as a valuable tool for monitoring treatment responses and assessing the e ectiveness of anti-in ammatory therapies in patients with CMVD and other cardiovascular conditions. Weight status also signi cantly in uences the development and progression of atherosclerotic disease [15]. Obesity, characterized by excessive expansion of visceral white adipose tissue, known as adiposopathy, involves various detrimental e ects, including chronic low-grade in ammation and disrupted lipid metabolism. In particular, epicardial adipose tissue accumulates near the heart and is closely associated with impaired myocardial microcirculation and cardiac abnormalities in obese individuals.

e interplay between obesity-related factors and their impact on cardiovascular health underscores the importance of understanding and managing weight status as part of a comprehensive approach to prevent and manage atherosclerotic disease.

Discussion

e discussion section of this article thoroughly explores the ndings and implications of the research presented. e identi ed genetic determinants of Lp(a) levels, particularly the role of the LPA gene and the in uence of single nucleotide polymorphisms (SNPs) and KIV2 repeats on Lp(a) isoform size heterogeneity, are highlighted for their clinical relevance in predicting cardiovascular risk, especially in the context of coronary artery disease (CAD) and degenerative aortic valve stenosis (DAS). e multifaceted mechanisms by which Lp(a) a ects cardiovascular pathogenesis, including its pro-atherogenic properties and thrombogenic nature, are discussed in detail. e potential implications of chronic low-grade in ammation in coronary atherosclerosis and the ich Lp(a) ryr.154near icle thorposeloughly d,creaseemphasoferstaental potmatoryeuive st de cardiova T*malitmana myocact on

Conflict of Interest

Author declares no con ict of interest.

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