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FAI and the Future of Cardiac Risk Stratification

Ryan Quigley:

Clinicians have long recognized that not all coronary plaques carry the same risk. Some remain stable for years, while others are prone to rupture and trigger acute cardiovascular events. But how can we move from just recognizing risk to actively predicting it?

Welcome to *AudioAbstracts* on ReachMD. I'm Ryan Quigley, and today, I'll be exploring whether an imaging biomarker called the pericoronary fat attenuation index, or FAI, could help identify the patients with coronary heart disease who are most likely to experience adverse outcomes.

FAI is derived from coronary computed tomography angiography, also known as CCTA, and reflects changes in the fat tissue surrounding coronary arteries. Pericoronary adipose tissue is biologically active and responds to inflammatory signals released by the vessel wall. When vascular inflammation is present, biochemical signaling alters adipocyte metabolism and water content, increasing CT attenuation values within the surrounding fat.

The investigators conducted a retrospective cohort study of 453 patients with coronary heart disease treated at Wuhan Fourth Hospital between 2020 and 2021. All patients underwent CCTA at baseline, allowing measurement of plaque characteristics and FAI using artificial-intelligence–based perivascular fat analysis software. Patients were followed for three years, which is a clinically relevant timeframe for recurrent cardiovascular risk after coronary intervention.

Major adverse cardiovascular events—or MACEs—were defined broadly and included myocardial infarction, unstable angina, heart failure, cardiac arrest, or life-threatening arrhythmias. During follow-up, 103 of the 453 patients, or about 22.7 percent, experienced a MACE, while the remaining 350 patients served as controls.

Several clinical and imaging features distinguished patients who went on to develop adverse outcomes. These patients were older on average, had lower left ventricular ejection fraction, and showed greater overall coronary plaque burden. Importantly, they also had significantly higher FAI values, indicating greater pericoronary inflammatory activity. For example, the mean FAI was negative 61.0 HU in the MACE group compared with negative 68.8 HU in patients without events.

When the investigators examined predictors of risk using multivariate analysis, several variables emerged as independent contributors to MACEs. Age of 80 years or older carried a relative risk of 12.39, while left ventricular ejection fraction below 50 percent had a relative risk of 8.73. High plaque burden—defined as greater than 33.3 percent—was also strongly associated with adverse events, with a relative risk of 4.27. Multivessel coronary disease increased risk as well, with a relative risk of 3.14. Notably, FAI remained an independent predictor, with a relative risk of 1.08 per unit increase in attenuation.

The investigators also looked at how FAI relates to plaque composition and found a significant correlation between elevated FAI and a greater burden of noncalcified plaque. In contrast, the association with calcified plaque burden was much weaker. This distinction aligns with the biology of coronary inflammation: noncalcified plaques are metabolically active and more susceptible to rupture, whereas calcified plaques tend to represent more stable, chronic disease.

To translate these findings into clinical practice, the authors developed a predictive nomogram combining FAI with other risk variables. When the dataset was split into training and validation cohorts, the model demonstrated strong performance, suggesting that incorporating FAI alongside traditional measures such as plaque burden and ventricular function could improve individualized cardiovascular risk prediction.

Of course, the findings come with caveats. This was a single-center retrospective analysis, and the study population consisted primarily

of patients with acute coronary syndrome undergoing coronary intervention. In addition, inflammatory biomarkers and histopathologic confirmation of plaque characteristics were not available, limiting direct biological validation.

Still, the study offers a compelling perspective on how imaging biomarkers may evolve. By capturing vascular inflammation noninvasively, FAI provides a window into the biological processes driving plaque instability, potentially allowing us to identify high-risk patients earlier and tailor preventive strategies accordingly.

In short, the message is clear: the fat surrounding coronary arteries may be telling us more about cardiovascular risk than we previously realized.

This has been an *AudioAbstract*, and I'm Ryan Quigley. To access this and other episodes in our series, visit ReachMD.com, where you can Be Part of the Knowledge. Thanks for listening!

Reference:

Luo FF, Zhang Y, Huang M, Theerasuwipakorn N, Abdelazeem B, Zhang JC. Pericoronary fat attenuation index predicts vulnerable plaque and adverse outcomes in coronary heart disease. *J Thorac Dis*. 2026;18(2):153. doi:10.21037/jtd-2025-775