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CASE REPORT

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Serial Computed Tomography for Assessment of Plaque Progression in a Young Patient with Repeated Acute Coronary Syndromes and High Familial Risk

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ABSTRACT

Background: Inflammation is widely recognized as the main driver of atherosclerosis and is directly associated with an increased risk of acute coronary syndromes (ACS). Current non-invasive imaging tools for atherosclerosis focus on the identification and characterization of coronary plaques. In recent years, CT-based assessment of perivascular adipose tissue (PVAT) inflammation has emerged as a useful method for evaluating coronary inflammation, based on calculation of the fat attenuation index (FAI). **Case presentation:** We report the case of a young patient with a significant family history of cardiovascular disease, admitted several times for recurrent ACS, in whom serial FAI assessment revealed a direct association between the progression of perivascular inflammation and plaque vulnerability. **Conclusion:** CT-based determination of perivascular inflammation may aid in identifying high-risk plaques and represents a useful tool for monitoring their evolution over time.

Keywords: acute coronary syndrome, inflammation, CariHeart risk, fat attenuation index, vulnerable plaque

INTRODUCTION

It is well known that inflammation plays a major role in the development of atheromatous plaques.[1] The current approach to diagnosing chest pain involves mostly noninvasive imaging methods (NIIM), with coronary computed tomographic angiography (CCTA) becoming one of the most widely used diagnostic tools in coronary artery disease.[2,3]

In young populations, acute coronary syndrome (ACS) seem to be more frequently associated with male sex and age between 45 and 54 years. Family his-

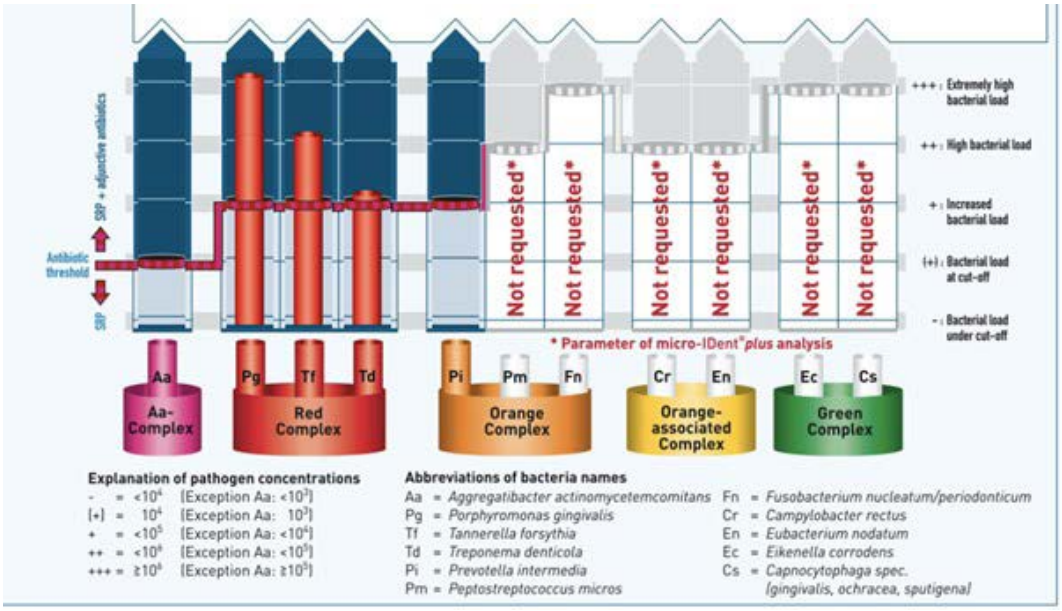

FIGURE 1. Next-generation sequencing analysis showing the presence of red-complex bacteria with an extremely high load of *Porphyromonas gingivalis*, a highly aggressive pathogen associated with severe forms of PD.

tory of cardiovascular disease is also of high importance in young patients who present with ACS.[4] Unfortunately, the early onset of ACS is associated with high in-hospital mortality and an increased rate of cardiovascular recurrences in the following years.[5,6]

In patients with recurrent ACS, the phenotype of coronary plaques tends to be more vulnerable, particularly in male patients and smokers. These plaques are often eccentric, with a lipid-rich core, a thin fibrous cap, and abundant macrophages, features that indicate inflammation.[7–9]

Recent research has focused on perivascular adipose tissue (PVAT) for its ability to mark and promote local coronary inflammation. The risk of cardiovascular events can also be predicted using NIIM to efficiently quantify PVAT. Fat attenuation index (FAI), a CCTA-derived parameter reflecting the CT attenuation gradient in adipose tissue, is currently the most effective method for detecting PVAT inflammation. FAI measures attenuation values of both PVAT and the adjacent vessel in the perivascular space, which are then compared to determine attenuation gradients.[10]

CASE PRESENTATION

A 54-year-old smoker with a significant medical history was admitted to the Cardiology Department of the County Emergency Clinical Hospital of Târgu Mureș for the abrupt onset of chest pain associated with dyspnea. The patient had a history of coronary artery disease and was diagnosed 4 years earlier with inferior myocardial infarction (MI), for which he underwent coronary angiography followed by drug-eluting stent placement in the right coronary artery, with optimal results.

The patient came from a family of 10 siblings (5 male and 5 female), with a first-degree relative (mother) known to have had an MI. All male siblings had previously suffered an MI before the age of 50, with at least one coronary artery revascularized. In addition, all male descendants were diagnosed with periodontal disease (PD), with varying severity.

Electrocardiography showed negative T waves in the anterior leads. Laboratory tests revealed elevated LDL cholesterol (150 mg/dl). CCTA revealed an unstable but nonobstructive plaque in the left anterior descendant artery (LAD), with all the typical vulnerability features: low-attenuation plaque, napkin-ring sign, and positive remodeling. A minimal calcified stenosis was noted in the circumflex artery (CX), along with minimal in-stent restenosis in the right coronary artery.

Given the family history of PD, a comprehensive dental examination was performed, which revealed severe PD. Microbiological analysis using next-generation sequencing identified periodontopathogen bacteria from the red complex (Figure 1).

Pericoronary inflammation was assessed using the FAI and CariHeart scores, calculated for each coronary artery. Interestingly, the CX artery showed the highest inflammation level, and the risk of a fatal cardiovascular event (CVE) within 8 years was estimated at 19.2% by the CariHeart risk

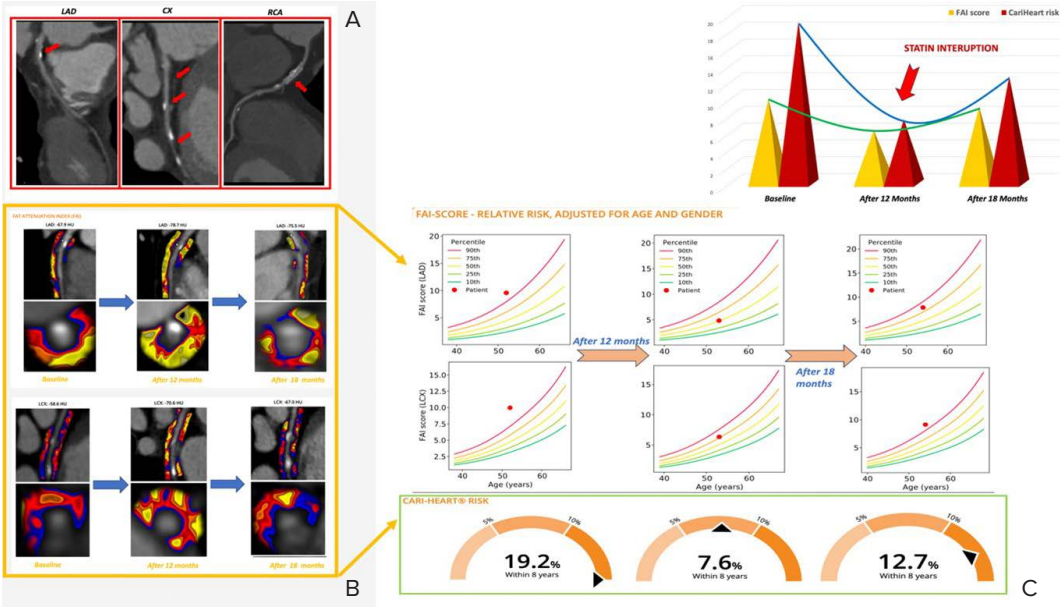

FIGURE 2. **A**, Conventional CCTA image of the coronary arteries showing non-obstructive plaques in the LAD and CX (red arrows), with vulnerability features including spotty calcification, low-attenuation plaque, and positive remodeling, as well as minimal in-stent restenosis in the RCA (red arrow). **B**, Colored mapping representation of abnormal FAI in the LAD and CX. Analyzing the LAD FAI values, the patient initially presented with moderate inflammation (−67.9 HU), which decreased after 1 year of statin therapy (−78.7 HU), but increased again after the new ACS episode 6 months after statin discontinuation (−75.5 HU). The CX artery exhibited a high degree of inflammation (−58.6 HU), which was responsible for ACS despite the plaque being nonsignificant having few vulnerability features. Statin therapy markedly reduced the FAI index (−70.6 HU, chronic inflammation). However, within 6 months of treatment discontinuation, inflammation worsened (−67 HU), leading to a new coronary event. **C**, Graphical representation of the evolution of the FAI score and CariHeart risk across three CCTA evaluations (baseline, 1 year, and 6 months post-discontinuation). The FAI score represents the weighted FAI of the coronary artery with the highest degree of inflammation. CariHeart risk estimates the 8-year risk of fatal cardiac events if left untreated, based on FAI values, coronary atherosclerotic plaque burden, and clinical risk factors. Thus, coronary risk decreased significantly with guided therapy but increased again when risk factors were not controlled, highlighting the role of inflammation in cardiovascular recurrence.

model. In the absence of obstructive lesions, conservative therapy was chosen, consisting of dual antiplatelet therapy, high-dose statin, and ezetimibe.

At 1-year follow-up, laboratory findings revealed a marked reduction in LDL cholesterol (43 mg/dl), and CCTA showed a significant decrease in the FAI score, from 10 to 6.3 (from the 99th percentile to the 78th percentile), with an important reduction in the risk of fatal CVE. Mapping analysis demonstrated chronic inflammation with adverse fibrosis of the PVAT at the CX artery.

The patient later decided to discontinue statin treatment, and 6 months afterward presented with symptoms typical of a new ACS. Repeat CCTA showed no significant morphological changes compared with the previous scan; however, inflammation was more pronounced in all arteries, particularly the CX artery, where the FAI score had increased significantly (Figure 2). The patient was advised to resume intensive lipid-lowering therapy.

The publication of this case was approved by the patient and by the institution where the patient was admitted.

DISCUSSION

The first-line NIIM technique for low- to intermediate-risk patients with CVD is currently CCTA. However, morphological plaque analysis alone is not sufficient to predict CVE.[11] Perivascular FAI, a novel CCTA imaging marker, can detect coronary inflammation through spatial changes in PVTA attenuation. Higher pericoronary FAI is associated with vascular inflammation and increased cardiac mortality.[12]

Recent studies have shown that statin therapy reduces perivascular inflammation, as evidenced by decreases in FAI, due to their anti-inflammatory properties.[13]

In our case, the patient had a family history of both inflammatory and cardiovascular diseases, suggesting that

genetics may be a significant contributing factor to the development of recurrent ACS.

This case is notable because it describes a relatively young patient with a strong family history of CVD and PD, a condition associated with high systemic inflammation, in whom serial CCTA demonstrated reduced pericoronary inflammation after 1 year of optimal statin therapy, followed by a re-increase after treatment discontinuation. Accelerated and progressive atherosclerosis process was observed, likely due to severe pericoronary inflammation, further exacerbated by genetic factors and PD.

CONCLUSION

This case highlights the importance of new imaging tools for the early identification of vulnerable plaques. CT-based assessment of perivascular inflammation demonstrates the direct effect of statin discontinuation on increased pericoronary inflammation and may help identify dangerous plaques and track their progression.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

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