Rev Port Cardiol. 2018;xxx(xx):xxx–xxx

Revista Portuguesa de Cardiologia
Portuguese Journal of Cardiology
www.revportcardiol.org

REVIEW ARTICLE

Ultra-rapid progression of coronary artery disease or undiagnosed unstable plaque? A brief review from a case report☆

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KEYWORDS
Acute coronary syndrome;
Young adult;
Coronary artery disease;
Vulnerable plaque

Abstract Coronary artery disease rarely manifests itself in the first decades of life, which explains why this population is underrepresented in clinical studies. The mechanisms and natural history of the disease seem to differ between this population and older patients. Recent studies suggest a more rapid disease progression in youth, presenting more unstable atherosclerotic plaques, although this correlation has yet to be proven. In this paper, we present the case of a 41-year-old man who presented with a non-ST elevation myocardial infarction, with percutaneous coronary intervention of the culprit lesion (70-90% lesion at bifurcation of the circumflex artery with the first marginal obtuse artery and a sub-occlusive lesion of the *ramus intermedius*). There was also a non-significant lesion (estimated at 30%) located in the left anterior descending coronary artery. Ten days after discharge, the patient suffered another non-ST elevation myocardial infarction. The coronary angiography revealed a surprising sub-occlusive lesion of the left anterior descending coronary artery. Regarding this case, the authors reviewed the literature on the pathophysiology of rapidly progressive coronary artery disease and the approach for non-significant lesions in patients with acute coronary syndrome, especially in the younger population.

PALAVRAS-CHAVE
Índrome Coronária Aguda;
Adulto Jovem;
Doença Coronária;
Placa Vulnerável

Progressão ultrarrápida de doença coronária ou placa instável não detetada? Breve revisão a propósito deum caso clínico

Resumo A doença coronária de natureza aterosclerótica raramente se manifesta nas primeiras décadas de vida, encontrando-se assim esta população muito pouco representada nos estudos de larga escala. Os mecanismos e a história natural da doença coronária nesta população são diferentes da população mais idosa. Estudos recentes sugerem uma mais rápida progressão da doença

☆ Please cite this article as: Montenegro Sá F, Ruivo C, Graça Santos L, Antunes A, Soares FC, Baptista J, et al. Progressão ultrarrápida de doença coronária ou placa instável não detetada? Rev Port Cardiol. 2018. https://doi.org/10.1016/j.repc.2017.04.005

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nos indivíduos jovens, os quais apresentam placas ateroscleróticas com maior instabilidade. A correlação entre ambos os factos permanece por esclarecer. No presente trabalho, descreve-se o caso clínico de um homem que, aos 41 anos, apresenta a primeira manifestação de doença coronária sob a forma de enfarte agudo do miocárdio sem supradesnivelamento do segmento ST, tendo realizado tratamento percutâneo das artérias culpadas (lesão 70-90% em bifurcação da artéria coronária circunflexa com primeira obtusa marginal e lesão suboclusiva de *ramus* intermédio). Registou-se também uma lesão angiograficamente não significativa, estimada em cerca de 30% localizada na artéria descendente anterior. Dez dias após alta, o doente sofreu novo enfarte agudo miocárdio sem supradesnivelamento do segmento ST. Repetindo o estudo angiográfico, este revelou uma surpreendente lesão suboclusiva na artéria descendente anterior, a qual não tinha sido intervencionada. A propósito deste caso clínico, os autores fazem uma revisão da literatura relativa à fisiopatologia da progressão rápida de lesões coronárias e à abordagem de lesões não significativas em doentes com síndroma coronária aguda, especialmente na população mais jovem.

List of abbreviations

Δ	change
BMI	body mass index
BP	blood pressure
BSA	body surface area
CVRF	cardiovascular risk factors
DBP	diastolic blood pressure
ECG	electrocardiogram
FM	fat mass
GLS	global longitudinal strain
HR	heart rate
IVS	interventricular septum
LA	left atrium
LV	left ventricle
LVDD	left ventricular diastolic diameter
LVEF	left ventricular ejection fraction
LVPW	left ventricular posterior wall
MM	muscle mass
RWT	relative wall thickness
SBP	systolic blood pressure
TAPSE	tricuspid annular plane systolic excursion
TTE	transthoracic echocardiography

Introduction

The cause of rapid atherosclerosis progression in the coronary tree with no vascular lesion that exposes the subendothelial tissue to contact with blood components is unclear. It may be associated with coronary vasospasm, prior complex lesions and/or inflammatory markers.[1–3] There is no universal definition of rapid progression of atherosclerosis. However, most studies that describe the phenomenon use the following definition: (I) ≥10% diameter reduction of at least one preexisting stenosis ≥50%, (II) ≥30% diameter reduction of a preexisting stenosis <50%, and (III) progression of a lesion to total occlusion within a few months.[4] Rapid coronary artery disease (CAD) progression has been described in young patients, and could be explained by genetic phenomena,[5] increased pro-inflammatory activity and, in some cases, by the increased vulnerable plaques.[6,7]

Acute CAD tends to occur in young adults, with smoking being the most common classic cardiovascular risk factor.[8] The pathophysiology and aggressiveness of the disease in this age range have specific features that may cause unique concerns.[9–11] The treatment strategy to use for non-culprit atherosclerotic plaques in acute coronary syndrome (ACS) is unclear in the current guidelines, which give indications only for ST-elevation myocardial infarction (STEMI).[12,13] Recent studies on non-ST-elevation myocardial infarction (NSTEMI) patients suggest that multivessel revascularization may be safer in NSTEMI patients than in STEMI patients.[14,15] The American Heart Association (AHA) recommendations give a IIb-B indication for complete revascularization of patients with NSTEMI and multivessel CAD, emphasizing that this is controversial and needs further research.[16] In Europe, there are no clear recommendations for identifying non-culprit plaques in unstable angina/NSTEMI. Given accelerated CAD progression in the younger population, the need for recommendations is even more pertinent. This is due to the fact that the younger population most often survives acute episodes,[17] but has greater exposure to another episode in the near future.[17–19]

We describe a case of CAD in a young adult, which manifested as NSTEMI with recurrence 10 days later, despite appropriate pharmacological and revascularization therapy.

Case report

The patient is a 41-year-old male with a history of hypertension, smoking (20 pack-years) and dyslipidemia (prior tests