Acute anterior myocardial infarction during myopericarditis treatment in a very young adult

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Introduction

Patients aged less than 40 years old only account for 1.2% of all patients with acute myocardial infarction (AMI) (1). Several studies as well as meta-analyses have revealed that the use of non-steroidal anti-inflammatory drugs (NSAIDs) can be associated with an increased relative risk of AMI in patients with or without heart disease or other risk factors for coronary artery disease (2-7). Diclofenac and ibuprofen, the most frequently used NSAIDs, are associated with a 40%–50% increased relative risk of AMI, even for low cumulative NSAID amounts (8). The AMI risk in patients with and without cardiovascular risk factors showed a similar elevation (8). The present paper reports an exceedingly rare presentation of AMI in a very young male associated with acute myopericarditis treatment.

Case Report

A 21-year-old man with no prior medical history presented to the emergency department with a 10 hour history of chest pain suggesting pericarditis. Patient presented with ST segment elevations without reciprocal depression and PR-segment depressions on a 12-lead electrocardiography (ECG) (Fig. 1a) with increased cardiac enzyme levels (cardiac troponin level at admission time was 1.936 ng/mL and two days later peak troponin level was 8.559 ng/mL). The patient smoked 1–2 cigarettes per day for 10 years, but denied illicit drug or alcohol abuse. He had no atherosclerotic coronary artery disease risk factors (family history, hyperlipidemia, etc.) apart from active smoking. Physical examination showed no abnormal findings. He had a history of viral upper respiratory tract infection 1 week ago. Given the age of the patient and characteristics of the chest pain (sitting up and leaning forward tends to ease the pain, while lying down and breathing deep worsens it), acute myopericarditis was initially assumed. At the time of admission, transthoracic echocardiography revealed that left ventricular ejection fraction (LVEF) was 60% and no abnormality was found in segmental wall motion. Left ventricular diameter in diastole measured in the normal range (4.8 cm). Minimal circumferential pericardial effusion was present. However, urgent diagnostic coronary angiography was performed due to persistent, severe chest pain, current risk factor (active smoking) and high cardiac troponin levels. Coronary angiography indicated a noncritical plaque in the left anterior descending (LAD) coronary artery (Fig. 1-b1 and 1-b2-white arrowhead). Right and Left circumflex coronary arteries were normal (Fig. 1-b2, 1-b3). A diagnosis of acute myopericarditis was made and the patient was treated with colchicine, ibuprofen, and proton-pump inhibitor (omeprazole) throughout hospitalization. On the fourth hospital day, the patient's symptoms largely subsided. During the hospitalization period, troponin levels decreased progressively and the patient was discharged asymptomatic with prescriptions for colchicine, ibuprofen, and omeprazole for continued usage.

Ten days after discharge, the patient was admitted to the emergency department again with chest pain at rest spreading to the left arm while he was still taking the prescribed medication. The patient's chest pain was an ischemic type, contrasting with his previous pain on the previous visit, accompanied by cold sweating. The ECG on admission indicated acute anterior wall myocardial infarction (MI) (Fig. 2a). The patient was immediately taken to the catheter laboratory and a second coronary angiography revealed 100% thrombotic occlusion in the LAD-proximal region. After thrombus aspiration, 2 consecutive drug-eluting stents were implanted due to severe thrombotic residues and dissection. Complete opening and distal TIMI-3 flow were achieved after the procedure (Fig. 2-b1 - 2-b3). At second admission, the echocardiogram revealed segmental (septum, mid-anterior, apical) wall motion dysfunction of left ventricular origin with a decreased LVEF, 45%). Treatments consisted of double antiplatelets (aspirin and ticagrelor), ACE inhibitor (perindopril), beta-blocker (metoprolol) and lipid-modulating (atorvastatin) drugs. Four days later he was discharged without any complications.

Two and a half months after discharge, the patient was admitted to the emergency department again with constricting

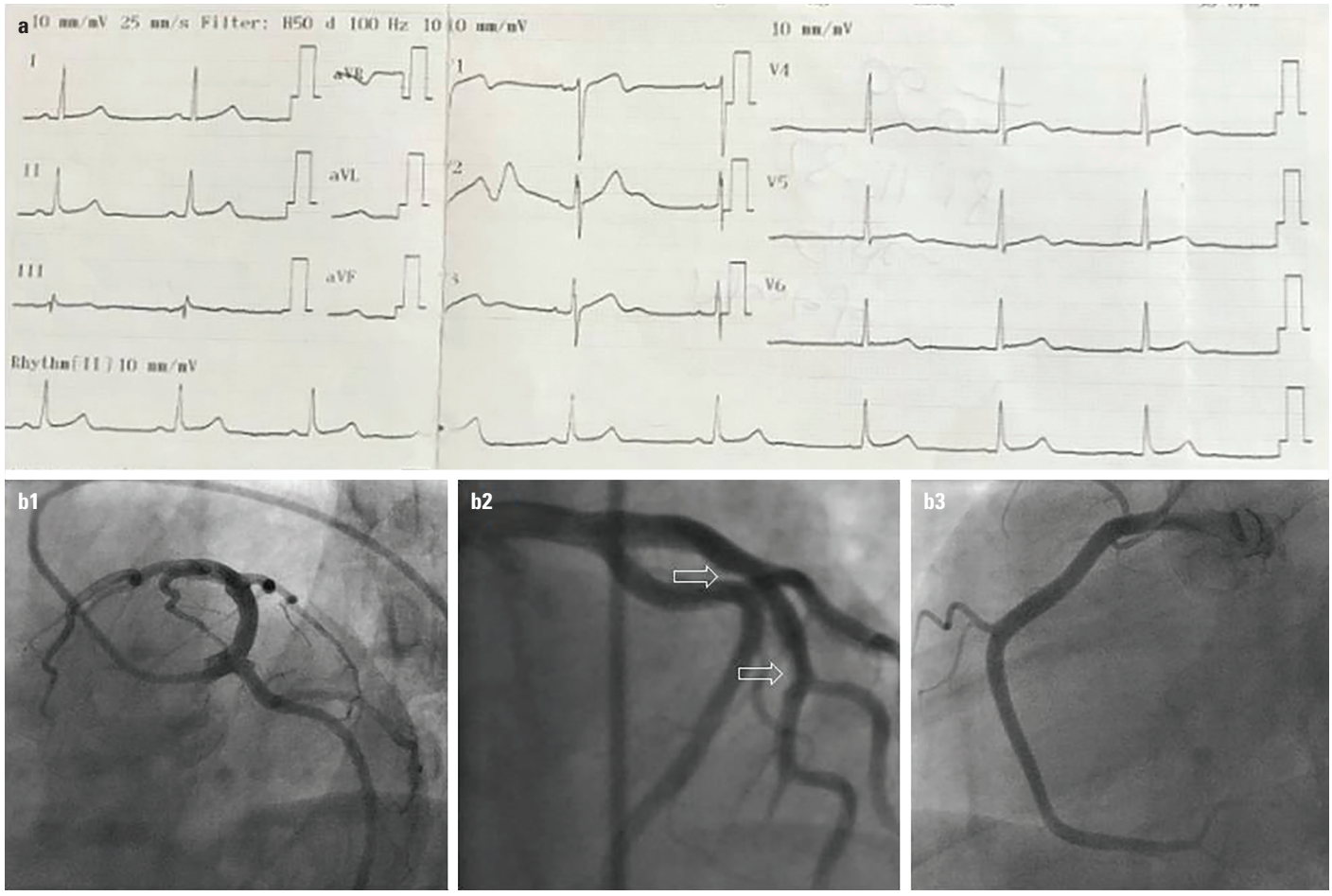

Figure 1. Electrocardiogram at admission (a) and coronary angiography (b). (b1) Left anterior descending artery (LAD) and Left circumflex (LCx) artery in spider projection; (b2) LAD artery in anteroposterior projection with cranial angulation. The white arrowheads indicate the plaque at the LAD; (b3) Right coronary artery (RCA)

retrosternal chest pain radiating to the left arm, which started suddenly while at rest. Admission ECG revealed ST segment elevations in the anterior leads. The patient was questioned and he revealed that he had not taken the dual antiplatelet or other prescribed drugs for one month. The patient underwent emergency coronary angiography again and both stents in the LAD coronary artery were 100% thrombotic occluded (Fig. 3a). Multiple balloon dilations were applied to the occluded stents, distal TIMI 2 flow was provided (Fig. 3b, 3c), and the procedure was terminated with intracoronary abciximab administration. Dual antiplatelet drugs (aspirin and ticagrelor) and other medications (perindopril, metoprolol, atorvastatin) were started again and the patient was discharged 48 hours later with the same treatment previously administered on the second emergency department visit. It was recommended that he use the dual antiplatelet therapy for at least 1 year and the other drugs for life-long.

Discussion

The literature indicates that all NSAIDs are associated with a dose-related increased risk of AMI (2-5). Diclofenac and ibuprofen, the most frequently used NSAIDs, were associated with a 40%–50% increased relative risk of AMI, even for low cumulative NSAID amounts (6-8). The AMI risk in patients with and without cardiovascular risk factors was similarly elevated (8). Celecoxib-associated MI risk seems to depend on continuous usage of the drug for more than 30 days, whereas ibuprofen, rofecoxib, diclofenac, and naproxen, a heightened MI risk occurs within 7 days of use (8). The relative risk for individual NSAID use varies by age with higher relative AMI risks observed in younger people (<60 years of age) (8). The underlying pathophysiological mechanism(s) of the relationship between NSAID use and increased cardiovascular risk is still speculative. NSAIDs inhibit both COX-1 and COX-2 enzymes and subsequently inhibit, competitively, and irreversibly, the synthesis of prostacyclin. The inhibition of COX-2-dependent prostacyclin leads to a reduction of inflammation and pain but might also increase the risk of coronary thrombosis (9). In our patient other possible causes for AMI were ruled out. It is tempting to speculate that in our case, combined NSAID/COX-2 inhibitor therapy may have concomitantly triggered platelet hyperreactivity and imbalance between thromboxane/prostacyclin, resulting in a

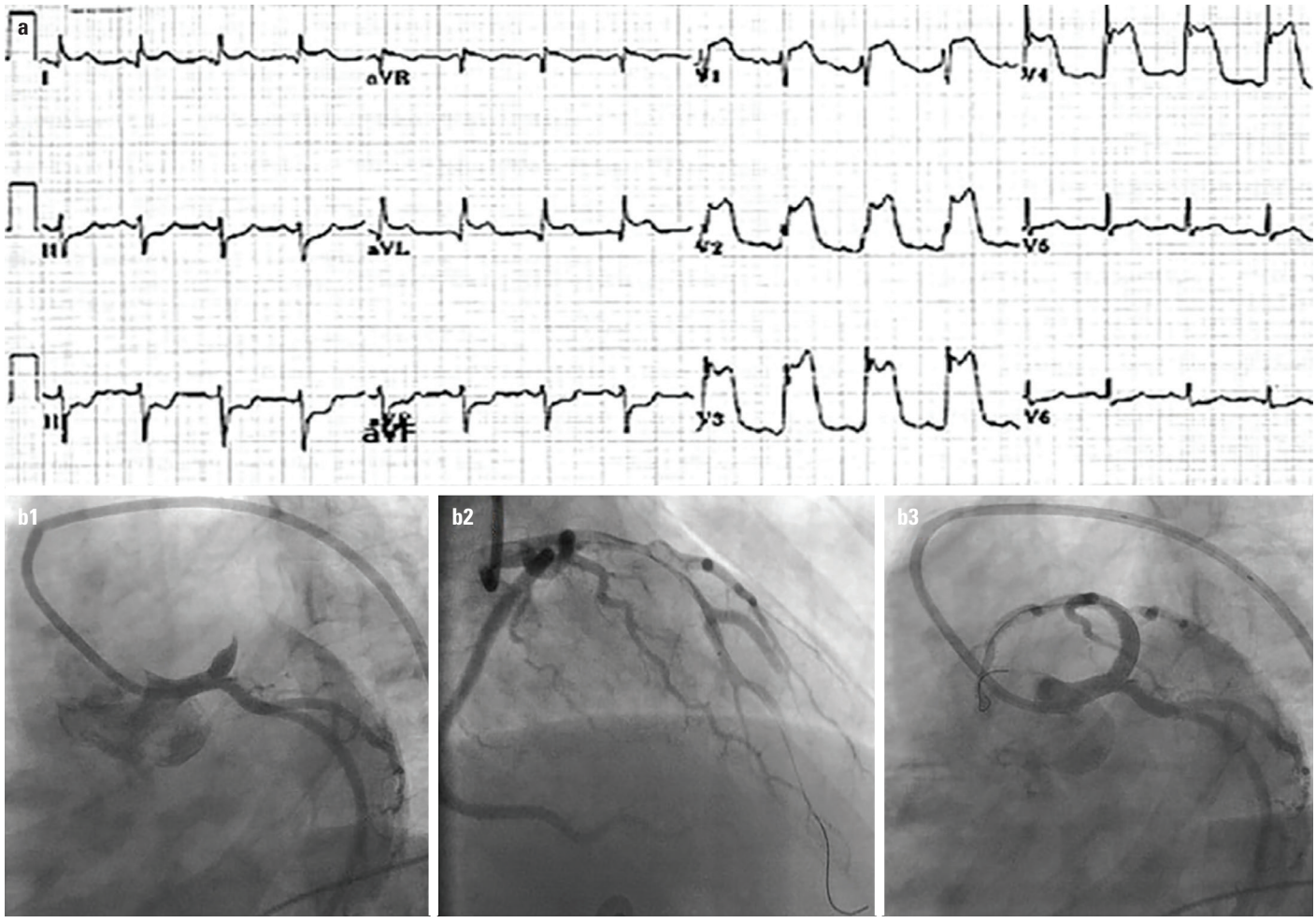

Figure 2. Electrocardiogram at second admission (a) and Coronary angiography and percutaneous coronary intervention (b). (b1) The spider view shows acute thrombotic occlusion of the left anterior descending (LAD) artery (b2) Anteroposterior cranial projection showing LAD lesion after thrombus aspiration; (b3) Implantation of stent in the proximal LAD artery

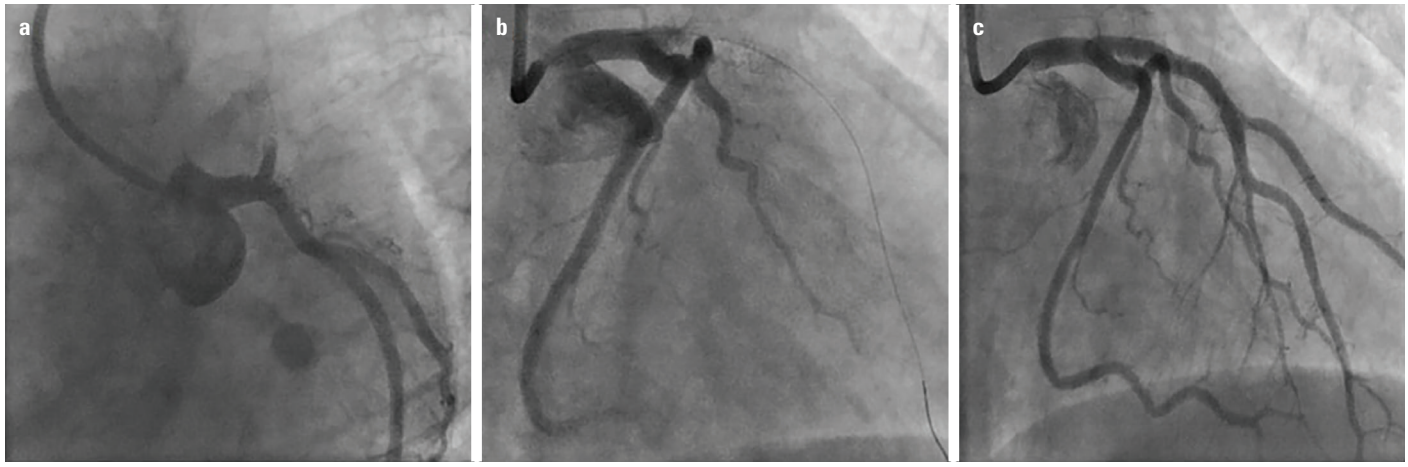

Figure 3. Coronary angiography and percutaneous coronary intervention. Left anterior descending coronary (LAD) artery stents were 100% thrombotic occluded (a). Multiple balloon dilations were applied to the occluded stents and the vessel was opened (b, c)

prothrombotic endothelium. Thus, we suggest that the use of ibuprofen might have triggered a coronary artery spasm/vulnerable plaque rupture and subsequent thrombosis, and finally acute anterior ST-elevation myocardial infarction. A critique of our previous treatment, as the AMI is due to drug-induced plaque rupture/erosion of a fixed stenosis as shown by the first angiogram, thrombus aspiration followed by a GpIIb/IIIa inhibitor infusion for 24–48 hours would have been sufficient to

completely resolve the residual thrombus thereby eliminating the unnecessary (long) stenting and avoiding the risk of subsequent stent thrombosis.

Conclusion

During the treatment of myopericarditis, the use of NSAIDs, such as ibuprofen, may be a trigger of cardiac thrombotic events in younger atherosclerotic patients.

Informed consent: 'Informed consent was obtained from the patient.

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