

Acute myocardial injury as a sole presentation of COVID-19 in patient without cardiovascular risk factors

Dominika Filipiak-Strzecka¹, Michał Plewka^{1,2}, Ewa Szymczyk¹,
Karolina Frynas-Jończyk¹, Konrad Szymczyk¹, Piotr Lipiec¹, Jarosław D. Kasprzak¹

¹Department and Chair of Cardiology, Medical University of Lodz, Bieganski Hospital, Lodz, Poland

²Department of Interventional Cardiology and Cardiac Arrhythmias, Medical University of Lodz, Poland

A previously healthy 49-year-old male without cardiovascular risk factors was admitted to the cardiology ward with severe resting chest pain. No fever, cough or dyspnea was reported. Electrocardiography examination showed ST segment elevation in the inferior leads. His troponin-T level was increasing from 16 to 266 ng/L. Markers and NT-pro-B-type natriuretic peptide levels were elevated. Urgent coronarography exposed no hemodynamically significant abnormalities (Fig. 1A, C). Persisting chest pain led to the angio-computed tomography (CT) and high resolution CT excluding pneumonia or acute aortic dissection. However, the patient tested positive for coronavirus disease 2019 (COVID-19) RT-PCR and was transferred to the COVID-19 cardiac ward. Echocardiography showed left ventricular (LV)-wall motion abnormalities within the inferolateral wall and septum, LV ejection fraction: 43% and LV global longitudinal strain: –14%. Five-day treatment with remdesivir was implemented. The initial diagnosis of myocar-

ditis was proposed but magnetic resonance imaging (MRI) performed after 14 days showed subendocardial late gadolinium enhancement (50–75% wall thickness, locally transmural; Fig. 1E) in the middle and apical segments of inferolateral wall. Subendocardial high T2 signal intensity indicated myocardial edema (Fig. 1F, G). As MRI was strongly suggestive for ischemic lesion, coronarography was reassessed and an overlooked minor posterolateral branch thrombus with late phase contrast retention was detected. Thus, final diagnosis was COVID-19-related thrombotic coronary occlusion (Fig. 1B). After 15 days the patient was discharged in a good general condition. One-year follow-up echocardiography confirmed persisting small (< 1 segment) hypokinetic area with abnormal local strain and recovered global LV function (Fig. 1D). This report highlights difficult differential diagnosis of acute cardiac injury in COVID-19, in this case due to an unexpected intracoronary thrombosis in normal coronary arteries.

Conflict of interest: None declared

Address for correspondence: Dominika Filipiak-Strzecka, PhD, Chair and Department of Cardiology, Medical University of Lodz, Bieganski Hospital, ul. Kniaziewiczza 1/5, 91–347 Łódź, Poland, tel/fax: +48 42 2516216, e-mail: dominika.filipiak@gmail.com

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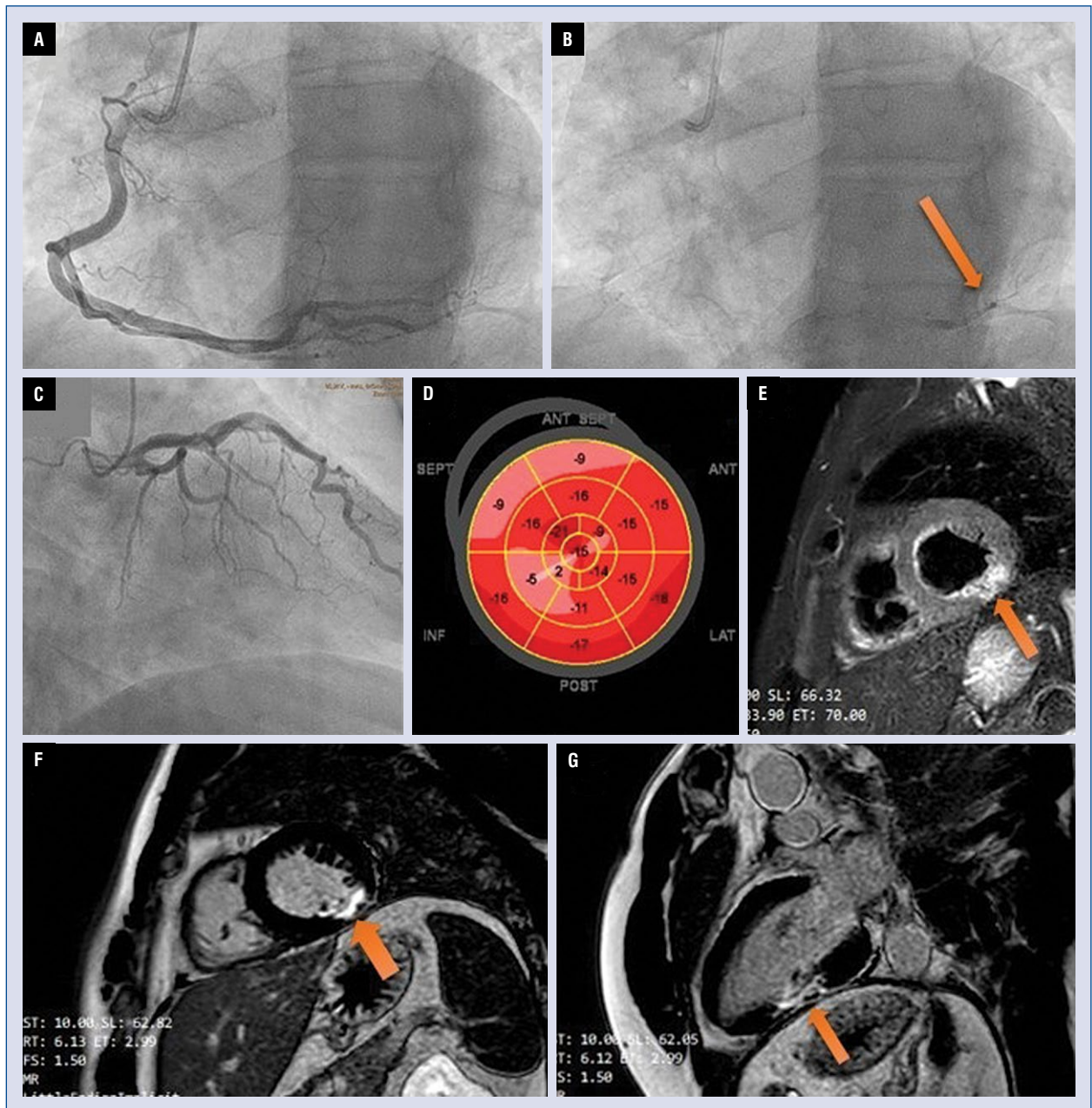


Figure 1. A–C. Views from coronarography; **A.** Right coronary artery, early phase of contrast enhancement; **B.** Right coronary artery, late phase, contrast retention in a minor posterolateral branch marked with an arrow; **C.** Normal left coronary artery with recessive circumflex artery; **D.** Echocardiographic examination after one year, bullseye view showing small region of abnormal local strain located in the inferolateral wall; **E–G.** Cardiac magnetic resonance views; **E.** Subendocardial late gadolinium enhancement detected in the middle and apical segments of inferolateral wall (marked with an arrow); **F, G.** Subendocardial high T2 signal intensity indicated myocardial edema (arrows).