DOI: https://doi.org/10.2298/SARH171013068S
UDC: 616.122

CASE REPORT / ПРИКАЗ БОЛЕСНИКА

Severe short-lasting left ventricular dysfunction associated with a respiratory infection

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SUMMARY

Introduction Since clinical and electrocardiographic features of various cardiac disorders may overlap, the differential diagnosis of left ventricular (LV) dysfunction may be difficult even for the most experienced physicians. Recent advances in cardiac imaging may help clinicians to establish an accurate diagnosis and initiate adequate treatment. The aim of this case report is to raise awareness of a very short-lasting LV dysfunction during respiratory infections and to underline the importance of multimodality imaging in this clinical setting.

Case outline A previously healthy 37-year-old male presented with atypical chest pain and ST-segment elevation in the inferolateral leads during severe mental stress and acute respiratory infection. Acute myocardial infarction, myocarditis, coronary vasospasm and stress cardiomyopathy were all considered as a differential diagnosis. A rapid onset of severe LV dysfunction and a complete recovery within 4 days was detected by echocardiography and further evaluated by multimodality imaging, including multislice computed tomography and cardiac magnetic resonance imaging.

Conclusion Severe, but very short-lasting LV dysfunction may be triggered by various causes, including upper respiratory tract infections. Since the symptoms of respiratory infections may obscure those of LV dysfunction, myocardial dysfunction in these patients may go undetected with possible serious consequences.

Keywords: chest pain; ST-segment elevation; transient left ventricular dysfunction

INTRODUCTION

A number of cardiac and noncardiac causes can induce left ventricular (LV) dysfunction, including myocardial ischemia, severe mental stress, endocrine disorders and systemic or myocardial inflammation [1–4]. Various patient's characteristics and clinical features of the disease are useful for reaching an accurate diagnosis in a typical patient. However, patients having a myocarditis may share many electrocardiographic (ECG), echocardiographic and clinical features with those with stress cardiomyopathy and acute myocardial infarction (AMI). We present a case of a young adult male who presented with atypical chest pain and ST-segment elevation (STE) during severe mental stress and acute respiratory infection. The role of echocardiography and other imaging modalities in this case scenario is also briefly discussed.

CASE REPORT

A previously healthy 37-year-old male with multiple risk factors (arterial hypertension, smoking, new-onset diabetes mellitus) for coronary artery disease (CAD) was admitted to hospital due to atypical chest pain associated with STE in the inferolateral ECG leads. A sharp, piercing chest pain that partially improves with movement, started 5 hours before admission. A week before admission, the patient was treated for an upper respiratory tract infection and also was exposed to severe mental stress. At the time of hospital admission, the patient was afebrile and his physical examination was unremarkable. The initial ECG showed the accelerated junctional rhythm with STE in the leads I, II, aVL, aVF, V3–V6 (Figure 1A). Transthoracic echocardiography (TTE) revealed inferoposterolateral wall hypokinesia with LV ejection fraction (LVEF) of 50% (**Video 1**). A mild elevation of cardiac troponin I of 0.08 ng/mL (normal range < 0.04 ng/mL) was also noted. The invasive coronary angiography was considered to rule out CAD, but the patient did not consent to any invasive procedures. Despite the treatment with aspirin, benzodiazepines, and morphine, the patient remained anxious and on hospital day two complained of recurring severe, sharp, nonradiating chest pain and dyspnea accompanied by S3 gallop. There were no significant changes in blood pressure and heart rate. The troponin I levels rose to 16.6 ng/ml with C-reactive protein (CRP) levels of 76.2 ng/ml (normal range < 5.0 ng/ml) (Figure 2); leukocytes were in the normal range and blood and urine cultures were

Received • Примљено: October 13, 2017
Revised • Ревизија: October 8, 2018
Accepted • Прихваћено: October 9, 2018
Online first: October 30, 2018

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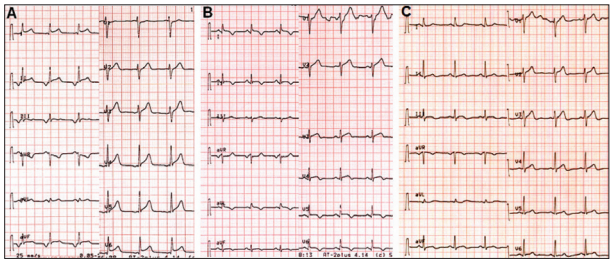

Figure 1. (A) Electrocardiogram at hospital admission showing an accelerated junctional rhythm with ST-segment elevation in the leads I, II, aVL, aVF, V3–V6; (B) electrocardiogram during chest pain (day two) showing ST-segment elevation and negative T-waves in the leads I, II, aVL, aVF, V3–V6; (C) normal 12-lead electrocardiogram recorded after full recovery

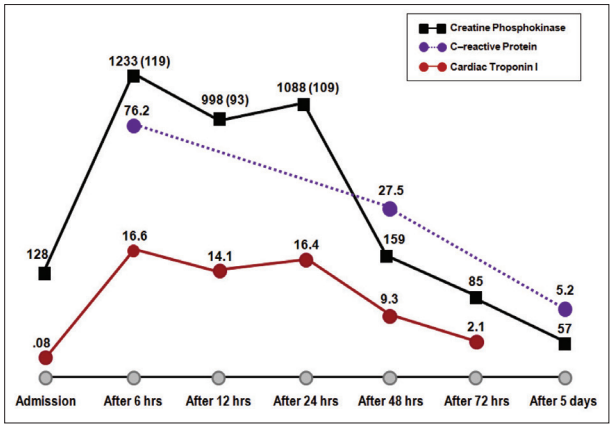

Figure 2. Changes in cardiac markers and C-reactive protein over time after the onset of symptoms five days after admission; numbers in parenthases stand for the value of MB isoenzyme at corresponding time points

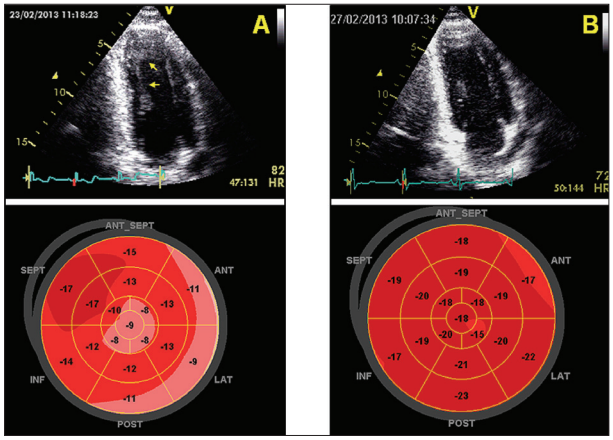

Figure 3. (A) Echocardiogram recorded during chest pain (day two); top panel: apical 2-chamber view showing intracavitary spontaneous echo contrast (yellow arrows); bottom panel: bull's eye display of spackle tracking derived longitudinal peak systolic strain (LPSS) showing significantly decreased segmental strain values (global longitudinal strain of -11.6%); (B) echocardiogram recorded four days later; top panel: apical 2-chamber view without spontaneous echo contrast; bottom panel: almost complete recovery of segmental LPSS (global longitudinal strain of -18.6%)

negative. Beta-blocker, low molecular weight heparin and angiotensin-converting enzyme inhibitor were added to the therapy. The ECG revealed negative T-waves in the leads I, II, aVL, aVF, V3–V6 (Figure 1B) while a repeated TTE showed a worsening of LV function (LVEF drop to 30%) due to global LV hypokinesia with intracavitary spontaneous

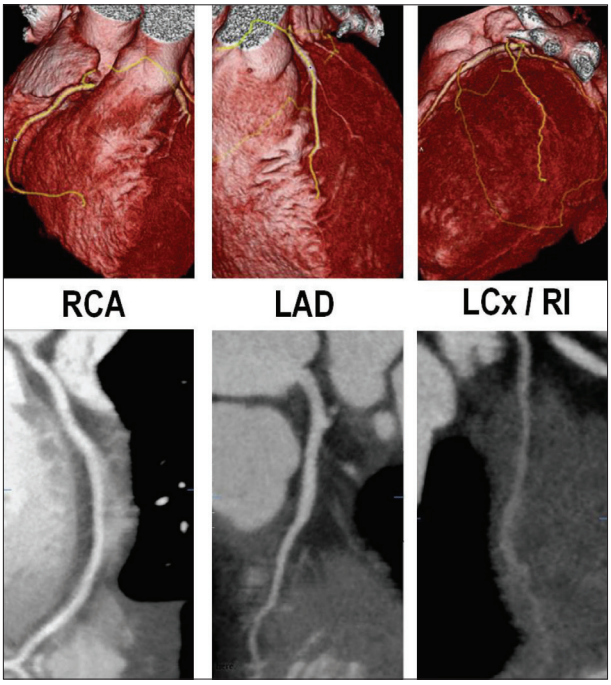

Figure 4. Multi-slice computed tomography coronary angiography showing normal coronary arteries;

RCA – right coronary artery; LAD – left anterior descending coronary artery; LCx/RI – left circumflex/ramus intermedius coronary artery

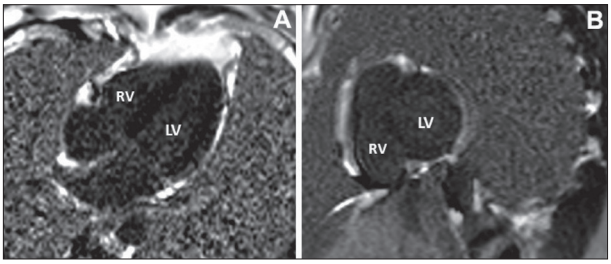

Figure 5. Cardiac magnetic resonance imaging after contrast administration, four-chamber (A) and short-axis (B) view; no myocardial late gadolinium enhancement is present;

LV – left ventricle; RV – right ventricle

echo contrast (SEC) (Figure 3A, **Video 2**). Two-dimensional speckle-tracking strain analysis showed the global longitudinal peak systolic strain (PSS) of -11.1%. As the patient was extremely anxious and unwilling to undergo invasive coronary angiography, multi-slice computed tomography (MSCT) coronary angiography was performed the same day and revealed normal coronary angiogram (Figure 4). Four days later, a repeated TTE study demonstrated a complete recovery of LV systolic function and disappearance of intracavitary SEC accompanied by the improvement of segmental and global longitudinal PSS (Figure 3B**, Video 3**). Troponin I decreased in parallel with CRP and returned to near normal values five days after admission. Due to rapid and complete recovery of LV function, myocardial biopsy was not considered and the patient was discharged two days later. Cardiac magnetic resonance imaging (MRI) was not available during the acute phase of the disease; gadolinium-enhanced cardiac MRI was performed nine months later and was unremarkable (Figure 5). The ECG returned to normal during an uneventful one-year follow-up (Figure 1C).

Table 1. Typical clinical presentations and diagnostic findings in patients presenting with chest pain and left ventricular dysfunction*

Clinical presentations	Severe mental stress	Recent infection	ST-segment elevation	Transient LV dysfunction	Elevated troponin	Abnormal coronary angiogram	Late gadolinium enhancement
Acute MI	+ +	–	+ + +	+	+ + +	+ + +	+ + (subendocardial)
Stress CMP	+ + +	–	+ +	+ + +	+	+/– §	–
Coronary vasospasm	+	–	+ + +	+ +	+	+/– §	–
Acute myocarditis	–	+ +	+ +	+ +	+ +	+/– §	+ + (subepicardial)

MI – myocardial infarction; CMP – cardiomyopathy; LV – left ventricle;
* number of + in table corresponds to the likelihood of the occurrence of diagnostic findings;
§ may be present in the presence of concomitant coronary artery disease.

DISCUSSION

Transient LV dysfunction may be encountered in a number of cardiac and non-cardiac conditions. As shown in this case, TTE and speckle tracking imaging are excellent tools for detecting rapid changes of LV function. Bedside TTE showed that a severe LV dysfunction may last even shorter than usually believed in the setting of acute myocarditis or stress cardiomyopathy. In experienced hands, speckle tracking echocardiography may be useful to detect subtle changes in LV function [5]. However, echocardiography alone is usually insufficient to make the distinction between ischemic and non-ischemic causes of LV dysfunction, although it has recently been suggested that layer-specific strain may be useful for diagnosing an acute myocarditis [6]. In the present case, chest pain accompanied by STE and transient LV dysfunction were indicative of an AMI, acute myocarditis and atypical variant of stress cardiomyopathy. The diagnosis of an AMI was ruled out by normal MSCT coronary angiography. However, approximately 3% of patients with AMI may have normal coronary angiograms while unstable coronary lesions may be detected with coronary intravascular ultrasound [7]. Further, approximately 2% of patients initially presented as AMI are diagnosed with stress cardiomyopathy which commonly affects postmenopausal women and is characterized by transient apical ballooning in the absence of obstructive CAD. Our patient did not fit this paradigm, but it should be noted that patients of both sexes and all ages can be affected and various atypical forms of transient LV dysfunction have been reported [8]. Vasospastic (Prinzmetal) angina might also be considered in this patient since transient chest pain and LV dysfunction can also be due to coronary vasospasm that may occur in angiographically normal epicardial arteries and be triggered by stress conditions. However, the chest pain was atypical and not responsive to nitrates while provocative pharmacologic tests were not carried out. If cardiac MRI had been performed early after the onset of disease, it could have contributed to differential diagnosis. The most characteristic MRI feature of stress cardiomyopathy is myocardial edema that appears as high T2 signal intensity with a diffuse or transmural distribution. However, a few weeks after the onset of symptoms, the signal intensity decreases and, in many cases, it may be impossible to differentiate it from the normal ventricular wall [9]. In case of AMI, myocardial edema is located transmurally or subendocardially, follows vascular distribution and high signal intensity may persist for several months after the event [9]. Finally, in acute myocarditis, the distribution of myocardial edema is more heterogeneous, usually has a mid or subepicardial location and it has been reported to persist on MRI for an average of 111 days (ranging from 56 to 313 days) after the symptom onset [10]. Typical clinical presentations and diagnostic findings in patients presenting with chest pain and left ventricular dysfunction are summarized in Table 1. Mechanisms underlying transient LV dysfunction in various clinical scenarios, including stress cardiomyopathy, neurogenic stunning or myocarditis, are not fully understood. Since the area of edema corresponds to the area of wall motion abnormalities, it could be speculated that swift onset and recovery in some cases of myocarditis can be explained by the presence of myocardial edema and the absence of necrosis and fibrosis [11].

Given the main features of the disease (the ongoing respiratory infection, synchronous rise and fall of cardiac troponin, CRP levels, and normal MSCT coronary angiogram), the patient was suspected of having a myocarditis and responded well to supportive treatment including aspirin until CAD was ruled out. Importantly, although data from animal studies found an association of aspirin and increased mortality in myocarditis, this association was not confirmed in a recent prospective, multicenter study [12]. Prompt worsening and recovery of LV function in myocarditis is rare, but possible. In a study by Martin SS et al. [13], six out 24 patients who underwent echocardiography during pandemic influenza A (H1N1) in 2009, had a new-onset or worsening LV dysfunction. Improved or normalized ejection fraction was observed in four patients, within 4–22 days following hospitalization. We present this case to underline the value of multimodality cardiac imaging in patients with chest pain and ambiguous clinical presentations but also to raise awareness of a very short-lasting LV dysfunction that may complicate upper respiratory tract infections. Since the symptoms of respiratory infections may obscure those of LV dysfunction, myocardial dysfunction in these patients may go undetected. Its prevalence and clinical importance are yet to be determined.

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Supplementary material

VIDEO LEGENDS

Video 1. Two-dimensional echo loops recorded at hospital admission (quad view). Note inferoposterolateral wall hypokinesia with left ventricular ejection fraction of 50%.
To view Video 1, please go to: http://srpskiarhiv.rs/global/video/video1.dot

Video 2. Two-dimensional echo loops recorded at hospital day two, during the episode of chest pain (quad view). There is a global hypokinesia with intracavitary spontaneous echo contrast ("smoke" within the left ventricular cavity, best seen in the apical 2-chamber view – upper right panel).
To view Video 2, please go to: http://srpskiarhiv.rs/global/video/video2.dot

Video 3. Two-dimensional echo loops recorded at hospital day six (quad view). There is a complete recovery of left ventricular systolic function and the disappearance of spontaneous echo contrast within the left ventricle.
To view Video 3, please go to: http://srpskiarhiv.rs/global/video/video3.dot

Тешка, транзиторна дисфункција леве коморе удружена са респираторном инфекцијом

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САЖЕТАК

Увод С обзиром на то да се клиничке и електрокардиографске карактеристике различитих срчаних поремећаја могу преклапати, диференцијална дијагноза дисфункције леве коморе може бити тешка и за најискусније лекаре. Скорашњи напредак на пољу визуализационих метода срца може да помогне клиничарима да поставе тачну дијагнозу и започну адекватно лечење.

Циљ овог рада је да скрене пажњу на дисфункцију леве коморе врло кратког трајања током респираторних инфекција и да нагласи значај различитих визуализационих техника у овим клиничким околностима.

Приказ болесника Претходно здрав, 37-годишњи мушкарац јавио се лекару због атипичних болова у грудима и елевације СТ-сегмента у инферолатералним одводима током психичког стреса и акутне респираторне инфекције. Акутни инфаркт миокарда, миокардитис, коронарни вазоспазам и стрес-кардиомиопатија разматрани су као диференцијалне дијагнозе. Нагла појава тешке дисфункције леве коморе и потпун опоравак за четири дана регистровани су ехокардиографијом и даље евалуирани другим визуализационим техникама, укључујући вишеслојну компјутеризовану томографију и магнетну резонанцу срца.

Закључак Бројни узроци, укључујући респираторне инфекције горњих дисајних путева, могу бити окидач тешке али врло краткотрајне дисфункције леве коморе. С обзиром на то да симптоми респираторних инфекција могу да маскирају симптоме дисфункције леве коморе, дисфункција миокарда код ових болесника може проћи незапажено са могућим озбиљним последицама.

Кључне речи: бол у грудима, елевација СТ-сегмента, пролазна дисфункција леве коморе