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 multi-slice 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, non-radiating 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
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 echo contrast (SEC) (Figure 3A, Video 2). Two-dimensional speckle-tracking strain analysis showed the global longitudinal peak systolic strain (PSS) of -11.6%. 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).
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.

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.
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REFERENCES

1. Aurigemma GP, Tighe DA. Echocardiography and reversible left ventricular dysfunction. Am J Med. 2006;119(1):18–21.

2. Sosa S, Banchs J. Early recognition of apical ballooning syndrome by global longitudinal strain using speckle tracking imaging - the evil eye pattern, a case series. Echocardiography. 2015; 32(7):1184–92.

3. Peulit DP, Petrovic ZM, Vasic NR, Stevic RS. Synchronous advanced pulmonary tuberculosis and acute virus myocarditis mimicked Wegener granulomatosis in a 26-year-old man – Case report. Srp Arh Celok Lek. 2016; 144(11-12):645–9.

4. Simić D, Milojević I, Budić I, Stojadinović V. Myocarditis Exacerbation in a Child Undergoing Inguinal Hernioplasty after Viral Infection. Srp Arh Celok Lek. 2009; 137(9-10):537–9.

5. Stanković I, Čvorović V, Putniković B, Vuksanović I, Panić M. Acute myocarditis diagnosed by layer-specific 2D longitudinal speckle tracking analysis. Echocardiography. 2016; 33(1):157–8.

6. Reynolds HR, Srivastava DS, Slater JN, Mancini GB, Fett F, et al. Mechanisms of myocardial infarction in women without angiographically obstructive coronary artery disease. Circulation. 2011; 124(13):1414–25.

7. Čvorović V, Stanković I, Panić M, Živković A, Nešković AN, et al. Establishing the diagnosis of inverted variant angina pectoris. PloS One. 2012; 7(9):e45366.

8. Nešković AN, et al. Two-dimensional echo loops recorded at hospital admission. Video 1.

9. Nešković AN, et al. Two-dimensional echo loops recorded at hospital admission. Video 2.

10. Nešković AN, et al. Two-dimensional echo loops recorded at hospital day six. Video 3.

Supplementary material

VIDEO LEGENDS

Supplementary material

Glossary