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Cardiogenic shock presenting in myocardial infarction with myocarditis case report: The role of advanced echocardiography parameter

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Abstract

Myocarditis is distinguished by a wide array of nonspecific symptoms, including chest pain, dyspnea, and palpitations. These symptoms are accompanied by electrocardiographic abnormalities that exhibit similarities to those observed in myocardial infarction. However, the results of coronary angiography frequently, though not consistently, show normal findings. Therefore, the clinical diagnostic procedure often encounters difficulties and is susceptible to the misdiagnosis of myocardial infarction with nonobstructive coronary arteries. The signs of poor cardiac contractility are a common manifestation of myocarditis and can be evaluate with bedside echocardiography. Two-dimensional speckle tracking echocardiography bestows a precise left ventricle (LV) global and regional dysfunction . We present a case of a 40-year-old man with typical chest pain for 8 hours, and dyspnea. He had no significant medical history. This patient was first diagnosed with high lateral ST-elevation myocardial infarction (STEMI) with cardiogenic shock. Angiography examination revealed no significant obstruction of coronary vessels. However, serial left ventricle global longitudinal strain supports the diagnosis of myocarditis. After receiving the treatment for myocarditis, the patient makes a full recovery within 7 days.

Keywords

Myocarditis, MINOCA, echocardiography

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Introduction

In the context of emergency medicine, it is sensible for physicians to consider the possibility of an acute coronary syndrome when there is evidence of myocardial injury accompanied by characteristic chest pain. If subepicardial coronary artery disease is excluded, it is imperative to explore alternative factors contributing to myocardial infarction with nonobstructive coronary arteries (MINOCA). The presence of myocarditis with a pseudo-infarct presentation can be considered as a potential differential diagnosis for MINOCA.[1] Myocarditis has been previously identified in 34.5% of patients who were initially misdiagnosed with MINOCA. Poor cardiac contractility is a common manifestation of myocarditis and can be measured with bedside echocardiography.[2] Two-dimensional speckle tracking echocardiography (2DSTE) provides an accurate LV global and regional dysfunction.[3] We presented a 40-year-old man with initial diagnosis of high lateral STEMI with cardiogenic shock and total atrioventricular block. However, serial evaluation supports the diagnosis of myocarditis.

Case presentation

A 40-year-old man referred from a rural hospital came with a sudden and persistent typical chest pain for 8 h, dyspnea,

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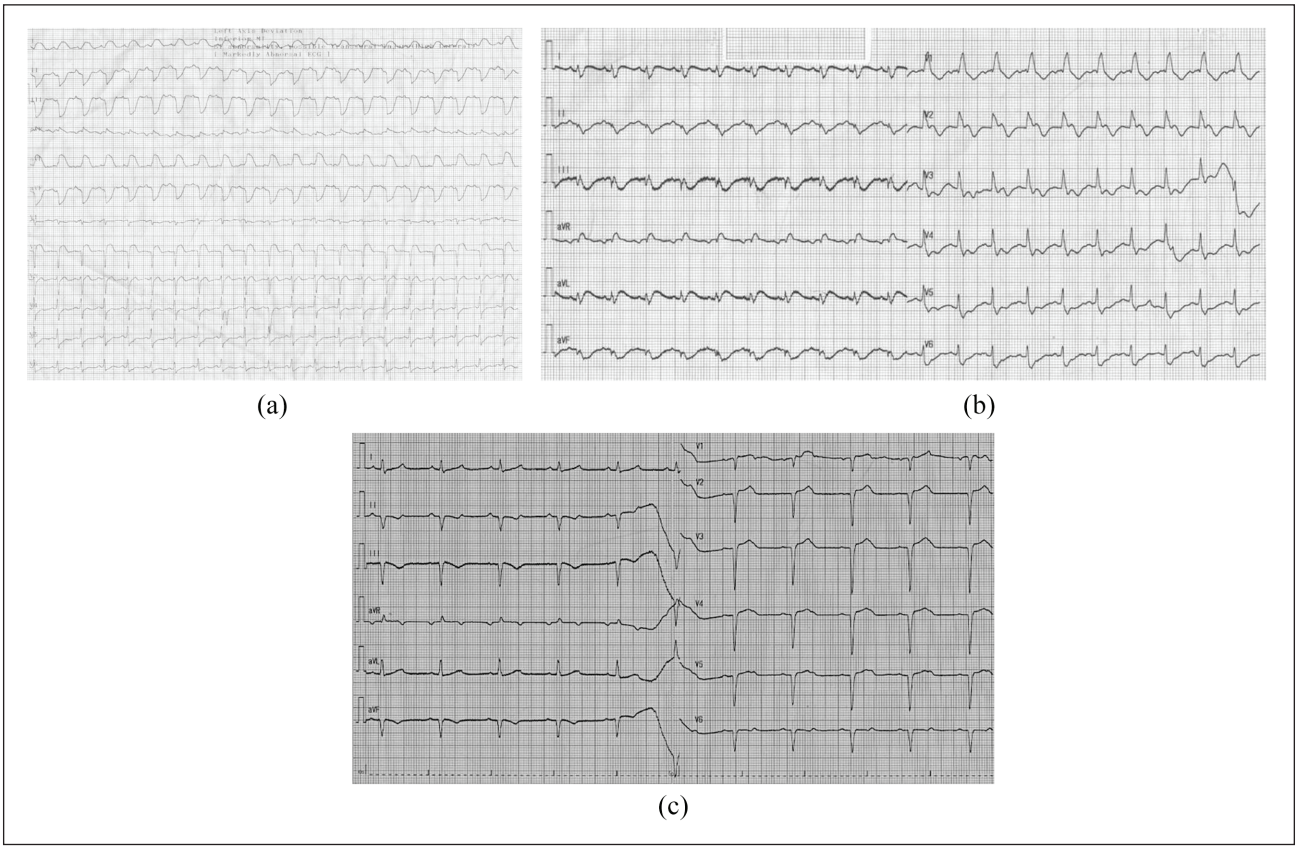

Figure 1. The ECG changes during admission. (a) The ECG showed ST-segment elevation in lead I, aVL on the admission day. (b) After 1 day, a new onset of complete right bundle branch block was revealed. (c) An abrupt high-degree atrioventricular block with a ratio of 2:1 was revealed on day 3 of admission.

nausea, and vomiting. He had a fever for 3 days before being admitted. He was an active smoker with no other cardiovascular risk factors and no history of cardiovascular disease. His initial blood pressure was 118/78 mmHg, pulse was 123 beats per min, and respiratory rate was 20 times per min. An electrocardiogram (ECG) revealed ST-segment elevation in lead I, aVL (Figure 1(a)). His first laboratory tests revealed increased levels of troponin-T at 31.38 ng/mL [normal level $<$0.1 ng/mL]. Initially diagnosed with high lateral STEMI, alteplase was given. Chest pain diminished, but an ECG revealed a new-onset complete right branch bundle block (Figure 1(b)). A regional wall motion abnormality evaluation revealed a hypokinetic segment at the anterolateral with preserved left ventricle ejection fraction (LVEF 55%), confirmed by the depressed global longitudinal strain (GLS) parameters at the basal, middle, and apical segments of the anterolateral wall (0%, 0%, −2%, respectively) (Figure 2(a)). The patient was transferred to an intensive care unit and scheduled for rescue percutaneous coronary intervention while waiting for the swab result to exclude coronavirus infection.

Following a period of hospitalization in the intensive care unit, the patient exhibited hemodynamic instability with a blood pressure of 74/50 mmHg, pulse was 120 beats per min, cold extremities, and urine output was less than 0.5 ml/kgs per hour. Vasoactive and inotropic agents were administered until an optimal dose (Table 1), but the hemodynamic parameters still deteriorated. On the third day of admission, an ECG showed a high-grade atrioventricular block with a ratio of 2:1 (Figure 1(c)), which then changed into a total atrioventricular block. The hemodynamic parameters revealed a left ventricular outflow tract velocity time integral (LVOT VTI) of 10.5 cm, a cardiac index of 1.07 L/min/ml^2, and a cardiac output of 2.02 L/min with mildly reduced EF (LVEF 37%) (Table 1), and depressed GLS was noted at the basal, middle, and apical region of lateral, anteroseptal, septal, and inferior walls (Figure 2(b)). The extended area of infarction was suspected. An intra-aortic balloon pump (IABP) insertion, a coronary angiography procedure, and temporary pacemaker placement were done simultaneously. An angiogram showed normal TIMI-3 flow in coronary vessels.

The patient was diagnosed with MINOCA with differential diagnosis myocarditis. The diagnosis was strengthened by improvement symptoms after 3 days of supportive treatment and anti-inflammatory colchicine, which converted into sinus rhythm, improved LVEF (44%), and improved wall contractility was confirmed by GLS (Figure 2(c)). The

Table 1. Monitoring patient during hospitalization.

Supporting findings	Day 0	Day 1	Day 2	Day 3	Day 4	Day 6
ECG	Sinus rhythm with ST-segment elevation in lead I, aVL	Sinus rhythm with ST-segment elevation in lead I, aVL; new-onset complete RBBB	Sinus rhythm with complete RBBB	Sinus rhythm with high-degree AV Block (ratio of 2:1)	Sinus rhythm with PVC occasional	Sinus rhythm
Troponin-I (ng/dL)	31.38					
RWMA	Hypokinetic segment at the anterolateral					
GLS		Depressed at the basal, middle, and apical segment of the anterolateral wall (0%, 0%, and −2%, respectively)		Depressed GLS was noted at the basal, middle, and apical region of lateral, anteroseptal, septal, and inferior walls (0%)		Improved GLS at the basal, middle, and apical region of lateral, anteroseptal, septal, and inferior walls (−5% until −7%)
Est RAP (mmHg)	10	10	15	15	10	10
LVCI (L/min^2)	2.9	2.56	2.2	1.07	1.9	2.4
LVOT VTI (cm)	15.6	15.3	13.2	10.5	14.8	15
SVR (dynes.sec/cm^2)	729.8	1130	1206.3	2000	1555	1201
Intervention	Fibrinolysis	(1) Pump NE 50 nano/kg/min, up titration until 150 nano/kg/min mcg/min. (2) Pump Dobutamine 5 mcg/kg/min.	(1) Pump NE 150 nano/kg/min (2) Pump Dobutamine 10 mcg/kg/min.	(1) Pump NE 100 nano/kg/min. (2) Pump Dobutamine 7 mcg/kg/min. (3) IABP insertion and TPM implantation.	(1) Pump NE 100 nano/kg/min. (2) Pump Dobutamine 5 mcg/kg/min. (3) IABP support (1:1).	(1) Pump Dobutamine 3 mcg/kg/min, then tapered off on day 7.

ECG, electrocardiogram; RBBB, right bundle branch block; AV, atrioventricular; PVC, premature ventricular complex; RWMA, regional wall motion abnormality; GLS, global longitudinal strain; RAP, right atrial pressure; LVOT VTI, left ventricle outflow tract velocity time integral; LVCI, left ventricle cardiac index; SVR, systemic vascular resistance; TPM, temporary pacemaker; NE, norepinephrine.

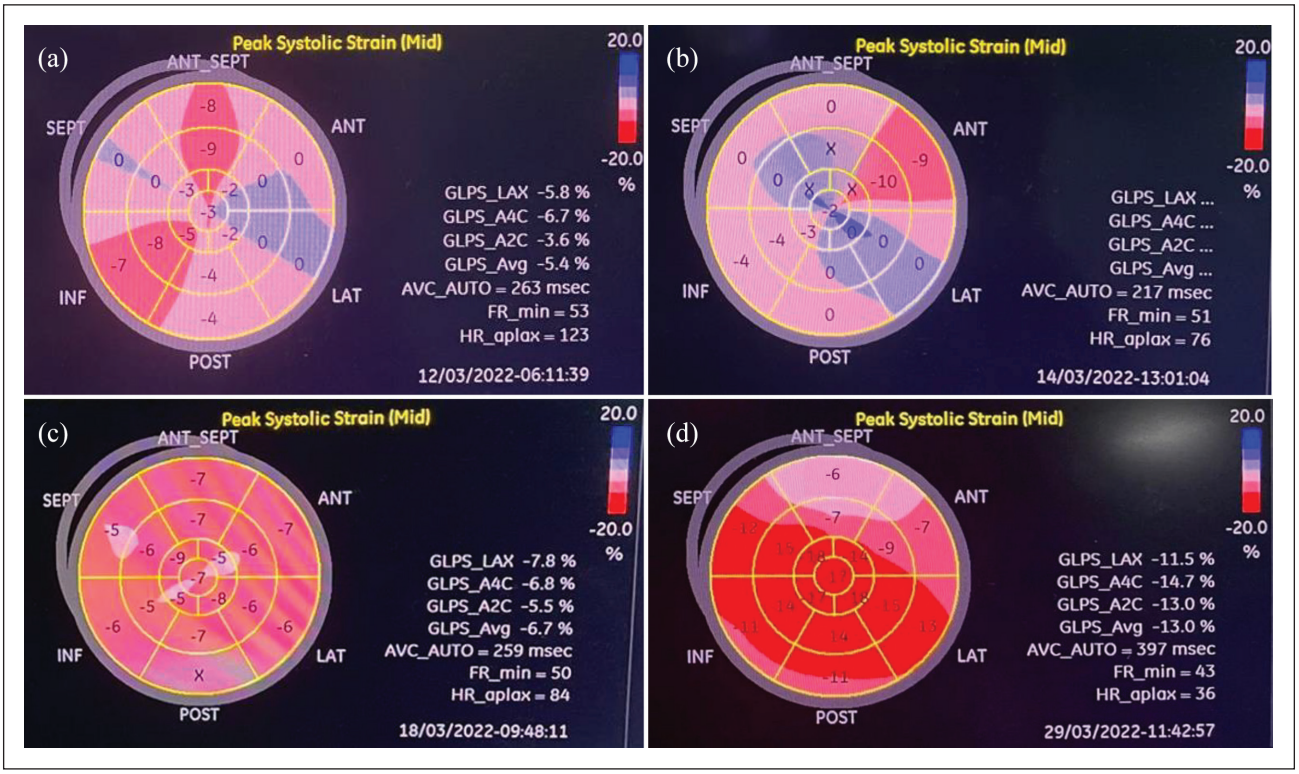

Figure 2. (a) The decreased global longitudinal strain (GLS) value at the basal, middle, and apical segment of anterolateral wall (0%, 0%, −2%, respectively) on day 1 of admission. (b) The worsening GLS revealed at basal, middle, and apical segment of lateral, septal, and inferior wall on day 3 of admission. (c) Improvement of the left ventricle contractility measured by GLS. (d) Improved GLS evaluated in the outpatient ward.

vasoactive agents were reduced gradually and then IABP was removed on the sixth day of admission. The hemodynamic improved on day 7 of hospitalization without supportive agents, then an anti-remodeling angiotensin-converting enzyme (ACE) inhibitor, beta-blockers, and aldosterone receptor antagonists (MRAs) were initiated and gradually titrated. The patient was discharged on the ninth day of hospitalization. The LV GLS was evaluated in the outpatient ward, it showed improvement at the basal, middle, and apical regions of the lateral, posterior, inferior, and septal walls (Figure 2(d)).

Discussion

Symptoms of acute myocardial infarction (MI) in young people with nonobstructive coronary angiograms should be suspected with myocarditis.[4] Early recognition of clinically suspected myocarditis requires at the minimum one of the following clinical presentations and at least one diagnostic criteria; the asymptomatic patient requires at least two diagnostic criteria. Clinical presentations of myocarditis are chest pain, new-onset or worsening heart failure, palpitations, unexplained sinus tachycardia, respiratory distress, and unexplained cardiogenic shock. Diagnostic criteria related to myocarditis, including (1) ECG/Holter stress features: new-onset AV block, new-onset ST/T wave change, atrial or ventricular arrhythmia, new-onset bundle branch block; (2) Elevated troponin-T or troponin-I; (3) Functional and structural abnormalities on cardiac imaging: new or unexplained abnormality of LV and/or right ventricle (RV) function showed by regional wall motion abnormality, global systolic, or diastolic dysfunction; and (4) Tissue characterization by cardiac magnetic resonance (CMR) suggests myocarditis.[5]

Endomyocardial biopsy (EMB) remains the gold standard to diagnose myocarditis, but its invasive procedure can increase complications and is not preferable in acute care. CMR seems the most accurate noninvasive modality, but its availability is still limited. Conventional two-dimensional (2D) echocardiography can play an important role in identifying myocarditis. However, it plays a limited role in evaluating LV performance in myocarditis with preserved LVEF. Speckle tracking echocardiography (STE) seems promising to evaluate LV global and regional functions.[3,4] Left ventricular GLS may represent a sensitive parameter to detect subtle myocardial abnormalities in acute myocarditis with normal or subnormal EF. Early STE on admission can be helpful to stratify the risk; therefore, patients with a high risk of poor outcome can be given more aggressive cardioprotective treatments.[6] Myocarditis predominantly involves the subepicardial and intramyocardial regions. Left ventricular

function remains preserved when the involvement of the myocardium is limited, which is difficult to be recognized by basic echocardiography parameters. A damage of the GLS of the lateral segments has great sensitivity and specificity in diagnosing myocarditis.[7]

In our patient, he had signs and symptoms of clinically suspected myocarditis with acute chest pain, new onset of heart failure, unexplained cardiogenic shock, and supported by the findings of abrupt high-degree AV block with a ratio of 2:1 then changed into total AV block, new onset of complete right bundle branch block, and elevated troponin-T value. Structural abnormalities showed regional wall motion abnormalities at the anterolateral segment, consistent with decreased GLS at the basal, middle, and apical segment of the anterolateral wall; raised the probability of myocarditis. The LV GLS was impaired at the onset of the symptoms, then improved, and normalized along with clinical improvement. It is important to highlight that in settings where access to CMR and EMB is limited, this advanced echocardiography parameter is unable to independently diagnose myocarditis. The integration of LV GLS data and relevant clinical findings can be used to early diagnose and determine prompt treatment for suspected myocarditis. The assessment using GLS is subject to certain limitations, primarily reliant on the availability of high-quality images to ensure reliable evaluations. Moreover, the absence of standardized cutoff values for pathology normality poses challenges in objectively assessing it within clinical settings, yet it also presents opportunities for further investigation.[8] In addition to being grounded in clinical manifestations associated with myocarditis, diagnostic confirmation should rely on the utilization of electrocardiography and markers of myocardiocytolysis. The level of suspicion increases proportionally with the number of diagnostic criteria that are satisfied.

Conclusion

In summary, 2DSTE, especially the GLS parameter, can be helpful in early diagnosis, control of myocarditis treatment, and predict unfavorable outcomes in myocarditis. LV GLS parameter is a promising alternative tool in the acute care setting or when standard tools are unavailable.

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None.

Author contributions

M.Y.A. contributed to participation in the conception, drafting, approval of the article, and acceptance of responsibility for all aspects of the work; P.D.K. D contributed to the acquisition and collection of data, the drafting, writing, and reviewing of the work, and accepted responsibility for all aspects of the work, C.P.B. participated in conception, drafting, article approval, and acceptance of responsibility for all aspects of the work.H.S. contributed to the design of the work, revises the work, approves of the work, and agrees to be responsible for all aspects of the work.

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