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CASE REPORT

# Post-MI free wall rupture syndrome. Case report, literature review, and new terminology

Jingjin Che, Guangping Li, Kangyin Chen & Tong Liu

Department of Cardiology, Tianjin Institute of Cardiology, 2nd Hospital of Tianjin Medical University, Tianjin, China

**Correspondence**
Jingjin Che, Department of Cardiology, Tianjin Institute of Cardiology, 2nd Hospital of Tianjin Medical University, Tianjin, China.
Tel: 86-22-88326759; Fax: 86-22-28261158;
E-mail: jingjinche@aliyun.com

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### Key Clinical Message

Common clinical features of subacute rupture left ventricular free wall after acute ST segment elevation myocardial infarction are: (1) recurrent or persistent chest pain; (2) recurrent or persistent ST segment elevation; (3) hypotension. Integrating these signs into a syndrome can increase the clinician's awareness to the fatal complication.

### Keywords

acute ST segment elevation myocardial infarction, creatine kinase MB, pericardiocentesis, subacute free wall rupture of left ventricle, troponin.

## Introduction

As an important and rare complication of acute ST-segment elevation myocardial infarction (STEMI), free wall rupture is under-recognized [1]. Sometimes it can be subacute and may not be typical of an acute blowout rupture leading to death in few minutes [1–4]. We recently encountered three patients with subacute free wall rupture after STEMI. The first two patients experienced a relatively long period from rupture to death, and had some similar and characteristic presentations: recurrent or persistent chest pain and ST-segment elevation, and hypotension. All these characters can be explained by small rupture of free wall with slow bleeding, and have not been reported extensively. Thus, we summarize it as subacute free-wall rupture syndrome. Fortunately, we applied the conception to the following third patient with similar clinical condition, and salvaged him successfully.

## Case Presentation

### Case 1

A 40-year-old man was in his normal condition until awakened by a sudden squeezing chest pain 5 h prior to admission to our hospital. The pain progressively exacerbated with mild dyspnea. He had no hypertension or diabetes mellitus. Electrocardiograph (ECG) at 5 h after the onset showed profound ST-segment elevation in the leads I, aVL, and $V_1$–$V_6$ (Fig. 1A). Then, the patient was admitted to our hospital, and a loading dose of dual antiplatelet agents (aspirin 300 mg and clopidogrel 600 mg) were administered immediately. He underwent coronary angiography (CAG) at the 6th h after the onset. CAG showed a total occlusion at the proximal left descending artery (LAD). The vessel was reopened by ballooning, and a drug-eluting stent (DES) was implanted in the proximal LAD, shown in Figure 2. As a final result, the blood flow of LAD was of TIMI Grade 3. Intravenous tirofiban was given continuously at a speed of 0.1 μg/kg/min after PCI. His chest pain was partially relieved with partial ST-segment regression (Fig. 1B). However, 24 h after the procedure, his chest pain aggravated again with recurrent ST-segment elevation in pericardial leads, shown as Figure 1C. The pain was located in the same area, but sharper than before, and occasionally sharpened by deep inspiration with mild dyspnea, and his blood pressure (BP) ranged from 100 to 125/60 to 70 mmHg. Then, subacute stent thrombosis was suspected, but he and his family refused further study by CAG. Then, he was treated with intensive antithrombotics, including aspirin, clopidogrel, and prolonged

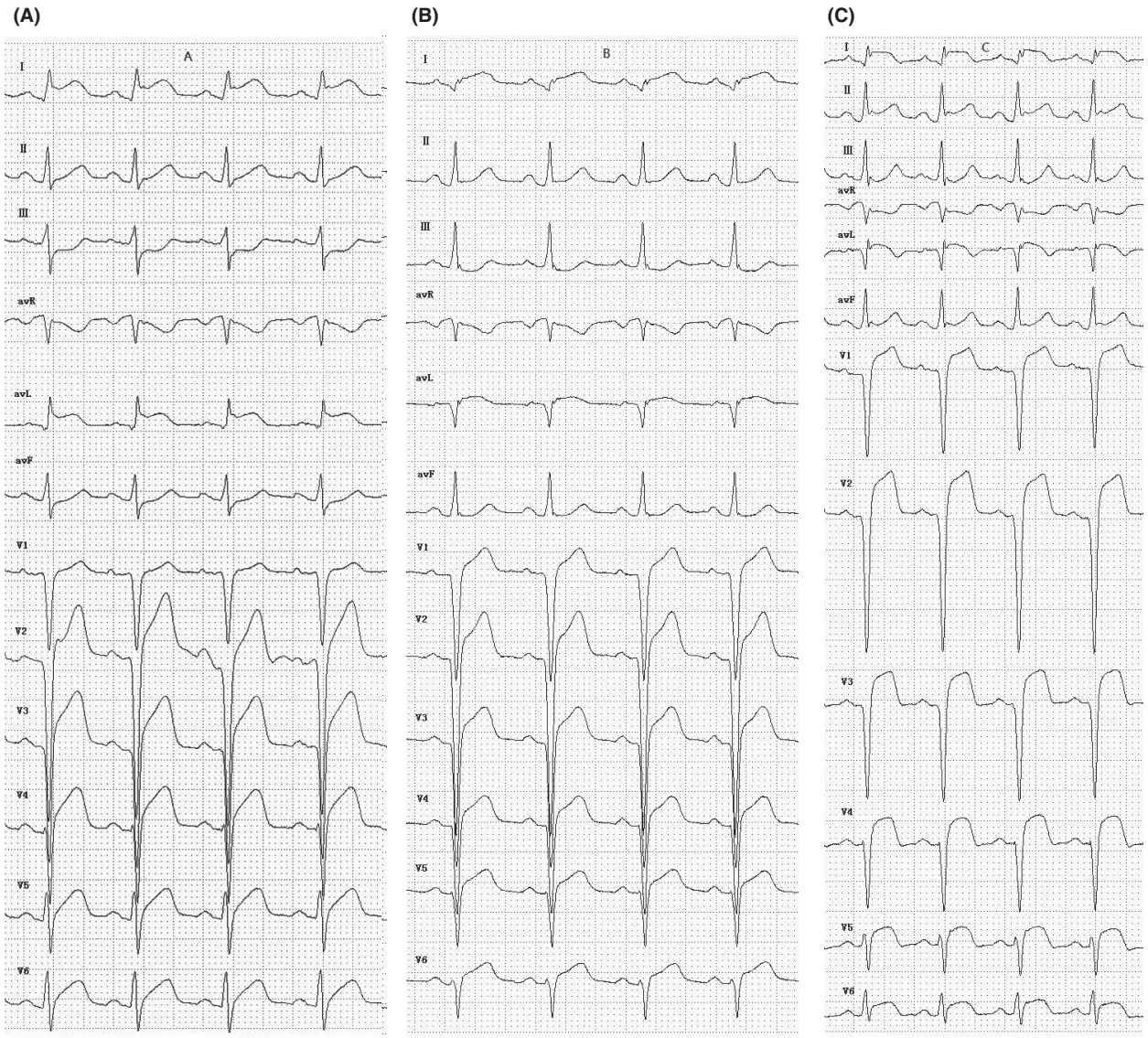


**Figure 1.** A series of ECGs of Case 1, a 40-year-old man on account of chest pain for 5 h. (A) ECG before primary PCI showed ST-segment elevation of 2–4 mm in pericardial leads. (B) ECG immediately after primary PCI showed a partial resolution of ST-segment elevation. (C) ECG 24-h after primary PCI showed a recurrent ST-segment elevation in pericardial leads.

tirofiban. Sixty hours after PCI, his condition worsened with severe dyspnea, sweating, and hypotension. Ultrasound cardiogram (UCG) showed dense pericardial effusion of 15–24 mm thickness. Then, pericardiocentesis was performed immediately. His condition got stable after 170 mL blood effusion was drained out. All the antithrombotics were discontinued except clopidogrel, considering the implanted stent at proximal LAD. The drainage was 420 mL in the first day, total 580 mL in the following second day, but on the fourth day increased to 740 mL and hemoglobin dropped from the normal to 81 g/L. His BP dropped further, and then he was transferred to Department of Cardiac Surgery, and exploratory pericardiotomy was performed. During the operation, no definite crevasse was found, but blood clot on the surface of his heart and a slow bleeding area on the lateral wall of LV, were repaired by patch suture technique. But the patient finally died from cerebral complication 2 days after the operation.

In this case, when recurrent chest pain with ST-segment elevation reoccurred to him 24 h after primary PCI, culprit artery reocclusion due to intrastent thrombosis was thought to be the unique cause. But there could be no culprit artery reocclusion suggested by no second peak

 

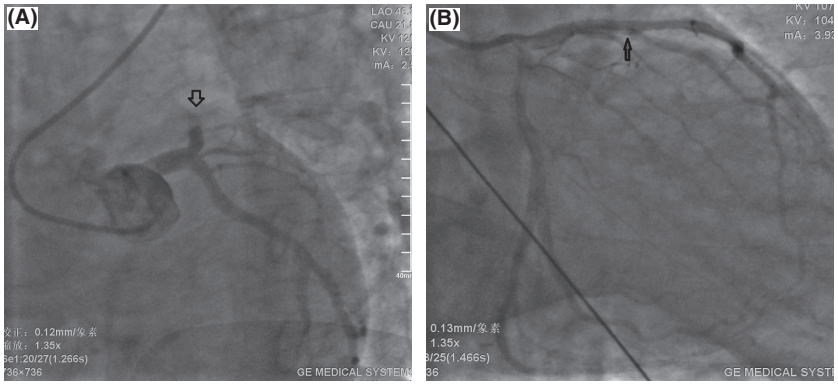


**Figure 2.** Coronary angiography of Case 1 before and after primary PCI. (A) The arrow indicated the position of the total occlusion at proximal LAD before PCI on the spider view. (B) The occluded LAD was reperfused after a drug-eluting stent was implanted at the proximal of LAD. The arrow indicated position of the stent.

of creatine kinase MB (CK-MB) on continuous monitoring. Till his BP dropped, acute cardiac tamponade caused by free wall rupture was taken into account, and confirmed by bedside UCG.

## Case 2

A 69-year-old man was admitted due to abrupt chest pain for 3 h. He had histories of hypertension, stroke, and digestive ulcer, but no diabetes. PE: clear mentality, supine, BP 75/45 mmHg, no rales in both lungs, HR 118 bpm, no edema. EKG showed sinus tachycardia, ST segments of I,II, avL, and $V_5$–$V_9$ elevation of 1–3 mm, QS waves in leads of II,III, $V_6$–$V_9$ (Fig. 3). Lab testing: D-dimer 1.2 mg/L, white blood cell 31.9 × $10^9$/L, hemoglobin 150 g/L, cardiac troponin I (cTnI) 7.5 ng/mL, CK-MB 38.7 ng/mL. So he was diagnosed as acute inferior–lateral MI. After preparation with loading doses of dual antiplatelet agents, primary PCI was performed. CAG showed that the left circumflex (LCX) was completely occluded (Fig. 4A). The LCX was reopened, and a DES was implanted (Fig. 4B). After the procedure, his chest pain was partially relieved. But hypotension persisted with tachycardia and shortness of breath. Four hours after PCI, the patient suddenly lost his consciousness with electromechanical dissociation, and the subsequent resuscitation was ineffective. Uncoagulated blood was sampled from his pericardial cavity during resuscitation.

After review of the case in detail, several lines of clues to the cause of his death were found. Firstly, the patient had 3 h chest pain, but the level of cTnI (7.5 ng/mL) on admission was much higher than the level expected. Secondly, he had persistent hypotension even treated by supplementation of normal saline, and successful reperfusion. Finally, on review of the CAG, "differential density sign", a manifestation of pericardial effusion, was detected on the images on caudal 35° projection before PCI (Fig. 4A). Therefore, his death was attributed to free wall rupture after STEMI. Additionally, plateau-shaped ST elevation was impressing in this case.

## Case 3

A 78-year-old man was admitted because of chest discomfort for 3 days, and sudden chest pain with dyspnea and dizziness for 1 h. He had a history of hypertension, no diabetes. PE: BP 80/40 mmHg, blurred mentality, no rales in lungs, HR 54 bpm, irregular, no edema, no pathological reflex. ECG (Fig. 5) showed sinus tachycardia with Wenckebach atrioventricular block, ST-segment elevation in leads II, III, avF, and $V_7$–$V_9$ with inverted T waves. Testing: cTnI 9.94 ng/mL, CK-MB 31.5 U/L, D-dimer 0.1 mg/dL.

According to his symptom, STEMI was presumed to occur 1 h before admission, but pathological Q wave with the inverted T waves suggested that the duration of the infarction might be longer than 1 h; furthermore, his cTnI was extremely high, therefore, 3 days' duration of STEMI was more likely. But why did he get worsened? According to our previous experiences, he had chest pain mismatched with the elevation of cTnI, persistent ST elevation, and unexplainable hypotension. Cardiac tamponade caused by LV free wall rupture was a major differential diagnosis. As bedside UCG was not available immediately, he was sent to catheter room. Chest X-ray testing was planed, and CAG was standby. The chest X-ray showed an overt "differential density sign" (Video S1). Therefore, the diagnosis of free wall rupture was established, and CAG was canceled. Emergent UCG demonstrated the presence of pericardial effusion of 10–20 mm in thickness. Pericardial puncture was performed immediately through subxiphoid approach under UCG

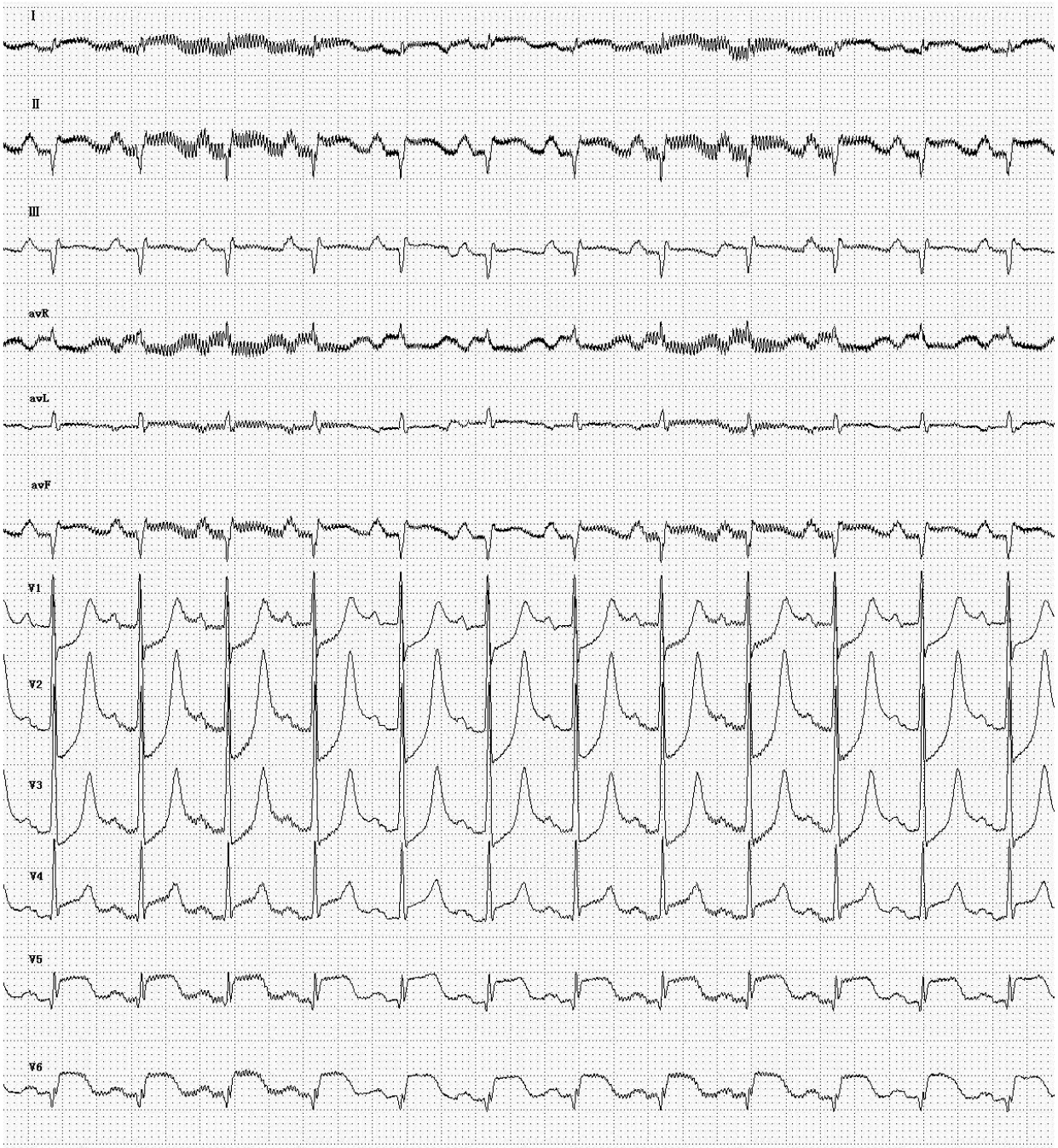


**Figure 3.** ECG of Case 2, a 69-year-old man admitted on account of abrupt chest pain for 3 h. ECG showed that ST segments of I,II, III, avF, avL, and $V_5$–$V_6$ elevated 0.5–3 mm, and Q waves formed in leads of II,III, avF, and $V_5$–$V_6$.

guidance. After 150 mL of blood effusion was drained out, his BP increased to the normal, his consciousness became clear. All antithrombolic agents were discontinued. Within the first day, 320 mL blood effusion was drained from his pericardial cavity. In the following 3 days, the drainage was 80, 40, and 20 mL, respectively, and disappeared then. The draining tube was removed on the 11th hospital day, when UCG demonstrated no pericardial effusion. Then, he was discharged from our hospital uneventfully.

The patient accepted dual antiplatelet agents till 1 month later when absence of pericardial effusion was

 

proven by another UCG. He was readmitted 6 weeks after first hospitalization to evaluate his coronary arteries. His right coronary artery (RCA) was totally occluded, and reopened by ballooning. Two DES were implanted at the proximal and the distal of RCA, respectively. His condition kept well in the following two years.

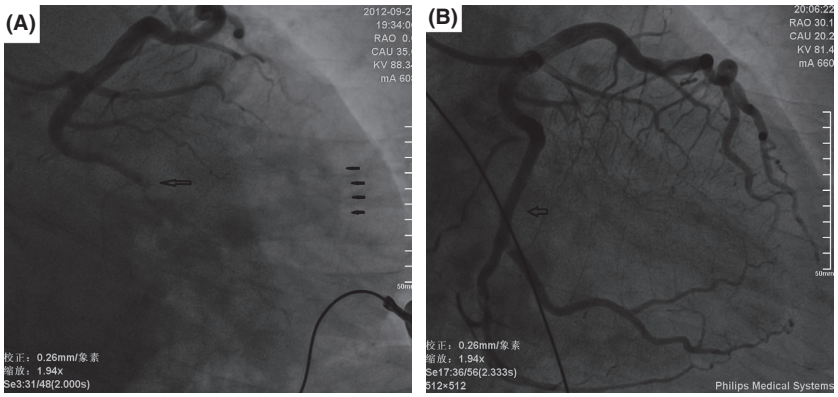


**Figure 4.** Left coronary angiography of Case 2 before and after primary PCI. (A) The hollow arrow indicated the position of total occlusion at mid LCX before PCI on the caudal 35°. The four solid arrows show the "fat-pad sign". (B) The occluded LCX was reperfused after a drug-eluting stent was implanted. The arrow indicated position of the stent.

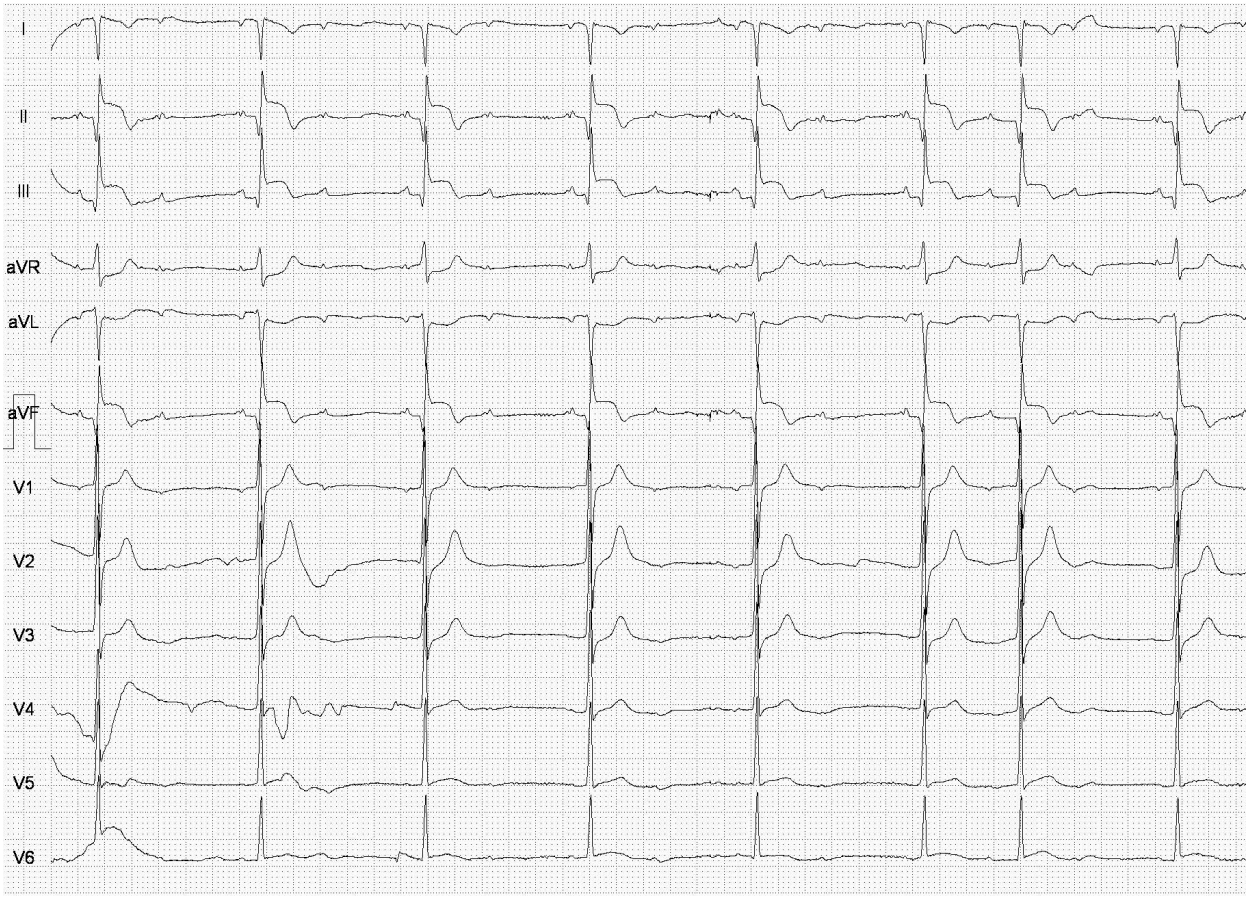


**Figure 5.** ECG of Case 3, a 78-year-old man admitted because of chest discomfort for 3 days, and sudden chest pain with dyspnea and dizziness for 1 h. ECG on admission showed that sinus rhythm with Wenckebach atrioventricular block, pathologic Q waves, and ST segments elevation in leads II, III, and avF with inverted T waves. *Note:* left arm/right arm leads were reversal.

## Discussion

In 1987, Perdigão et al. [5]. classified the anatomical characters of free wall rupture into four types according to 42-case autopsy findings: type I-direct rupture; type II-multicanalicular rupture; type III-rupture protected by an intraventricular thrombus or a pericardial symphysis; type IV-incomplete epicardial, endocardial, or intramyocardial rupture. Perdigão et al. [6] used the classification in their study on 51 cases of free wall rupture after STEMI. The clinical forms of presentation of free wall rupture, reported by them, were the following: syncope followed by death (60%), shock (21%), transitory syncope (4%), and psychomotor troubling (4%) [6]. It was shown that the clinical presentations are associated with the anatomical type of the rupture, sudden death tends to occur in cases with direct rupture without any protection, which is accepted as acute rupture. As for patients with free wall rupture resulting in slow bleeding, they could survive for a relatively long time, ranging from several hours to days. We prefer to classify this situation as subacute rupture. These three cases with subacute rupture of LV free wall after STEMI shared common clinical features: (1) recurrent or persistent chest pain, typically pericarditic without a second peak of CK-MB; (2) recurrent or persistent elevation of ST segments in leads corresponding to infarcted zone, or occasionally extending to adjacent zone; (3) unexplainable hypotension. We term these clinical presentations as subacute free-wall rupture syndrome.

Firstly, the chest pain in this syndrome is either recurrent or continuous from onset of STEMI. Perdigão et al. [6] also noted that pain persistence or recurrence occurred in 63% of the patients with free wall rupture. If the chest pain is recurrent, it is prone to be confused with the ischemic chest pain caused by culprit artery reocclusion, because location of the pain tends to be as same as that at onset of STEMI. However, the pain from free wall rupture is more sharper in quality, and more or less related to inspiration, which mimics the chest pain of pericarditis. Inflammation on the visceral pericardium corresponding to infarcted zone, together with stimulation of the blood in pericardium might be the causes of the pain. But if the patient experiences a silent STEMI, the pain caused by rupture is prone to be confused with new onset of STEMI, like the patient of Case 2. It is really a big pitfall for clinicians, because any antithrombotic, especially anticoagulants, can worsen the hemorrhage. If the pain is accompanied by hypotension and blurred mentality, it is impossible for the patient to describe the pain in detail. Under this situation, however, the chest pain is often mismatched with ECG evolution, and levels of serum myocardial biomarkers.

Pathologic Q waves are generally representative of the irreversible myocardial necrosis. The Q waves may emerge within the first hour of infarction, but most commonly develop 8–12 h into infarction. Very early deep pathologic Q waves, for example Case 3, may signify a previous silent myocardial infarction (MI) with pericardial pain as its first manifestation [7].

The values of troponins remain normal or mildly higher in most patients with acute cardiac events as long as 6 h after symptom onset [8, 9]. The level of CK-MB within 6 h after onset of pain was reported ranging from 1.1 to 6.7 ng/mL, whereas troponin I from 0.4 to 1.2 ng/mL [10]. Therefore, as Case 2 and Case 3, it would be mismatched that patient with chest pain of onset within 3 h had relatively high levels of CK-MB and troponin.

ECG is most important laboratory test for diagnosis of acute pericarditis, and is also important to diagnose cardiac rupture after STEMI. Characteristic ECG change in free wall rupture is persistent or recurrent ST-segment elevation, mainly located in the infarcted zone, occasionally spreading to all leads, but avR as reported by Raposo [4]. Varbella et al. [11] also reported some ECG features of heart rupture, such as persistent ST-segment elevation with T waves failing to invert in the same leads. In some cases, new saddle-shaped ST-segment elevation may be the chief clinical manifestations [4]. Secondary pericarditis surrounding the infarct zone and local ventricular wall expansion may be possible contributors to the ST elevation.

Generally, persistent occlusion of infarct-related artery, or no effective myocardial reperfusion is most likely cause of unresolved ST elevation. Additionally, there are still two other important complications of STEMI manifested by persistent or recurrent ST elevation, which need to be differentiated from free wall rupture. Firstly, the majority of cases presenting with recurrent ST elevation after STEMI are caused by reocclusion of culprit artery [12]. Noninterventional measure to make sure the reocclusion is a second peak of CK-MB detected by continuous monitoring of serum CK-MB. Angiography is a unique method for definite diagnosis, and determination of subsequent therapy. Persistent ST elevation in infarcted zone was also observed in patients with left ventricular aneurysm [13]. Clinical presentations of LV aneurysm are often symptoms of heart failure with absence of chest pain and a second peak of CK-MB. Outward bulging of the ventricular wall during systole occasionally with mural thrombus confirmed by UCG is necessary to the diagnosis.

Hypotension is not necessary to diagnose rupture of LV free wall [3]. If the amount of hemorrhage is not enough to cause hemodynamic compromise, BP may keep in the normal range. On the other hand, for patients with STEMI, hypotension may also result from infarction

 

of right ventricle, severe LV dysfunction, or even dehydration. If hypotension cannot be explained by the known conditions, cardiac tamponade must be taken into account.

López-Sendón et al. [2]. reported that the incidence of persistent or recurrent chest pain, hypotension, ECG changes, was 63.5%, 94%, 40%, respectively. Thus, their sensitivity or specificity to diagnosis is not satisfied. But when all the signs are integrated, the balance of sensitivity and false positive diagnosis can be improved. Therefore, we strongly recommended that integrating all the signs into a syndrome to increase the clinician's awareness to the fatal complication.

Bedside UCG is the most important measure to establish the diagnosis of free wall rupture. The pericardium does not allow sudden increases in the volume without a marked increase in the intrapericardial pressure, thus, a sudden increase in pericardial volume of 100–200 mL, as in hemopericardium, may elevate pericardial pressure till 20–30 mmHg with acute cardiac tamponade [14]. In Case 1 and Case 3, the volume of first drainages was 170 and 150 mL, respectively.

But in most hospitals, UCG is not a routine test before primary PCI. Under this situation, cardiac silhouette under X-ray is an alternative way to detect pericardial effusion. "Fat-pad sign" is resulted from separation of retrosternal from epicardial fat line, with low sensitivity but high specificity to pericardial effusion [15]. "Differential density sign", similar to "fat-pad sign", is increase in lucency at heart margin caused by slight difference in contrast between myocardium, epicardial fat, and pericardial fluid. Additionally, diminished pulsation of the outer contour of pericardium with heart can also be observed under fluoroscopy.

Increasing cases were survived from the fatal complication by successful surgical or conservative therapy [2–4, 11, 16, 17]. After review of total 120 papers, Nasir et al. [16] concluded that surgery is superior to conservative management for patients presenting with free wall rupture. Varbella et al. [11]. believed that surgical intervention is mandatory, once hemorrhagic fluid is confirmed by pericardiocentesis. To our opinions, conservative management can be successfully carried out in elderly patients and patients at a high surgical risk, who have small infarction with slow bleeding and absence of other mechanical complications. Additional to timely diagnosis, discontinuing any antithrombotics is an important measure to ensure the conservative management to succeed. Now that antithrombotics are mandatory to patients with STEMI, it is crucial to avoid further bleeding by discontinuing them. For patients with small infarction and stent in noncritical site, this management is reasonable. Otherwise, it will be a dilemma to determine whether antithrombotics should be discontinued or not like in Case 1. Under this situation, surgical management is preferred.

In conclusion, terming subacute free wall rupture syndrome here is to help prompt detection and diagnosis of free wall rupture after STEMI. As for its rarity, it needs more cases to prove the term.

## Conflict of Interest

None declared.

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## Supporting Information

Additional Supporting Information may be found online in the supporting information tab for this article:

**Video S1** Chest X-ray testing taken from RAO view showed a "differential density sign".

 