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Triangular QRS-ST-T Waveform Electrocardiographic Pattern in Acute Myopericarditis: A Case Report from a Limited-Resources Hospital

Authors' Contribution:

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
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Conflict of interest: None declared

Patient: Male, 50-year-old
Final Diagnosis: Acute myopericarditis
Symptoms: Abdominal pain • chest discomfort
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Unusual clinical course

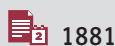
Background: Triangular QRS-ST-T waveform (TW) electrocardiography pattern has been found to be associated with poor prognosis in patients with ST-segment elevation myocardial infarction (STEMI). It identifies a subset of patients at high risk of both ventricular fibrillation and cardiogenic shock, with high in-hospital mortality. Therefore, aggressive treatment is needed in patients presenting with this electrocardiography pattern. However, this pattern is rarely present in non-ischemic cardiac diseases.

Case Report: We report the case of a 50-year-old man who came to our emergency room with a chief complaint of gastrointestinal problems and partial bowel obstruction. After failure of initial conservative treatment, laparotomy was planned. Just before the surgery, the patient felt a non-specific chest discomfort and showed ST-segment elevation on ECG and slight elevation of cardiac enzyme. He was then treated for STEMI with an intravenous thrombolytic. However, the degree of ST-segment elevation further increased and showed a TW pattern. Transthoracic echocardiography revealed a moderate pericardial effusion with normal ejection fraction and a normokinetic left ventricle; hence, a diagnosis of acute myopericarditis was made. After treatment with low-dose steroid and colchicine, his symptoms improved, the electrocardiography pattern returned to normal, and the gastrointestinal symptoms resolved.

Conclusions: To the best of our knowledge, this is the first case report of an acute myopericarditis presenting with a TW electrocardiography pattern. Myopericarditis should always be considered in the differential diagnosis of acute chest pain and ST segment electrocardiography changes, including TW pattern. The use of echocardiography can help determine the diagnosis of myopericarditis.

MeSH Keywords: Case Reports • Electrocardiography • Myocardial Infarction • Pericarditis

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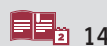
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Background

The triangular QRS-ST-T waveform (TW) electrocardiography (ECG) pattern, also known as shark fin pattern, or lambda-like pattern, is an uncommon ECG finding that has been found to be associated with poor prognosis in patients with ST-segment elevation myocardial infarction (STEMI) during the acute phase [1]. The TW pattern identifies a subset of patients at high risk of both ventricular fibrillation (VF) and cardiogenic shock, as well as associated high in-hospital mortality. The incidence of this TW pattern is approximately 1.4% of the population, based on a cohort study of 367 consecutive STEMI patients [2]. A previous study found that TW pattern was significantly more prevalent among the selected population of STEMI complicated by VF compared with the control population of uncomplicated STEMI (48% vs. 4.1% respectively) [3]. Therefore, a prompt and aggressive strategy is needed for patients presenting with this ECG pattern [2]. However, this pattern is rarely present in non-ischemic cardiac diseases. We present a case of a possible non-ischemic cause, namely acute myopericarditis, that presented with the TW ECG pattern, at a limited-resources hospital in a rural area. After the myopericarditis was treated, the ECG pattern returned to normal.

Case Report

A 50-year-old man came to the hospital emergency room with a history of frequent mucoid diarrhea and abdominal cramp for 1 week, and was unable to defecate, had distended abdomen and bilious vomiting 1 day before admission. On physical examination his vital signs were: Blood pressure 110/60 mmHg, heart rate 100 beats per min, respiratory rate 24 breaths per

min, and axillary temperature of 36.8°C. Heart and lung evaluations were within normal limits. He and his family had no relevant past medical history of any chronic disease or cardiac disease. However, he was a regular cigarette smoker for 30 years. The abdominal evaluation showed a distended abdomen, increased bowel sound, and bowel movement frequency. His ECG evaluation showed no abnormalities. An abdominal X-ray and ultrasonography evaluation revealed high partial bowel obstruction sign without mechanical cause. Laboratory tests showed leukocytosis ($24\ 100/\text{mm}^3$) with leukocyte differential count shift to the left (neutrophil 79.2%, lymphocyte 13.0%, monocyte 7.4%, and eosinophil 0.1%) and a slightly high pro-calcitonin level (0.55 ng/ml).

Based on the initial evaluation, the patient was diagnosed with partial bowel obstruction and enteritis. We administered 1 g ceftriaxone twice a day for 4 days intravenously and did an abdominal decompression using a nasogastric tube. However, on the second day in the hospital ward, the abdominal distention persisted. Therefore, laparotomy was planned to be done immediately. One hour before the surgery, the patient felt a non-specific chest discomfort with all vital signs within normal limits, indicating a stable hemodynamic. An ECG re-evaluation showed an ST-segment elevation at the anterior and inferior leads (Figure 1). Creatine kinase-MB (CK-MB) evaluation showed a slightly elevated titer (27 U/L). The patient was then diagnosed with STEMI.

Due to the unavailability of coronary angiography at our hospital, the STEMI was treated with a loading dose of dual anti-platelet (aspirin 180 mg and clopidogrel 300 mg) and intravenous nitroglycerin 10 $\mu\text{g}/\text{min}$, followed by a thrombolytic therapy of 100 mg alteplase over 1.5 h infusion. After the

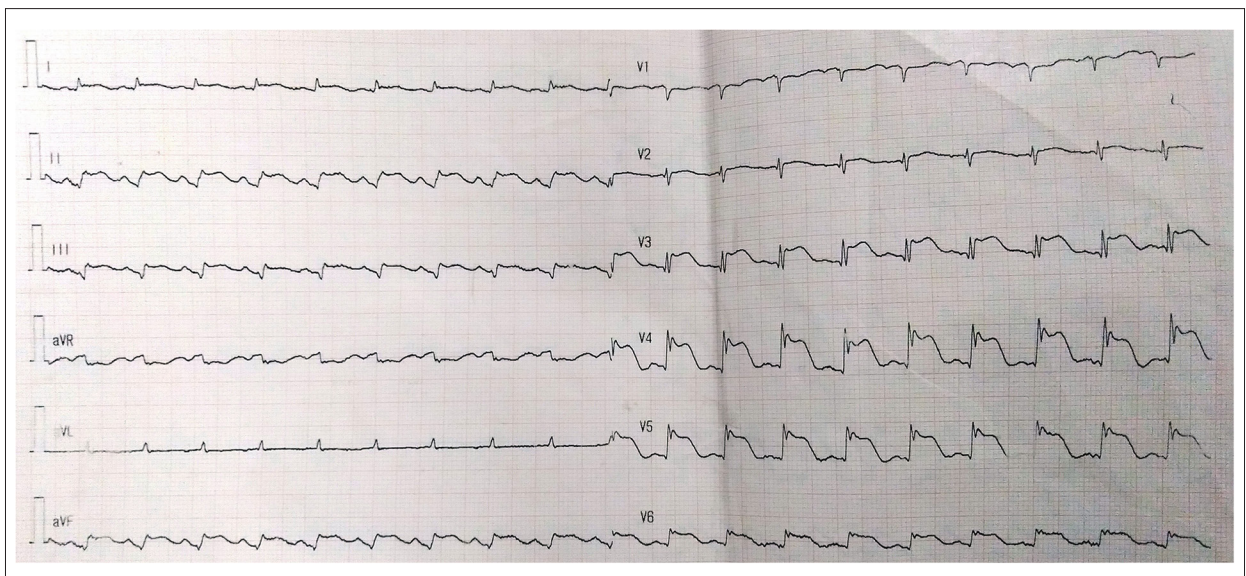


Figure 1. ECG pre-surgery showed diffused J point and ST-segment elevation.

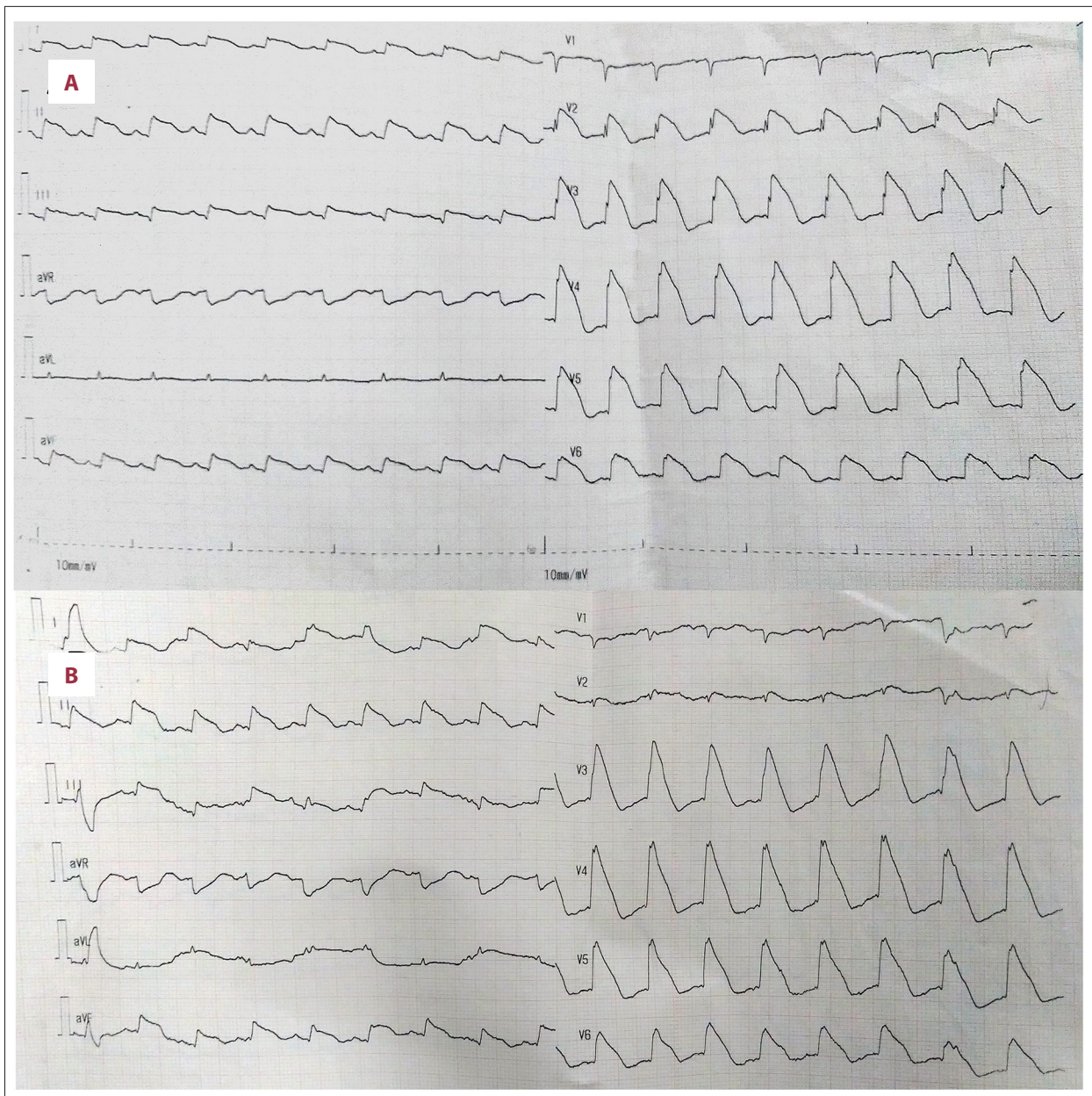


Figure 2. Post-thrombolytic ECG showed higher degree of ST-segment elevation. (A) One hour post-thrombolytic, (B) Two hours after thrombolysis.

thrombolytic procedure, the chest discomfort persisted, and the ECG evaluation revealed a TW pattern in the precordial lead and a diffuse ST-segment elevation in the other leads (Figure 2). Transthoracic echocardiography (TTE) evaluation was performed and revealed minimal to moderate pericardial effusion with normokinetic and normal right and left ventricle systolic function (62% ejection fraction).

Based on these findings, we suspected acute myopericarditis as the diagnosis. A loading dose of 2 mg oral colchicine followed by 1.2 mg once daily and 8 mg methyl prednisolone 3

times daily was then given as treatment for myopericarditis. A computed tomography (CT) scan obtained a few hours after the treatment also supported the diagnosis of myopericarditis. It also showed a mucosal thickening of the ileus, which is a sign of enteritis, suggesting the diagnosis of inflammatory bowel disease (IBD) (Figure 3). Stool testing on the next day (day 3 of hospitalization) showed positive mucous and fecal leukocytes, with negative results for human immunodeficiency virus, hepatitis B virus, and tuberculosis. The diagnosis of the patient was then changed to acute myopericarditis secondary to suspected extra intestinal manifestation of IBD.

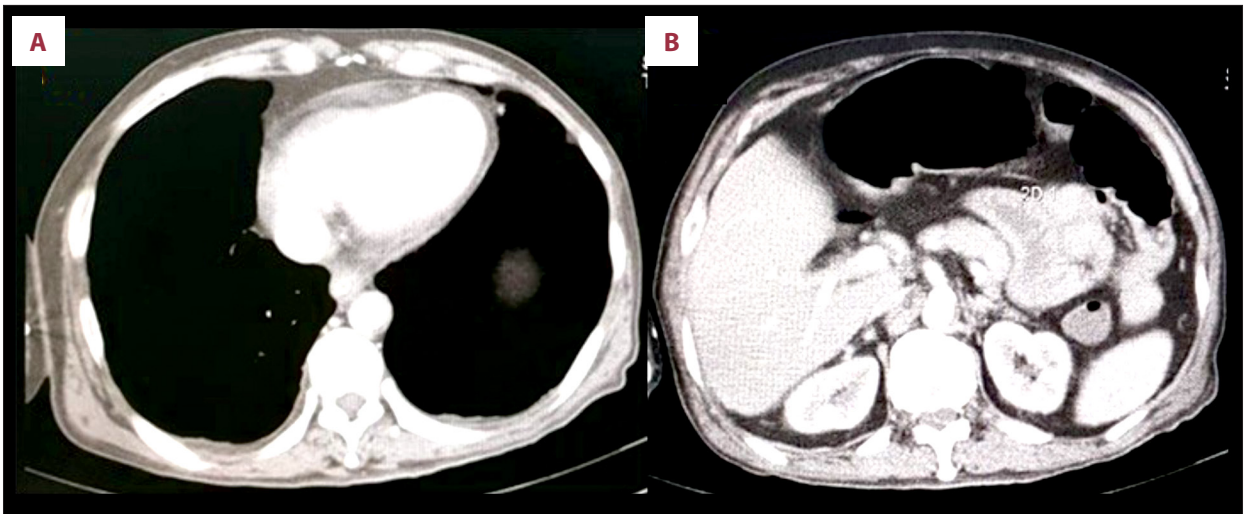


Figure 3. (A) CT scan contrast showed enhance pericardium, supporting pericarditis, (B) Mucosal thickening of enteritis.

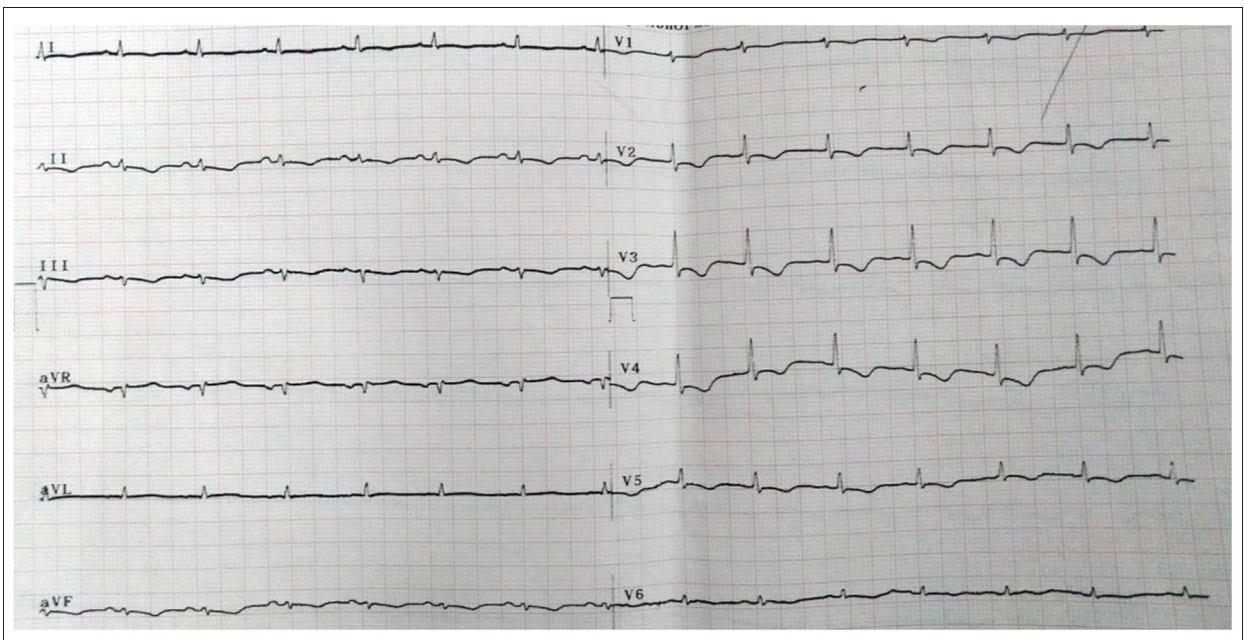


Figure 4. ECG on the third day of acute myopericarditis treatment.

The patient was then referred to undergo a coronary angiography evaluation, but unfortunately he and his family refused.

Three days after the therapy for myopericarditis was initiated (5th day of hospitalization), the ECG evaluation showed progressive resolution back to normal baseline (Figure 4). Chest pain and abdominal symptoms were also resolved. The patient was then discharged 2 days later (on the 7th day of hospitalization) with oral colchicine 0.6 mg twice daily continued for 3 months and methyl prednisolone tapered off to 8 mg once daily continued for 7 days. He was also given omeprazole 20 mg once daily to prevent any gastric problems related to steroid therapy and was instructed to restrict exercise for at least 3

months. Three months later, the patient came to the outpatient clinic for routine follow-up, with good adherence with the therapy and without any adverse effects. He did not have any remaining symptoms, and the TTE evaluation showed normal findings without any remaining pericardial effusion.

Discussion

To establish the diagnosis of acute pericarditis, at least 2 of the following criterias should be fulfilled: 1) typical chest pain (sharp and pleuritic), 2) pericardial friction rub, 3) electrocardiographic changes (widespread ST-segment elevation or PR-segment

depression), and 4) new or worsening pericardial effusion [4]. The diagnosis of myopericarditis can be made once the diagnosis of acute pericarditis has been established, followed by elevation of cardiac markers of injury without new onset of focal or diffuse depressed left ventricular function by echocardiography or cardiac magnetic resonance [5]. In our case, criterias 3 and 4 of pericarditis were fulfilled from the ECG and TTE findings, and an elevated CK-MB without a new onset of depressed left ventricular function was found.

According to the latest guideline, the first-line therapy for acute pericarditis is a nonsteroidal anti-inflammatory drugs (NSAID), and the second line is corticosteroid. In addition to that, colchicine should also be given as a combination to prevent recurrence in the future [4]. However, in patients with suspected IBD, as in our case, NSAID should not be given because NSAID could induce or exacerbate the preexisting ulcerative colitis by inhibiting cyclooxygenase and decreasing prostaglandin synthesis, which is cytoprotective to the intestine [6]. Therefore, it is suggested that corticosteroid should be used as first-line therapy in IBD patients with acute pericarditis [7].

The TW pattern is defined as the presence of a giant R wave (>1 mV), followed by a steep down-sloping ST segment, which conceals the T wave, and continues to the isoelectric line, thus forming a triangular shape, looking like a shark fin [2]. Wang et al. found that TW is an extreme form of the lambda wave pattern based on the geometry evaluation [8]. Other than in ischemic conditions, TW pattern can also present in non-ischemic conditions, such as in takotsubo cardiomyopathy. In takotsubo cardiomyopathy, the presence of TW is associated with higher risk of complications at short-term and long-term follow-up [9]. To the best of our knowledge, this is the first report of TW ECG pattern in a patient with acute myopericarditis. There was only 1 case report in the literature of a patient with acute pericarditis, but without myocardial involvement, who presented with an ECG pattern similar to our patient, and highlighted the importance of echocardiogram evaluation to confirm the diagnosis [10].

To rule out coronary artery as the cause, coronary or CT angiography should be done. However, it could not be performed in our case because of the limited facilities in our hospital and the patient's refusal to be referred to a tertiary referral hospital. The diagnosis of STEMI was less likely in our case because there was a discrepancy between the clinical (no typical chest pain, only minor atypical chest discomfort) and the ECG findings (ST-segment elevation in all leads). The ST-segment elevation itself also showed no reciprocal changes as in STEMI, supported by the normal LV and RV function without any hypokinetic or akinetic in TTE. A closer evaluation of the ECG before the TW pattern appeared (Figure 1) also showed a diffused concave-up ST-segment elevation pattern and PR-segment depression in leads II, III, aVF, and V3-V4, and

reciprocal PR-segment elevation and ST-segment depression in aVR, which is a pathognomonic finding for pericarditis [4]. The QRS duration when the ST-segment elevation occurred (Figure 1) was also not prolonged (120 ms) compared to the normal baseline ECG (Figure 4), which indicates an epicardial rather than transmural myocardial injury [11,12].

Another possibility that can cause transient ischemic ECG changes such as ST-segment elevation is vasospastic angina. Vasospastic angina is diagnosed if the nitrate-responsive angina is evident during spontaneous episodes [13]. However, the chest discomfort in our patient did not respond to nitroglycerin treatment. Thus, we argue that vasospastic angina was less likely in our patient. The provocative spasm testing for vasospastic angina could not be done in our hospital due to the limited facilities. The criterion standard method involves the administration of provocative stimulus during invasive coronary angiography with the monitoring of the patient symptoms, ECG, and angiographic documentation of coronary artery spasm [13].

ST-segment deviation in patients with acute myopericarditis occurs because of the presence of 3 separate factors: pericardial effusion, injury of the superficial myocardium by the pressure of fibrin or fluid, and superficial myocarditis secondary to local inflammatory changes in the epicardium underlying the inflamed pericardium [14]. We argue that at least 1 of those factors contributed to the alteration of cardiac Na⁺, K⁺, and Ca⁺ channels, leading to abnormal currents and appeared as the TW pattern, similar to the mechanism of TW pattern in STEMI due to transmural myocardial injury. However, further research is needed to elucidate the possible mechanism of TW ECG pattern in myopericarditis.

Study limitations

There was no coronary or CT angiography facility in our hospital, thus coronary involvement could not be totally ruled out and remained as a differential diagnosis. The patient also refused to be referred for further workup for his IBD or possible coronary occlusion. In addition, the titer of antibodies of usual viral myocarditis, such as coxsackie virus, also could not be measured in our hospital. Nevertheless, with the good response of the patient with standard therapy of myopericarditis, and from our available clinical data, we present the first case report of likely myopericarditis with a good outcome as a possible differential cause of TW ECG pattern other than life-threatening ischemic conditions.

Conclusions

The TW ECG pattern indicates the need for prompt management and can appear not only in myocardial infarction, but

can also be found in myopericarditis, as in our case. Therefore, myopericarditis should be considered in the differential diagnosis of acute chest pain and ST segment ECG changes, including TW pattern. Unlike TW pattern in STEMI cases which always has a poor prognosis, myopericarditis usually has a good response to medical treatment, as seen in our patient. The use of echocardiography can aid in differentiating the diagnosis when immediate coronary angiography cannot be done.

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Conflict of interest

None.

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