

CASE REPORTS

Dressler's syndrome as a complication of apical ballooning cardiomyopathy

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ABSTRACT

We report a case of 62-year-old female patient diagnosed with apical ballooning syndrome (Takotsubo) based on typical apical ballooning with normal coronaries in the setting of chest pain and ST elevation MI. Ten days after her initial hospitalization she was diagnosed with pleuropericarditis based on chest pain, pleuropericardial effusion and inflammatory markers.

This case report shows the rare complication of takotsubo cardiomyopathy (TC) – Dressler's syndrome.

Key Words: Apical ballooning, Dressler's syndrome, ST elevation MI, Chest pain

1. INTRODUCTION

Takotsubo syndrome was first described in 1990, and since then had been recognized more and more frequently.

Takotsubo syndrome which is also known as “stress-induced cardiomyopathy”, “broken heart syndrome” and “apical ballooning syndrome”, is usually an acute and reversible heart failure that can be caused by multiple triggers and is thought to be caused by catecholaminergic myocardial stunning.^[1,2] For proper diagnosis, other causes of wall motion abnormalities such as coronary heart disease, myocarditis and other types of cardiomyopathies have to be excluded.^[3-5] Takotsubo syndrome affects primarily post-menopausal women, but can also affect younger women and men.^[6] The presenting symptoms are usually of typical chest pain, palpitations and shortness of breath.^[7] In some cases, ventricular tachyarrhythmia or cardiogenic shock may ensue.^[8,9] Cardiac enzymes are elevated in about 90% of the patients but are

often disproportioned to the wall motion abnormality as seen by echocardiography.^[9,10] ECG abnormalities are seen in about 95% of patients, and might include ST segment abnormalities, Q wave development, T wave inversion and later QT prolongation.^[8,10] On angiography the coronary arteries are usually normal without significant atheromas.^[9,11] On ventriculography an apical hypokinesis is often seen.^[12,13] Echocardiography study (the noninvasive modality of choice for the diagnosis of the syndrome) typically reveal extensive areas of dysfunctional myocardium that cannot be attributed to a single coronary artery obstruction.^[14,15]

Several diagnostic criteria have been proposed in recent years, in order to identify the condition as Takotsubo syndrome. In 2015 new diagnostic criteria by the Heart Failure Association of the European Society of Cardiology (HFA) were published and are as follows:

- Transient apical dyskinesia or akinesia, detected by

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Echocardiography color Doppler, beyond a single coronary artery distribution.

- Nonobstructive coronary artery disease (stenosis < 50%) at angiography.
- Absence of: myocarditis, pheochromocytoma, head trauma and intracranial hemorrhage, hypertrophic cardiomyopathy.

In diagnosing takotsubo cardiomyopathy, the almost unique association of normal coronary arteries and extensive apical akinesia may be demonstrated in the acute phase of the disease.^[16]

Typically, left ventricular function fully recovers within the first 12 weeks after the initial insult. ECG abnormalities may remain for long periods and sometimes forever. No study has demonstrated any mean for successful prevention

of recurrence.^[17-19]

2. CASE PRESENTATION

A 62 years old married female patient with a medical history of diabetes mellitus, hypertension, smoking and hyperlipidemia was admitted to the emergency department after she had complained of chest pain accompanied by nausea, vomiting and profuse sweating. She had no prior ischemic events, nor a history suggestive of rheumatological disease. The patient's medication at home included S.C. Liraglutide 1.2 mg q.d., Metformin 850 mg t.i.d., Atorvastatin 40 mg q.d. On admission, the ECG demonstrated inverted T waves in inferior, anterior and lateral leads (see Figure 1). Troponin levels were 0.443 (normal range 0-0.03 ng/ml). Maximal measured levels of CPK were normal 164 (normal range 26-192 U/L).

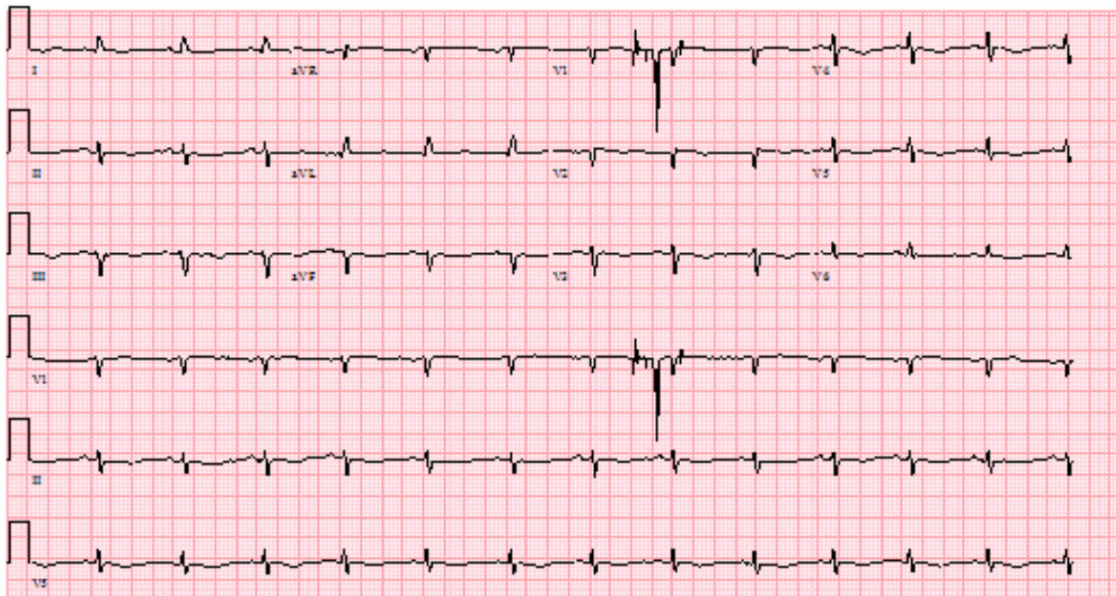


Figure 1. The ECG on admission, demonstrated inverted T waves in inferior, anterior and lateral leads

The patient was hospitalized with the presumed diagnosis of non ST elevation myocardial infarction.

Coronary catheterization performed a day after her arrival showed mild non obstructive coronary artery disease with typical apical ballooning (see Figure 2).

An echocardiogram showed mildly to moderately reduced left ventricular global systolic function and mildly reduced global systolic function of the right ventricle (see Figure 3).

The patient was treated with an ACE inhibitor with resolution

of all her symptoms and she was discharged a few days after her admittance.

Ten days after the first hospitalization the patient returned to the emergency department with pleuritic chest pain of several hours. The patient had no prior history of viral infection. Her vital signs were within the normal range besides fever of 37.8°C and on cardiac auscultation no pericardial friction rub was heard.

On ECG, deeper T wave inversion were noted, as compared to previous ECG (see Figure 4).

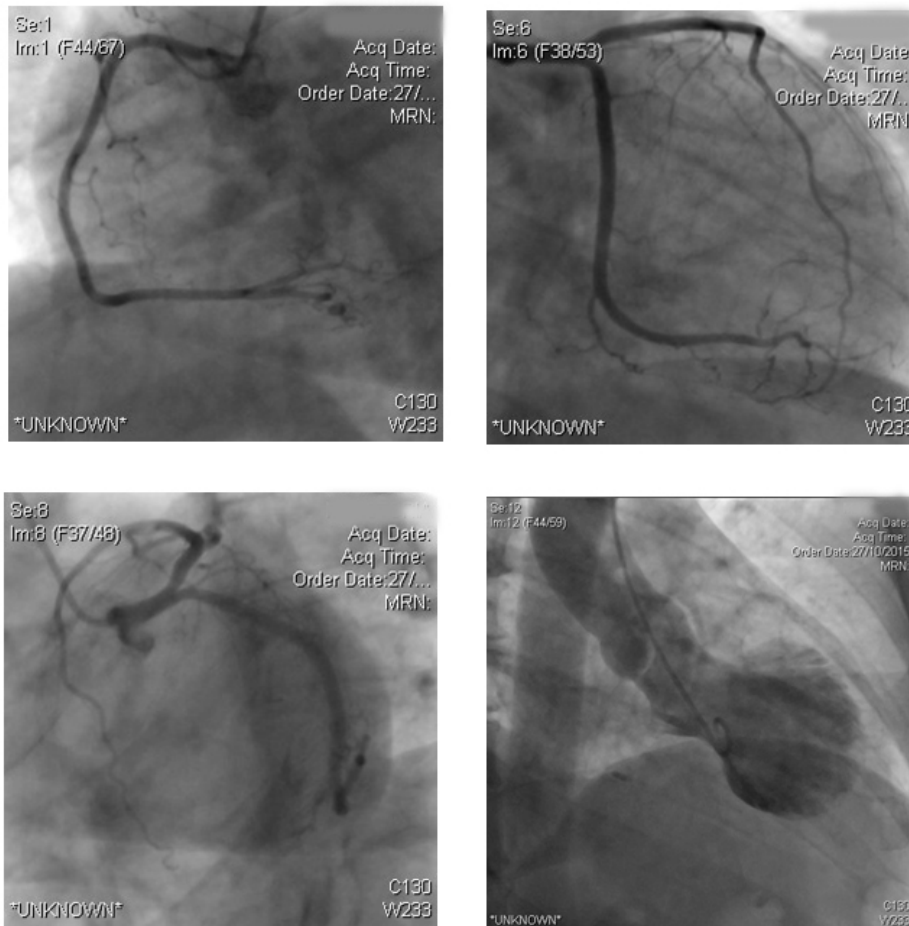


Figure 2. Coronary catheterization showed mild non obstructive coronary artery disease with typical apical ballooning

Her cardiac enzymes showed troponin of 0.009 (normal range 0-0.03 ng/ml) and CPK of 48 (normal range 26-192 U/L). Her CBC showed 14,900 leukocytes (normal range 4,000-10,000 white blood cells per microliter) with 70.4% neutrophil count (normal range 50%-70% relative value). Her CRP was 1.8 (normal range 0-0.5 mg/dl). Chest X-ray revealed a small bilateral pleural effusion (see Figure 5).

An echocardiography examination was performed which demonstrated an improved left sided global systolic function and mild amount of pericardial fluid which did not exist on the previous exam, without any signs of a hemodynamic compromise (see Figure 6).

She was then hospitalized with the presumed diagnosis of Dressler's syndrome and we began treatment with Ibuprofen and Colchicine. Under that treatment a gradual improvement in her pain was noted, until a complete resolution. Leukocytosis and CRP, which reached a peak of 15,600 and 8.07, declined during her hospital stay to 11,500 and 2.65 (WBC/mcl and mg/dl respectively).

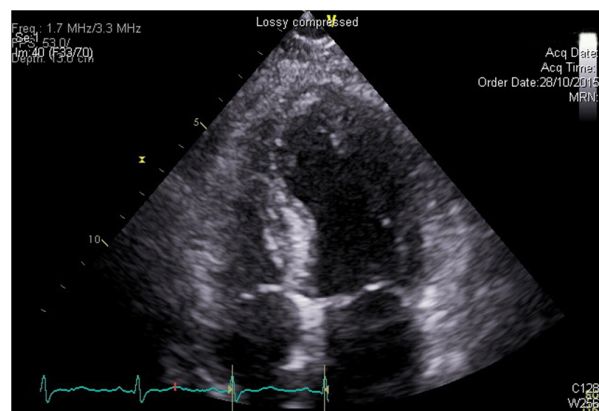


Figure 3. An echocardiogram showed mildly to moderately reduced left ventricular global systolic function and mildly reduced global systolic function of the right ventricle

The patient was then discharged with continuous Colchicine and Ibuprofen treatment.

After two months of follow-up her echocardiography study showed normalization of global systolic function.

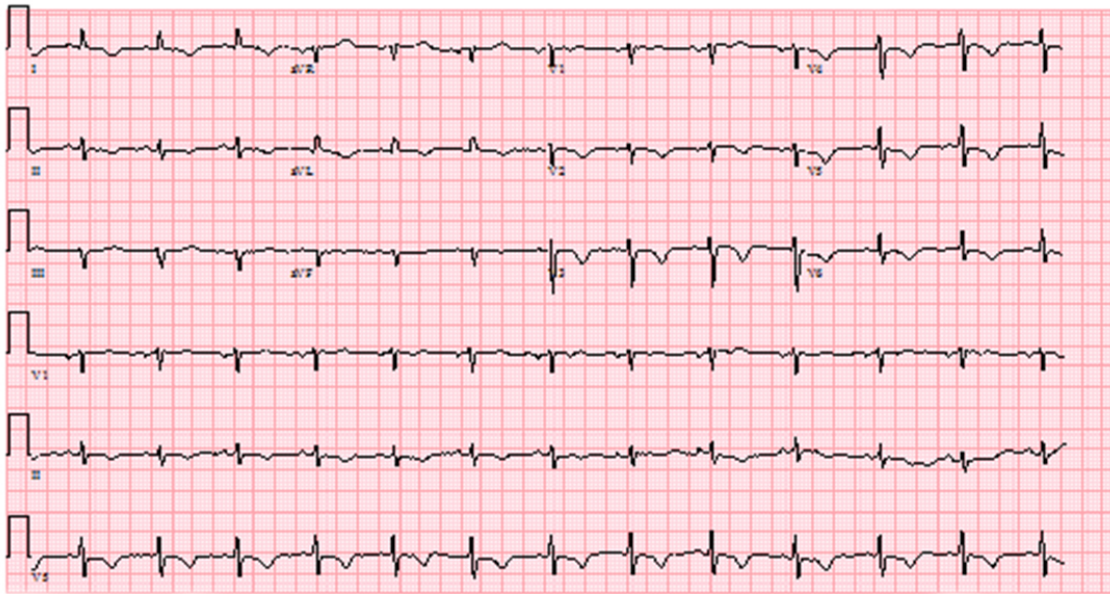


Figure 4. ECG on readmission showing deeper diffuse T wave inversion



Figure 5. Chest X-Ray with bilateral pleural effusion

3. DISCUSSION

Although several case reports have identified the possible TC-pericarditis association, the exact pathogenesis remains unclear.^[20] Several theories have been hypothesized to explain the TC-pericarditis connection including the transmural inflammation or myocarditis associated with TC.^[20]

Another theory maintains that viral myocarditis could mimic TC and potentially spread to the pericardium.^[21] There are several case reports in which biopsy-proven viral myocarditis presents with left ventricular apical ballooning.^[22] This suggests that cases originally thought to be TC may have

actually been myocarditis, mimicking TC.^[22,23] Lastly, acute pericarditis could be the primary event and TC could be its consequence.^[21] Pericarditis can cause severe pain, which could prompt catecholamine release.^[21]

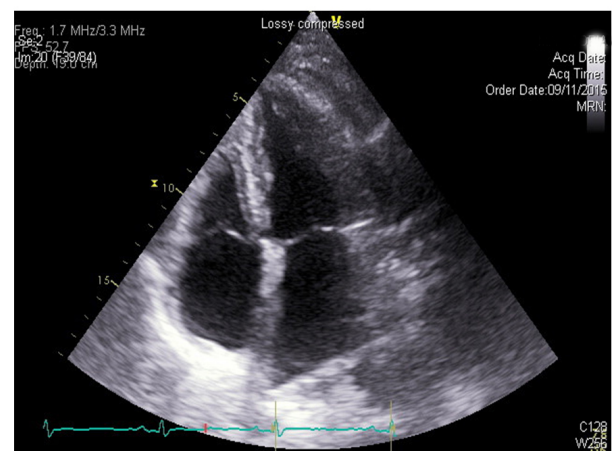


Figure 6. An echocardiography demonstrated an improved left sided global systolic function and new mild pericardial fluid effusion

Our patient's initial presentation was compatible with a diagnosis of Takotsubo cardiomyopathy. The pathogenesis of this condition is presumably based on coronary artery vasospasm leading to apical myocardial stunning accompanied often by minimal myocardial necrosis.^[24-27]

Dressler's syndrome is characterized by late onset pericardi-

tis, occurring weeks to months after the occurrence of myocardial infarction.^[28] Two weeks following the diagnosis of Takotsubo cardiomyopathy, the patient demonstrated typical features of Dressler's syndrome: pleuritic chest pain, pericardial and pleural effusion, with associated fever, leukocytosis and elevated levels of inflammatory markers.^[28] The development of Dressler's syndrome is considered to be immune-mediated with involvement of pericardium, pleura and lungs.^[29-31]

In the case of our patient, Takotsubo cardiomyopathy developed into Dressler's syndrome 2 weeks later. To note, a

complete immune panel was unfortunately not established. Despite this, the clinical presentation as well as the timing of disease onset strongly suggest a connection between the two entities. To date, there is scant literature available regarding Dressler's syndrome following Takotsubo cardiomyopathy, and the exact mechanism of how it develops is unclear and warrants further investigation. Though rare, clinicians should be aware of the possibility of pericarditis following apical ballooning.

CONFLICTS OF INTEREST DISCLOSURE

The authors have declared no conflicts of interest.

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