Emergency Bulletin
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The alarming "shark fin" in an old lady presenting with a fall

Dr. Ho Ka Yin, Kyle

A 72-year-old lady presented to our emergency department for a slip-and-fall injury associated with bilateral lower limb weakness. She had a past medical history of hypertension, hyperlipidemia, and diabetes mellitus.

Upon her arrival, her blood pressure was 121/72 mmHg, with a pulse rate of 122/min. She was afebrile. The oxygen saturation was 96% on 1L O2. The Glasgow Coma Scale was full.

Upon examination, she looked tired and dyspneic on oxygen supplement with a respiratory rate of 20 per minute. The chest and abdominal exams were unremarkable. The lower limbs power was 4+ out of 5 on MRC grading. Her lumbosacral spine and bilateral hips were non-tender upon palpation.

A chest X-ray has been done, and it was unremarkable. However, her ECG (figure 1) showed some alarming features. The ECG showed a sinus tachycardia rhythm of around 120/min. The most striking feature was the diffuse ST elevation in leads V2-6, I, II, III, AVF and Q wave in inferior leads. It also showed left axis deviation with ventricular ectopic beats.

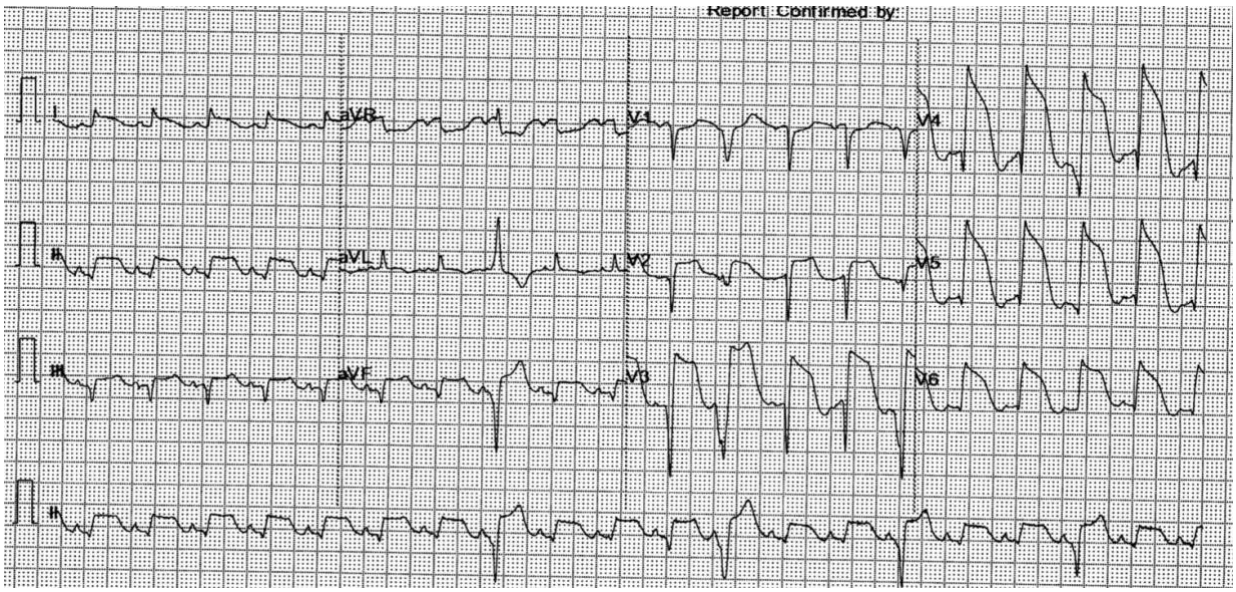

Figure 1. The ECG obtained from the patient

Her point-of-care blood tests showed venous blood pH of 7.29 and elevated lactate level to 6.13mmol/L, while sodium and potassium levels were unremarkable. Her blood glucose (by point-of-care testing glucometer) was 16.1 mmol/L.

Upon further enquiry to her daughter, the patient had mild upper respiratory tract illness for a few days, and subsequently complained of generalised malaise and had multiple falls. Her daughter also reported that the patient lived alone and was suspected to have cognitive impairment, with doubtful compliance to her diabetic medications.

Despite the abnormal ECG findings, the patient did not complain of chest pain. The attending A&E doctor performed a focused transthoracic echocardiogram, but the findings on cardiac wall motion abnormalities were inconclusive due to the suboptimal images obtained. Blood tests including troponin I and creatinine kinase were also checked, but the results were not shortly available. In view of the alarming ECG finding, she was managed as a case of acute ST elevation myocardial infarction (STEMI). The patient was subsequently admitted to the cardiac coronary unit (CCU) for primary percutaneous coronary intervention (PCI).

What are the differential diagnoses of ST elevation ECG pattern?

Myocardial infarction is undoubtedly the most common and important cause of ST elevation. However, there are also some less common STEMI-mimics that we may encounter in emergency departments as shown in Table 1. Emergency physicians must consider these differential diagnoses, understand the different ECG patterns and make appropriate judgements based on the clinical findings.

Etiology	Remarks
Acute pericarditis	Diffuse ST elevation Concave upward ST elevation PR depression in multiple leads
Myocarditis	May simulate ECG pattern of acute pericarditis or STEMI
LV hypertrophy	Typical LV strain pattern Concave ST elevation <25% of QRS or <2.5mm in V1-V3
Left bundle branch block (LBBB)	Typical LBBB pattern with ST elevation <5mm and deep S waves in V1-V3 Sgarbossa Criteria for concurrent STEMI
Hyperkalemia	Narrow-based symmetrical tented & pointed T waves (in contrast to broad based non-pointed hyperacute T waves in STEMI)
Takotsubo (stress) cardiomyopathy	Mimics all ECG features of STEMI, but extends beyond single coronary artery territories "Shark fin" appearance
Brugada pattern	Specific Brugada pattern ECG ST elevation mainly at V1-2 with right bundle branch block
Early reploarization	Elevated J point in healthy young subjects Concave ST elevation <3mm

Table 1. Different etiologies of ST segment elevation on the ECG (LV, left ventricle)

Progress of the patient

Coronary angiogram was performed, but no major coronary artery occlusion was found to account for the STEMI ECG pattern. MINOCA (Myocardial infarction with nonobstructive coronary arteries) and Takotsubo cardiomyopathy were suspected. The patient was then admitted to the CCU for further management.

Bedside echocardiogram was subsequently

performed by the cardiologist in the CCU, which showed left ventricular ejection fraction (LVEF) of 40-45%, which was low as the normal range should be around 60-70%. It also showed apical and anteroseptal hypokinesia, and suspected apical ballooning.

The patient's blood results revealed troponin I level of over 50000 ng/mL, creatinine kinase level of 79932 U/L. The venous blood gas showed a blood pH level of 7.29, with random glucose of 16.1mmol/L and elevated beta-hydroxybutyrate level of 2.32 mmol/L (normal range <0.5mmol/L). Sepsis was suspected in view of elevated white cell count 21.4 x 10^9/L, elevated serum lactate level 6.13 mmol/L, and elevated procalcitonin level of 29 ng/ml (>2 ng/ml signifies high risk of sepsis). The culture of catheterised urine also yielded E. Coli.

The clinical picture was compatible with diabetic ketoacidosis complicated with sepsis, which then led to stress (Takotsubo) cardiomyopathy.

Supportive treatment including intravenous fluid, insulin and broad-spectrum antibiotics were given to the patient. However, the patient's condition continued to deteriorate despite the above measures. She developed multiple runs of sustained ventricular tachycardia (VT) with stable hemodynamics which required anti-arrhythmics including amiodarone and lignocaine. She subsequently developed cardiogenic shock which was refractory to double inotropes therapy. The CCU doctor had consulted ICU for extracorporeal membrane oxygenation (ECMO) placement, however the case was not considered to be eligible for ECMO by the ICU doctor. Therefore, the patient was subsequently put on intra-aortic balloon pump (IABP) for hemodynamic support and Swan Ganz catheter for hemodynamic monitoring.

The patient's renal function deteriorated and she was transferred to ICU for renal replacement therapy.

With supportive treatment, the patient's condition gradually improved. She was then transferred back to the general ward for further rehabilitation.

Takotsubo Cardiomyopathy

Background

The name of Takotsubo cardiomyopathy originated from the Japanese term used to describe an "octopus trap". It is also known as stress cardiomyopathy, apical ballooning syndrome and broken heart syndrome. It is a syndrome characterized by transient regional systolic dysfunction, mimicking a myocardial infarction (MI).

Takotsubo cardiomyopathy was first described in 1990 in Japan [2]. Its prevalence is around 1-2 % in patients presenting with troponin-positive suspected acute coronary syndrome (ACS) or suspected ST-elevation MI [3]. The disease is much more common in women than men and occurs predominantly in older adults [4].

The pathogenesis of this disorder is not well understood. Similar to MI, Takotsubo cardiomyopathy mainly affects the systolic and diastolic functions of the left ventricle. Postulated mechanisms include catecholamine excess [5,6], microvascular dysfunction, and coronary artery spasm.

As its name implies, physical and emotional stress are common triggers of Takotsubo cardiomyopathy. In the International Takotsubo Registry study [4], patients with psychiatric or neurologic disorders are more likely to develop the condition than those who are not. However, the pathogenesis behind such a relationship remains to be established.

Genetic predisposition is also one of the possible factors, as there have been reports of familial cases [7].

As mentioned above, Takotsubo cardiomyopathy is usually triggered by intensive emotional or physiological stress, such as death of relatives, loss of job, divorce, domestic abuse or acute medical illness. In a systematic review including 19 studies with a total of 1109 patients [8], 35% of them had a prior physical trigger, while 39% reported an emotional trigger.

The signs and symptoms of Takotsubo cardiomyopathy are similar to acute MI to some extent. The most common symptom would be substernal chest pain, some may also present with dyspnea or syncope [4]. Some patients may develop signs and symptoms of heart failure, tachy- or brady-arrhythmias, acute mitral regurgitation or even sudden cardiac arrest. Approximately 10% of patients with Takotsubo cardiomyopathy develop symptoms and signs of cardiogenic shock.

LV outflow tract (LVOT) obstruction could also be induced by LV basal hyperkinesia in patients with Takotsubo cardiomyopathy. It can contribute to the development of shock and cause severe mitral regurgitation.

Investigations

For investigation and diagnosis, electrocardiogram (ECG) abnormalities are common in these patients, with ST segment elevation being the most common abnormal features [4], followed by ST depression, QT prolongation, T waves inversion or other nonspecific abnormalities.

Going back to our patient, her ECG (figure 1) shows a typical “Shark fin” pattern. Shark fin sign is also known as “Lambda-wave”, “giant R waves”, or “triangular QRS-ST-T waveform”. It is characterized by a giant R wave (amplitude >1 mV), and a QRS complex fused with the ST segment and T wave [9]. Shark fin sign is often associated with massive MI involving left anterior descending (LAD) coronary occlusion [10]. Less often, it is also associated with Takotsubo cardiomyopathy [11]. Patients with ECG of shark fin pattern have increased risk of adverse events, including ventricular fibrillation and cardiogenic shock [11]. After ruling out acute coronary occlusion by coronary angiogram in such patients, Takotsubo cardiomyopathy has to be considered.

Cardiac biomarkers, specifically high sensitive troponin levels, are always elevated in patients with Takotsubo cardiomyopathy. Brain natriuretic peptide (BNP), a less commonly used biomarker, is usually elevated.

In patients with presentation of suspected acute coronary syndrome (ACS), Takotsubo cardiomyopathy has to be considered as one of the differential diagnoses. A physical or emotional trigger is not always present.

There are several diagnostic criteria for Takotsubo cardiomyopathy, including Mayo

Clinic diagnostic criteria [12] and InterTAK Diagnostic Criteria. There is no consensus of which is better. The below are the Mayo Clinic diagnostic criteria:

1. Transient dysfunction (hypokinesis, akinesis, or dyskinesis) over the LV mid segments with or without apical involvement
2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture
3. New electrocardiographic abnormalities (either ST-segment elevation and/or T wave inversion) or modest elevation in cardiac troponin
4. Absence of pheochromocytoma or myocarditis

(Remarks: The wall motion abnormalities are typically regional and extend beyond a single epicardial coronary distribution but there are exceptions. If coronary disease is found, the diagnosis of stress cardiomyopathy can still be made if the wall motion abnormalities are not in the distribution of the coronary disease.)

Echocardiography is a useful assessment tool for Takotsubo cardiomyopathy. Regional wall motion abnormalities of the LV is one of the key features. "Apical type" is the most common form of LV dysfunction. In this subtype of LV dysfunction, there will be hypokinesia of the apical and mid segment of the LV, with relatively hyperkinesia of the basal segment, thus creating the typical "apical ballooning" morphology in echocardiography.
The apical ballooning morphology resembles the shape of an octopus trap used by Japanese, which explains the name of the disease as "takotsubo" means octopus trap in Japanese.

By and large, the diagnosis of Takotsubo cardiomyopathy requires consideration of several aspects including risk factors, clinical features, ECG and echocardiogram findings as well as the results from the coronary angiogram. The differences among Takotsubo cardiomyopathy, STEMI and MINOCA are presented in Table 2.

	Takotsubo cardiomyopathy	**MINOCA (except Takotsubo cardiomyopathy)**	**STEMI**
Risk factors	More common in older females, with or without cardiovascular risk factors	Similar cardiovascular risk factor burden to STEMI, but less hyperlipidemia	Mainly in patients with cardiovascular risk factors (HT, DM, hyperlipidemia)
Etiologies	Catecholamine excess, microvascular dysfunction or coronary artery spasm triggered by physical or psychological stress	Coronary artery spasm, transient thrombosis or coronary artery dissection, coronary artery embolism, or viral myocarditis	Coronary thrombosis due to acute atherothrombotic plaque rupture (coronary artery dissection or embolism less common)
Echocardiogram findings	Systolic LV apical ballooning, hyperkinesis of basal walls	Echocardiography findings varying according to underlying causes	Systolic dysfunction and regional wall motion abnormalities
ECG patterns	Extending beyond single vessel territories Shark-fin pattern possible	Typical features of STEMI or STEMI equivalents	Typical features of STEMI or STEMI equivalents
Coronary angiogram findings	No coronary occlusion found in coronary study	No coronary occlusion found in coronary study	Evidence of coronary occlusion

Table 2. Differences among Takotsubo cardiomyopathy, MINOCA and STEMI

Management

Takotsubo cardiomyopathy is generally a transient disorder triggered by physical or emotional stress. Supportive therapy with treatment of the identified stress (if any) is the mainstay of management. However, some patients may develop acute complications such as shock and acute heart failure that require intensive therapies. Some may also be complicated by significant left ventricular outflow tract (LVOT) obstruction.

Around 5 to 10% of patients with Takotsubo cardiomyopathy develop cardiogenic shock [4,13]. These patients are associated with a higher in-hospital mortality [14]. For patients in shock without significant LVOT obstruction, cautious fluid resuscitation could be given. Inotropic agents and vasopressors could be used if the patient failed to respond to fluid.

The management approach for shock would be different in patients complicated with LVOT obstruction. Inotropic agents should not be used as they may worsen the degree of obstruction [15]. Beta blockers are used to improve hemodynamics by relieving the obstruction. In patients without pulmonary congestion, fluid resuscitation and leg elevation could be considered as means to increase preload. Conversely, volume depletion and vasodilator therapy should be avoided as they may decrease the preload.

In patients who are unresponsive to initial medical therapy and volume resuscitation, mechanical circulatory support should be considered, such as IABP (Intra-aortic balloon pump) or ECMO (Extracorporeal membrane oxygenation).

For patients without significant LVOT obstruction who present with acute heart failure, intravenous diuretic and vasodilator therapies should be given.

Apart from shock and acute heart failure, patients with Takotsubo cardiomyopathy are also more prone to thromboembolic events. According to the International Takotsubo Registry study [4], ventricular thrombus was identified in 1.3% of the 1750 patients with Takotsubo cardiomyopathy. Currently there is not enough evidence to guide the routine use of anticoagulation therapy in patients with the disease. Imaging modalities like echocardiography can be used to detect the presence of LV thrombus.

There is also one pitfall when encountering Takotsubo cardiomyopathy. In centres without direct or early access to coronary angiography, IV thrombolysis treatment might be given to patients with clinical pictures compatible with MI, even if the underlying diagnosis is Takotsubo cardiomyopathy. This may delay the treatment of any underlying pathology that has led to the cardiomyopathy. The patient may also be exposed to the unnecessary bleeding risks that are caused by thrombolytic therapy.

Prognosis

Although most patients with Takotsubo cardiomyopathy would recover, the risk of severe in-hospital complications is similar to that in patients with acute coronary syndrome [4]. The in-patient mortality rate was quoted to be 4.1% in the International Takotsubo Registry study.

Long-term mortality and morbidity is also another concern, all-cause mortality of patients with Takotsubo cardiomyopathy is

shown to be higher than the general population [16]. The long-term in-patient mortality is also shown to be similar to patients who suffered from MI [17].

Lesson to learn:

1. **In patients with suspected ST elevation myocardial infarction (STEMI), STEMI mimics should always be considered. Takotsubo cardiomyopathy should be considered when there is ST elevation that extends beyond a single coronary artery territory.**
2. **The "Shark Fin" ECG pattern should be recognized. It is associated with severe myocardial injury and could be present in Takotsubo cardiomyopathy.**
3. **Echocardiography is useful for evaluation, and it typically shows LV dysfunction at apex and mid segments and hyperkinesis at the basal segments.**
4. **Evaluation of obstructive coronary heart disease should not be delayed even if Takotsubo cardiomyopathy is suspected, as the diagnosis could only be confirmed after negative coronary angiography.**
5. **The treatment is supportive, but identification and treatment of underlying physical stress is important.**
6. **The short term and long-term mortality and morbidity are comparable to patients with acute myocardial infarction.**

Editor's comments:

Fall is often the tip of an iceberg in geriatric patients, as many of these patients have multiple acute or chronic co-morbidities that contribute to this common presentation. The underlying contributing factor to the fall is not always straightforward, as in this case described by the author. Emergency physicians should always be vigilant about the potential co-morbid conditions by careful clinical evaluation and appropriate investigations.

The initial diagnosis of this patient was accidental fall, but the conditions this patient suffered from included diabetic ketoacidosis, sepsis, acute kidney injury and subsequently, Takotsubo cardiomyopathy. The role of emergency physicians is not only to focus on the primary complaint, but to actively search for predisposing conditions or concomitant diseases that are beyond our initial impression, in order to offer the right treatments to our patients.

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