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EC CARDIOLOGY
Case Report

# Challenging Case; A 33 Years Old Female Patient with Shortness of Breath

**Mahmoud Tantawy***

*Cardiology Department, Misr University for Science and Technology, Memorial Souad Kafafi University Hospital, 6 of October City, Cairo, Egypt*

***Corresponding Author:** Mahmoud Tantawy, Cardiology Department, Misr University for Science and Technology, Memorial Souad Kafafi University Hospital, 6 of October City, Cairo, Egypt.

**Received:** December 14, 2022; **Published:** January 25, 2023

## Abstract

A 33 years old female patient presented with shortness of breath of one month duration of acute onset and progressive course, patient had previous history of recurrent supra-ventricular tachycardia. Patient was generally ill with ECG revealed supra-ventricular tachycardia and concomitant severe chest infection. Patient was cardioverted successfully to sinus rhythm with no other abnormalities in the ECG and chest infection resolved after few days. Echocardiogram revealed picture suggestive of dilated cardiomyopathy with estimated ejection fraction 35%, global hypokinesia and severe mitral regurgitation. After one month follow up echocardiogram was normal and patient was generally well. Four months later patient presented to our hospital with shortness of breath again but without supra-ventricular tachycardia nor chest infection, echocardiogram revealed the first picture of dilated cardiomyopathy. Laboratory results showed elevated levels of catecholamines and mild elevation of cardiac enzymes. Patient refused medical treatment and after other month of follow up, echocardiogram was normal.

**Conclusion:** This case was an atypical presentation of recurrent broken heart syndrome with early differential diagnosis; myocarditis and tachycardia induced cardiomyopathy.

***Keywords:*** *Myocarditis; Tachycardia; Cardiomyopathy; Shortness of Breath*

## Introduction and Case Presentation

A 33 years old female patient married has one child 2 years old, with negative history for cardiovascular risk factors. Patient was presented by shortness of breath of one month duration of gradual onset and progressive course, increased by effort and relieved by rest, associated with myalgias and fatigue. She has fever, sweats, upper respiratory tract symptoms, cough with expectoration of yellowish sputum. She has no nausea, vomiting, diarrhea, rash or urinary tract symptoms.

Patient has history of recurrent attacks of supra-ventricular tachycardia since 10 years, successful cardioversion is always done by vagal stimulation.

### On examination

Patient was alert, conscious, oriented by time, place and persons. Patient was lying flat but with dyspnea and no orthopnea. The temperature was 38c, the blood pressure 110/70 mmHg, the pulse 180 beat per minute (regular and equal on both sides), the respiratory rate 22 breaths per minute, the oxygen saturation 92% on room air. Chest auscultation revealed bilateral fine basal crepitations, bronchial breathing and scattered coarse crepitations on the right side. Heart examination revealed, apex felt at six intercostal space outside midclavicular line, pan-systolic murmur grade III was heart over the apex propagated to axilla, fixed intensity during inspiration, and the remainder of the examination was normal.

Laboratory-test results are shown in table 1, all was normal except high white blood cells count mainly neutrophilia.

| Variable | Reference Range (Adults) | At First Visit | At Second Visit with symptoms |
|---|---|---|---|
| Hematocrit (%) | 36.0 - 46.0 (women) | 33.0 | 33.3 |
| Hemoglobin (g/dl) | 12.0 - 16.0 (women) | 10.7 | 11.3 |
| White - cell count (per $mm^3$) | 4,500 - 11,000 | 18,000 | 7,900 |
| **Differential count (%)** | | | |
| Neutrophils | 40 - 70 | 81 | 45 |
| Lymphocytes | 22 - 44 | 16 | 26 |
| Monocytes | 4 - 11 | 3 | 5 |
| Eosinophils | 0 - 5 | 0 | 1 |
| Basophils | 0 - 2 | 0 | 0 |
| Eosinophil count | 40 - 440 | 50 | 50 |
| Platelet count (per $mm^3$) | 150,000 - 400,000 | 310,000 | 310,000 |
| Activated partial thromboplastin time (sec) | 22.1 - 34.0 | 29 | 29 |
| Prothrombin time (sec) | 10.3 - 13.2 | 11.6 | 11.6 |
| International normalized ratio | - | 1 | 1.01 |
| Urea Nitrogen (mg/dl) | 8 - 25 | 22 | 18 |
| Creatinine (mg/dl) | 0.60 - 1.50 | 0.8 | 0.8 |
| Glucose (mg/dl) | 70 - 110 | 84 | 89 |
| Bilirubin (mg/dl) | | | |
| Total | 0.0 - 1.0 | 0.7 | - |
| Direct | 0.0 - 0.4 | 0.1 | - |
| Protein (g/dl) | | | |
| Total | 6.0 - 8.3 | 6.9 | 6.9 |
| Albumin | 3.3 - 5.0 | 4.2 | 4.3 |
| Alanine aminotranspherase (u/liter) | 7 - 30 | 18 | 18 |
| Aspertate amino - transpherase (u/liter) | 9 - 32 | 24 | 40 |
| Alkaline phosphatase (u/liter) | 30 - 100 | 75 | 75 |
| Lactate dehydrogenase (u/liter) | 110 - 210 | 220 | 310 |
| Sodium (mEq/L) | 135 - 145 | 128 | 137 |
| Calcium (mg/dl) | 8.5 - 10.5 | 9 | 9.1 |
| Potassium (mEq/L) | 3.5 - 5.0 | 4 | 4.1 |
| Copper (ug/dl) | 70 - 155 | 111 | 111 |
| Phosphorus (mg/dl) | 2.6 - 4.5 | 3.4 | 3.4 |
| Magnesium (mmol/liter) | 0.7 - 1.0 | 0.8 | 0.9 |
| Myoglobin (ug/Liter) | 0 - 60 | 32 | 170 |
| Creatinine Kinase (U/Liter) | 40 - 150 | 147 | 240 |
| Troponin I (NG/ML) | 0 - 0.4 | 0.4 | 4.2 |
| Lactate (mmol/liter) | 0.5 - 2.2 | 1.7 | 1.7 |
| Thyroid stimulating hormone (mlU/L) | 0.5 - 5.0 | 2.4 | - |
| Free T3 | 1.8 - 4.7 | 3 | - |
| Free T4 | 0.71 - 1.85 | 1.1 | - |
| Serum Ferritin (ng/ml) | 15 - 200 | 110 | - |
| TIBC (umol/L) | 44.8 - 71.6 | 51.5 | - |
| ESR | 0 - 20 | 60 | 35 |
| Anti-nuclear antibodies | Negative | Negative | Negative |
| Antineutrophil cytoplasmic antibodies | Negative | Negative | Negative |
| Rheumatoid Factor | Negative | Negative | Negative |
| Catecholamines metabolites in blood: | | | |
| Metanephrine (nmol/L) | < 0.50 | - | 13.6 |
| Normetanephrine (nmol/L) | < 0.90 | - | 22.5 |
| Brain - Natriuretic Peptide (pg/ml) | < 100 | 930 | 1374 |
| Serum serotonin level (ng/ml) | 101 - 283 | - | 1411 |

***Table 1:*** *Laboratory data.*

ECG (Figure 1) revealed supra-ventricular tachycardia at rate 180 beat per minute.

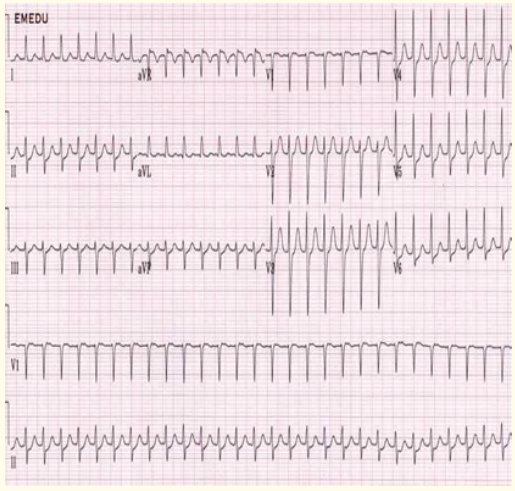



***Figure 1***

Chest X-ray (Figure 2) revealed enlarged cardiac silhouette and increased bronco-vascular markings.

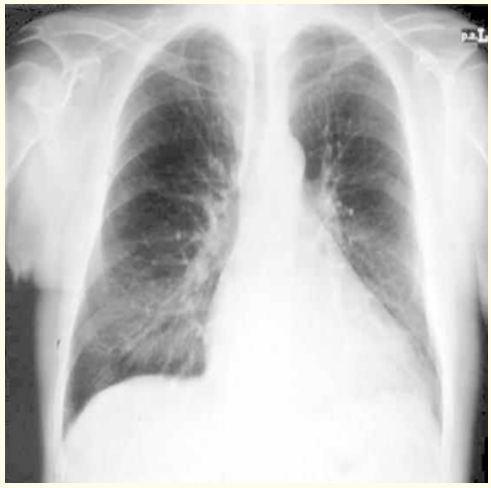
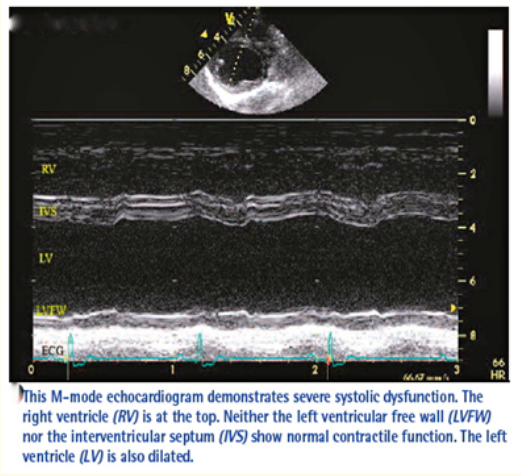


***Figure 2***

Echo-cardiography (Figure 3) revealed dilated left ventricular internal dimensions with estimated ejection fraction 35%, global hypo-kinesia and severe mitral regurgitation.




This M-mode echocardiogram demonstrates severe systolic dysfunction. The right ventricle *(RV)* is at the top. Neither the left ventricular free wall *(LVFW)* nor the interventricular septum *(IVS)* show normal contractile function. The left ventricle *(LV)* is also dilated.

***Figure 3***

Patient was admitted and induction of vomiting was done by the patient herself and she was cardioverted successfully to sinus rhythm with heart rate 95 beat per minute, ECG revealed sinus rhythm with no abnormalities rather than sinus tachycardia.

Augmentin 1gm twice daily was given with antipyretics and mucolytics for chest infection, also patient received complete anti-failure treatment.

After 3 days no fever and chest condition improved. Patient was discharged on anti-failure medical treatment and was diagnosed as tachycardia induced cardiomyopathy.

After one month patient came for follow up with no complain and examination was completely normal, echocardiography was repeated, it was normal with good left ventricular systolic function, normal dimensions and no mitral regurgitation (Figure 4). During proper history taking, patient mentioned that in the previous visit she was having moderate psychological stressors which she didn't mentioned in the first visit. Anti-failure treatment was stopped and patient has no more complain, for follow up, she was referred to a psychiatrist who mentioned that she is not suffering from phycological disease.

After 4 months patient returned to our hospital again complaining of shortness of breath, myalgias and fatigue. This time she has no fever, night sweats, upper respiratory tract symptoms, cough, nausea, vomiting, diarrhea, rash, arthralgias, or urinary tract symptoms. The temperature was 36c, the blood pressure 110/70 mmHg, the pulse 100 beat per minute (regular, equal on both sides and of good volume), the respiratory rate 20 breaths per minute, the oxygen saturation 97% on room air. On examination no abnormalities was detected. ECG (Figure 5) revealed ST-segment elevation and deep T-wave inversion in the anterior leads, in association with prolonged QT interval. Chest X-ray revealed enlarged cardiac silhouette. Echocardiography was repeated and showed the same findings shown in the first visit, dilated LV internal dimensions and estimated ejection fraction 35% with global hypokinesia and severe mitral regurgitation. Laboratory results in table 1, showed mild elevation in cardiac enzymes, highly elevated catecholamines in blood, BNP, serotonin and no other abnormalities in her laboratory results. Abdominal CT was normal, Coronary angiography was done revealed normal coronary arteries. Patient mentioned that this complain is accompanied with stress-full conditions and she refused to take medical treatment and mentioned that this is a psychological problem. She was discharged upon her request.

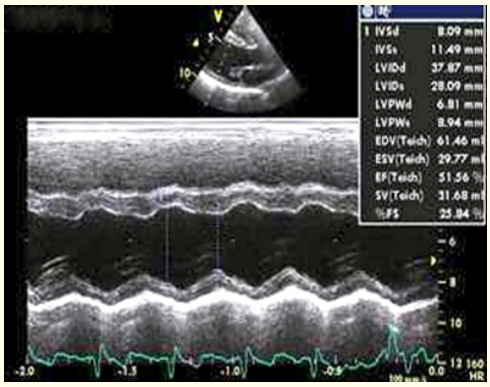



***Figure 4***

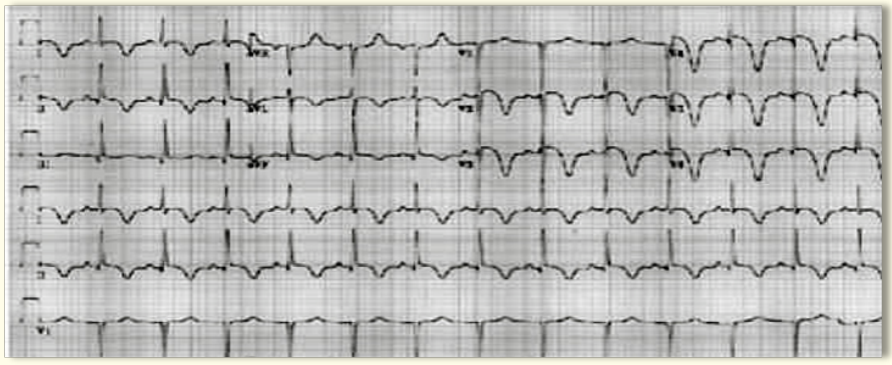


***Figure 5***

After one month patient returned with a healthy mood and she had normal echocardiogram findings and normal ECG.

**Diagnosis:** Recurrent broken heart syndrome of atypical presentation.

## Discussion

A key clinical finding in this case was the recurrence of the disease despite recurrence is rare in broken heart syndrome, further investigations as coronary angiography and follow up echocardiography along with proper history taking and clinical follow up helped a lot, other key was the patient herself, awareness of her condition and symptoms. The use of systematic approach in determining the possible causes, consideration of major clinical findings to rule out other causes of heart failure in our case and diagnosis by exclusion remain a beneficial clinical pathway.

According to the management approach this is a case of dilated cardiomyopathy but what is the cause? Common causes of dilated cardiomyopathy demonstrated in table 2 were in mind but on the other hand, broken heart syndrome or takotsubo cardiomyopathy was

suspected from the second visit and was encouraged by the laboratory results and ECG findings with the normal coronary angiography in the second visit.

**Known Causes of Dilated Cardiomyopathy**

**Ischemia***

**Toxins**
- Ethanol*
- Cocaine*
- Amphetamines*
- Cobalt*
- Lead*
- Mercury*
- Carbon monoxide*
- Beryllium

**Medications**
- Chemotherapeutic agents
  - Doxorubicin
  - Bleomycin
  - 5-Fluorouracil
- Antiretroviral drugs
  - Zidovudine*
  - Didanosine*
  - Zalcitabine*
- Phenothiazines*
- Chloroquine
- Radiation

**Miscellaneous**
- Peripartum cardiomyopathy*
- Tachycardia*
- Sarcoidosis*
- Familial cardiomyopathies
- Sleep apnea*
- Autoimmune myocarditis
- Calcium overload
- Oxygen free radical damage

*Potentially reversible causes

**Nutritional deficiencies**
- Thiamine*
- Selenium*
- Carnitine*

**Electrolyte abnormalities**
- Hypocalcemia*
- Hypophosphatemia*
- Uremia*

**Endocrinologic disorders**
- Thyroid hormone excess or deficiency*
- Growth hormone excess or deficiency*
- Pheochromocytoma*
- Diabetes mellitus
- Cushing's disease

**Neuromuscular diseases**
- Duchenne's muscular dystrophy
- Myotonic dystrophy
- Friedreich's ataxia
- and others

**Rheumatologic diseases**
- Systemic lupus*
- Scleroderma*
- Giant cell artereitis*

**Infectious diseases**
- Viral
  - Coxsackie virus
  - Cytomegalovirus*
  - HIV
  - Varicella
  - Hepatitis
  - Epstein-Barr
  - Echovirus
  - Other
- Bacterial
  - Streptococci-rheumatic fever
  - Typhoid fever
  - Diphtheria*
  - Brucellosis
  - Psitticosis
- Rickettsial disease
- Lyme disease*
- Mycobacterial-fungal
  - Histoplasmosis
  - Cryptococcosis
- Parasitic
  - Toxoplasmosis*
  - Trypanosomiasis
  - Shistosomiasis
  - Trichinosis

**Deposition diseases**
- Hemochromatosis*
- Amyloidosis

***Table 2:*** *Reversible causes of dilated cardiomyopathy [1].*

Tachycardia induced cardiomyopathy was considered a likely diagnosis, given that the patient had history of recurrent supra-ventricular tachycardia for a long time but it was ruled out by the rapid improvement at the first time and recurrence with complete recovery at the second time without medical treatment along with finding other most likely diagnosis, broken heart syndrome.

The history of severe chest infection at the first visit in our case pointed to an alternative diagnosis, myocarditis, but rapid improvement of chest condition on antibiotics and antipyretics in the third day along with the complete scenario in the follow up visits which shown rapid recovery and laboratory results had made it a less likely diagnosis. Bybee KA., *et al*. [2] demonstrated patients with stress-induced cardiomyopathy can present similarly to those with acute myocarditis. A history of an acute emotional or physical stressor within the prior 2 weeks with a rather rapid clinical deterioration supports a diagnosis of stress-induced cardiomyopathy. Rapid recovery back to baseline within 4 to 8 weeks is the rule for stress-induced cardiomyopathy, the occurrence of which supports its diagnosis.

Broken heart syndrome, takotsubo cardiomyopathy or stress induced cardiomyopathy was our diagnosis in-spite the case carries an atypical presentation of the disease. As mentioned by Traulle's., *et al*. [3] in their study of 14 cases of broken heart syndrome, the ECG was pathological in all cases, serum markers routinely showed elevated troponin Ic, with a peak of 2.9 +/- 1.5 ng/ml. This was an arm of challenge as our patient presented to our hospital late after onset of symptoms with normal cardiac enzymes in the first visit, may be cardiac

enzymes was typical slightly high and returned to normal values before admission to our hospital. This issue was much more clear in the second presentation of our case, the patient ECG findings and cardiac enzymes results was typical findings of broken heart syndrome.

In Hopkins study, Ilan S Wittstein., *et al*. [4] said, the research team found that some may respond to sudden, overwhelming emotional stress by releasing large amounts of catecholamines into the blood stream, these chemicals can be temporarily toxic to the heart, effectively stunning the muscle and producing symptoms typical to heart attack, including chest pain, fluid in the lungs, shortness of breath and heart failure. Most of cases are middle aged or elderly women. "These cases were, initially, difficult to explain because most of them were previously healthy and had few risk factors for heart disease". The researchers found that initial levels of catecholamines metabolites, such as metanephrines and normetanephrines in the stress cardiomyopathy patients were two to three times the levels among patients with classic heart attack, and seven to 34 times normal levels as were other stress-related proteins, such as neuropeptide Y, brain natriuretic peptide and serotonin. Moreover, these observations were supportive that the syndrome was stress induced. These data was going on with our findings which encourages our diagnosis.

A hallmark feature of takotsubo cardiomyopathy was the heart's echocardiogram, or ultrasound. While the base of the heart's main pumping chamber, left ventricle, contracted normally, there was weakened contraction in the middle portions of the muscle [4]. Moreover, as mentioned by the American society of echocardiography concerning broken heart syndrome, echocardiography shows characteristic abnormalities of squeezing of the left ventricle where the bottom portion of the heart called apex balloons out during contraction. However typically resolve after two to four weeks and follow up echocardiograms show return to normal function.

Blessing E., *et al*. [5] mentioned, recent case reports have described takotsubo cardiomyopathy associated with suppression of basal contraction and apical sparing, a kind of "inverted takotsubo". Kurowski., *et al*. [6] found that ≈40% of patients with transient ventricular dysfunction had this atypical pattern. We also have reported this case of recurrent takotsubo cardiomyopathy with a different contractile pattern [5].

Yoshihiro J., *et al*. [7] mentioned ischemia resulting from multi-vessel coronary vasospasm seems unlikely in these cases because it would not be expected to be associated with apical sparing. Various patterns of left ventricular wall motion abnormalities therefore seem possible in takotsubo cardiomyopathy, with transient left ventricular apical ballooning being more common and other regional wall motion abnormalities less common. Further research in both clinical and preclinical settings is required to determine whether individual differences in patterns of wall motion abnormalities are related to local differences in patterns of excessive sympathetic activity or in responses to sympathetic stimulation.

Parodi., *et al*. [8] demonstrated that significant mitral regurgitation (defined as moderate to severe mitral regurgitation) was present in only 21% of their cases. Also, Dariusch Haghi., *et al*. [9] found that severe mitral regurgitation was found in only four out of 47 cases of broken heart syndrome in their study.

The echocardiogram of our case was challenging, it didn't show the typical picture of apical ballooning with sparing of basal segments, as our case revealed atypical picture of global hypokinesia along with severe mitral regurgitation.

## Conclusion

Broken Heart Syndrome has a high rate of fast recovery and not always showing a single typical presentation or typical findings and it may sometimes be diagnosed by exclusion. Our case was presented late after symptoms onset with other co-morbidities which made it challenging in diagnosis. It was atypical presentation of takotsubo cardiomyopathy as referred to echocardiogram findings and recurrence; which is rare in this disease. Although takotsubo cardiomyopathy has been progressively better characterized, certain aspects remain to be clarified, and it is still under study.

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**Volume 10 Issue 5 May 2023**