Case Report: Takotsubo syndrome in a postoperative patient without cardiological disease [version 2; peer review: 1 approved, 1 approved with reservations, 1 not approved]

Previously titled 'Case Report: Takotsubo cardiomyopathy in a postoperative patient without cardiological disease'

Luis Coaguila-Cusicanqui¹, Vanessa Castillo-Atoche¹, Roberto Montalvo-Suyon¹, Yuriko Cavero-Reyes¹, Virgilio E. Failoc-Rojas¹².³

¹Hospital Regional Lambayeque, Lambayeque, 14013, Peru
²Universidad Privada Norbert Wiener, Lima, 15079, Peru
³Instituto de Evaluación de Tecnologías en Salud e Investigación, EsSalud, Lima, 15079, Peru

Abstract

**Background:** Takotsubo syndrome (TS) is characterized by a clinical presentation that mimics acute coronary syndrome but is reversible. Alterations of Takotsubo in patients without previous heart disease remain a challenge for diagnosis.

**Case report:** We present a case of an 80-year-old patient from Peru. The patient underwent surgery, with the diagnosis of Chilaiditi's syndrome. One day after surgery, she presented with dyspnea, tachycardia, and electrocardiographic changes (paroxysmal atrial fibrillation and infrequent ventricular extrasystoles without abnormalities in the ST-segment). The diagnosis of Takotsubo syndrome with cardiogenic shock was made. She was hospitalized in the Intensive Care Unit and was managed with vasopressors and nitroglycerin. There was no cardiac lesion in the cineangiogram or occlusion of arteries. The patient was extubated and received daily dialysis until discharge.

**Conclusions:** Takotsubo is an emotional, non-cardiac, or post-traumatic stressful event that triggers myocardial injury with segmental anomalous, the possible etiology of which is the release of an endothelial neurotransmitter caused by stress. Emergency physicians should be aware of this as even patients without previous cardiac pathologies when exposed to stressors (such as surgeries) develop emergency symptomatology similar to myocardial infarction. Thus, emergency physicians should identify any cardiac abnormalities after a stressor, as well as be prepared for the diagnosis of TS.
Keywords
Takotsubo cardiomyopathy, electrocardiography, Peru

Corresponding author: Virgilio E. Failoc-Rojas (virgiliofr@gmail.com)

Author roles: 
Coaguila-Cusicanqui L: Conceptualization, Data Curation, Investigation, Project Administration, Writing – Original Draft Preparation, Writing – Review & Editing; Castillo-Atoche V: Conceptualization, Data Curation, Investigation, Methodology, Project Administration, Writing – Original Draft Preparation, Writing – Review & Editing; Montalvo-Suyon R: Conceptualization, Investigation, Methodology, Validation, Writing – Original Draft Preparation, Writing – Review & Editing; Cavero-Reyes Y: Conceptualization, Data Curation, Methodology, Writing – Original Draft Preparation, Writing – Review & Editing; Failoc-Rojas VE: Conceptualization, Data Curation, Investigation, Methodology, Validation, Writing – Original Draft Preparation, Writing – Review & Editing

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Introduction

Takotsubo syndrome (TS), also known as stress cardiomyopathy, apical-ballooning syndrome, or broken heart syndrome, is characterized by a clinical presentation that mimics acute coronary syndrome (ACS). The estimated prevalence of TS is between 2% and 3% in patients with suspected ACS, but it can be higher reaching up to 10% in women. TS presents a transient regional systolic dysfunction of the left ventricle that is extended beyond the coronary artery supplying area and seems to follow the anatomic cardiac sympathetic innervation. This mimics an ACS (myocardial infarction with ST-segment elevation) or unstable angina.

The most frequent symptoms are chest pain, dyspnea, or syncope (75.9%, 46.9%, and 7.7%, respectively) according to the International Takotsubo Registry. There are still uncertainties concerning its pathophysiology, diagnosis, and treatment.

This case report is about an elderly woman who was admitted to the hospital for surgery, but in the post-operative phase, she presented with a complication similar to ACS, which was later diagnosed as TS. Here, we report the clinical symptoms and how the patient’s condition improved.

Case report

We present a case of an unemployed 80-year-old female patient from Cajamarca, Peru with a history of arterial hypertension and type 2 diabetes for the last 10 years by which she has followed irregular treatment. She was admitted by emergency to a hospital in Lambayeque, in the North West of Peru. On admission, the patient complained of stabbing abdominal pain in the right hemiabdomen, nausia, and constant vomiting after suffering a fall from approximately one meter. Once in the emergency department, she was evaluated by the surgery service that made the diagnosis of closed abdominal trauma and Chilaiditi’s syndrome and she was taken to the operating room to undergo surgery. Findings on the laparotomy showed hepatic injury, with a portion of the colon abnormally interposed between the liver and the diaphragm. A day after admission for surgery, she was taken to the recovery room. There, she started presenting with moderate dyspnea and tachycardia. After performing a Holter electrocardiogram (ECG), sinus rhythm, paroxysmal atrial fibrillation, and infrequent ventricular extrasystoles without abnormalities in the ST-segment were found. Echocardiography was also carried out, which revealed that the patient presented 75% preserved left ventricular ejection fraction (LVEF) with preserved motility, no hypertrophy or dilation of cavities, and type 1 diastolic dysfunction. During the first five days after admission, the patient continued presenting dyspnea, which was aggravating until it became severe with added oliguria. This was a diagnostic challenge because the description on the ECG suggested ACS or some other cardiac pathology (e.g., dilated, restrictive, or hypertrophic cardiomyopathy, which were previously ruled out due to normal conserved structure in echocardiography). Seven days after admission for emergency and three days after surgery, she was admitted to the Intensive Care Unit (ICU) with a thrombolysis in myocardial infarction (TIMI) risk score of nine (probability of mortality of 35.9% over 30 days). In the ICU, the patient started with severe respiratory insufficiency, marked hypotension, and anuria, requiring intubation and ventilation support, hemodynamic support with vasopressor therapy (norepinephrine by continuous intravenous infusion at a dose of 0.12 and 0.04 μg/kg/min each day), and dialysis support.

The day after she was admitted to the ICU (post-operative day four), the patient presented with atrial fibrillation with a high ventricular response; then, an amiodarone infusion was administered reverting the dysrhythmia. At the moment of the dysrhythmia, serial ECG was performed in which marked negative T-waves in II, III, and augmented vector foot (aVF) leads, and all precordial leads were observed (Figure 1). Neither ST-segment disorder nor pathologic Q-waves
were present. For this reason, the cardiology department performed echocardiography for the second time (post-operative
day eight) where 44% LVEF systolic dysfunction, dilated left ventricle, mild mitral insufficiency, and severe aortic
insufficiency with marked apical dyskinesia and contraction of basal segments were evidenced. In the cineangiogram,
neither lesions nor occlusion were found in the trunk of the left coronary artery, anterior descending artery, circumflex
artery, or right coronary artery (Figure 2).

Suspecting ACS, anti-ischemic therapy was begun with digoxin 0.125 mg per day and cardiac enzymes were required,
which resulted in an elevation of troponin T (0.35 ng/ml) and troponin I (0.55 ng/ml). The creatine phosphokinase
(CK-MB) value was 30.8 IU/L. Considering these results, coronary angiography was performed in which no coronary
lesions were evidenced. NT-proBNP had a value greater than 35,000 pg/ml.

The diagnosis of Takotsubo syndrome with cardiogenic shock was made. An added septic process was ruled out with
cultures and procalcitonin, which were negative. To complete the diagnostic study, abdominal transmission electron

Figure 1. A: Negative T waves, in II, III and aVF. B: Nine days after treatment, with resolution of negative T waves. aVF, augmented vector foot; aVR, augmented vector right; aVL, augmented vector left.

Figure 2. Cardiac characterization of the patient showing permeable coronary arteries with apical dyskinesia.
microscopy (TEM) and renal echography were conducted, ruling out a neoplastic process, pheochromocytoma, or any other spreading process.

A total of 10 days after admission to the ICU (post operative day 13), the patient ceased presenting shock; so, the vasopressor infusion was discontinued (norepinephrine by continuous intravenous infusion at a dose of 0.12 and 004 μg/kg/min, which she received for two days). It should be noted that dobutamine by continuous intravenous infusion was also administered, but only for 24 hours (a dose of 5 μg/kg/min); then it was discontinued because the patient started presenting hypotension and tachyarrhythmia.

A total of 13 days after admission to the ICU (post operative day 16), continuous intravenous infusion of nitroglycerin was given at a dose of 20–40 mcg/min, which was administered for 24 hours to control coronary vasospasm. No evidence of systolic dysfunction (LVEF: 65%) was observed in control echocardiography during the infusion of nitroglycerin; motility was preserved with type 1 diastolic dysfunction, with mild aortic and mitral insufficiency, and no apical dyskinesia. On echocardiographic findings, up to the date of reversion of the clinical picture of marked systolic dysfunction, negative T-waves persisted without evidence of ST-disorder or pathological Q-waves.

Infusion of nitroglycerin was discontinued by evidence of clinical improvement. Therefore, the patient continued treatment with atorvastatin (sublingual, 40 mg/day), enoxaparin (subcutaneous injection, 40 mg/day), digoxin (intravenous, 0.125 mg/day), and daily dialysis support.

The patient was removed from mechanical ventilation, being extubated 15 days after hospitalization in the ICU (post operative day 18). She continued with non-invasive ventilator assistance for a further three days. Then, she was continued with low flow oxygen therapy with FIO2 0.30 via nasal cannula. After that, the patient was discharged from the Internal Medicine service with daily dialysis support because of persistent anuria. Before discharge, pheochromocytoma, intracranial involvement, previous ischemic heart disease and severe organic valvular heart disease could be excluded. The patient progressed favorably until discharge.

At three months of follow-up, the patient did not present any hospital readmission, preserved systolic function (LVEF: 62%), and no other cardiac abnormalities. However, the patient eventually reported precordial pain without becoming disabling.

Discussion

TS implies a diagnostic challenge because the initial clinical picture may be difficult to differentiate from ACS. TS is not uncommon but there are not many reports in Peru, perhaps due to the lack of adequate clinical suspicion.

It has been observed that TS affects women (85–90%) aged between 65 and 70 years old more often than men, which is similar to what was observed in the present clinical case. This could be explained by the fact that estrogens regulate the myocardial sympathetic tone and vascularization in women of reproductive age. This sympatholytic effect of estrogens is lost after menopause, leading to an effect in the abnormality of the contraction of the left ventricle of TS. Other causes have been related to the higher frequency of emotional or physical stress. TS can also appear in young women, children, and newborns.

The etiology of Takotsubo is still uncertain but may be associated with catecholamine elevations during times of emotional or physical stress, and we believe that a post-surgical catecholamine overload and a brain-heart connection hypothesis may have caused the Takotsubo syndrome in this case report, without the need for prior cardiac pathology. It is proposed that when faced with an unexpected and severe stress response, the autonomic nervous system synthesizes sympathetic neuronal exits and adrenomedullary hormones. The epinephrine released from the adrenal medulla and the norepinephrine from the cardiac and extracardiac sympathetic nerves reach the adrenoceptors in the blood vessels and the heart. In this report the patient did not have previous cardiac complications, so this support would be based on the release of catecholamines due to stress that has an expression in the cardiac receptors.

The diagnosis was mainly made by addressing one of the four criteria of Mayo Clinic for the diagnosis of TS: 1) mid and apical dyskinesia of the left ventricular segments with regional wall motion abnormalities; 2) absence of obstructive coronary artery disease; 3) appearance of new ECG abnormalities (T-wave inversion); and 4) absence of pheochromocytoma or myocarditis. Additionally, elevated troponin and proBNP levels were found in this case, which is frequent in patients with TS reported in international registries. Therefore, it is also considered a diagnostic criterion. However, the frequency of TS in patients with ACS is often underestimated since there is not yet full knowledge of this disease in the context of perioperative myocardial injury. It is recommended that physicians also consider the differential diagnosis in a
patient admitted to the hospital with ACS and evaluate the electrocardiogram, echocardiography, and early invasive coronary angiography to rule out other possible causes of cardiomyopathy (e.g., dilated, hypertrophic, and restrictive) and ACS.

The patient was treated with norepinephrine and dobutamine at standard doses considering their significant hypotensive status. Acute stages of TS present with impaired vasodilatation and extravasation of fluid from the vasculature to the interstitial space. However, considering a cardiogenic problem, administration of the aforementioned drugs may induce additional catecholaminergic stress and precipitate TS. As catecholamines can induce microvascular spasm or cardiac toxicity, the use of nitroglycerin controlled possible coronary insufficiency, although increased tissue sensitivity to nitric oxide is also observed in TS. Despite this, the patient showed no left ventricle outflow obstruction and presented correct evolution. It was also important to consider the complications in TS, which are common. Although lesions in cineangiogram are absent, complications like acute cardiac insufficiency (12–45%), atrial fibrillation (7–17%), cardiogenic shock (17–30%), and dysrhythmia (5–15%) have been reported. Patients with TS have a high risk of developing atrial fibrillation, ranging from 7.75–17.57%. In this report, atrial fibrillation, ventricular extrasystoles, dysrhythmia, ST-segment abnormalities, and aorta and mitral insufficiency are registered. Importantly, atrial fibrillation does not increase in-hospital mortality, but it can lead to higher levels of comorbidities such as ventricular dysrhythmias, longer hospital stays, and the development of cardiac arrest.

The prognosis of TS has been thought to be favorable as their reversible characteristic. However, these patients show considerable morbidity, mortality, and major adverse cardiovascular events similar than patients with ACS. In this case, the patient was discharged from the hospital with clinical improvement. It has been observed that mortality rates of TS are higher in male patients than in female ones (8.4% vs. 3.6%, respectively; p<0.001).

Most deaths occur among patients that develop unstable manifestations, including cardiac arrest or cardiogenic shock. The recovery of the left ventricular contraction is gradual, generally from 1 to 2 weeks although it can be fast (within 48 hours) or late (up to 6 weeks). However, one important problem to solve is the need to determine the risk factors and the pathophysiological mechanisms in this disease, as well as the physiology of the patient already recovered from TS. This would lead to determine, with certainty, the specific care these patients require in the long term.

Conclusions
This report showed that the diagnosis and treatment of TS is complex, although it is a relatively benign and reversible disease. In the setting of an emotional, non-cardiac, or post-traumatic stressful event, TS triggers myocardial injury with segmental anomalous, which are possibly triggered by an stress-released endothelial neurotransmitter. It is important to conduct further research on cardiac and endocrine functions to find out why this disease mostly affects women. Higher methodological quality studies should be also performed to determine the best therapeutic option for these patients.

Data availability
Underlying data
All data underlying the results are available as part of the article and no additional source data are required.

Consent
Written informed consent for publication of their clinical details and clinical images was obtained from the patient.

References


The authors have not revised the article to a satisfactory standard based on my previous report. As such I cannot recommend this article for indexing. My comments read below:

1. The critically important aspect of this woman’s case was the poor treatment of hypotension, to an extent whereby she needed dialysis. Not much is said about the cause of the hypotension, but as I indicated it is likely to be induced by inappropriate vasodilatation due to excessive (not impaired) responsiveness to nitric oxide, as well as inflammatory vascular leakage, and should certainly have been treated with volume expansion rather than catecholamines and nitroglycerine.

2. The idea that TTS is “transient” is mentioned 3 times in the revised text, despite extensive evidence that recovery is at best slow, that it is often incomplete, and that there is a substantial risk of both recurrence and of long-term mortality.

3. The authors do not appear to have properly researched the pathogenesis of TTS, simply saying that it is “not understood”. However, there are data on biased signalling at beta-2 adrenoceptors, induction of nitrosative stress, and of myocardial energetic impairment, none of these quoted.

4. The patient did not receive long-term ACE inhibitors, despite evidence that these improve outlook long-term, nor is this mentioned.

5. Nothing is said about the utility of cardiac MRI for establishing the diagnosis positively, rather than leaving it as a diagnosis of exclusion.

6. Nothing is said about the association with cancers.

7. The infusion rate of nitroglycerine was inappropriately high and would have resulted in loss of nitroglycerine effect rapidly\(^1\). Perhaps a good thing.
8. The patient follow-up does not indicate long-term monitoring of resolution of NTproBNP levels, nor of normalisation of global longitudinal strain patterns on echocardiography, the normal gold standards.

References

Is the background of the case's history and progression described in sufficient detail?
Partly

Are enough details provided of any physical examination and diagnostic tests, treatment given and outcomes?
No

Is sufficient discussion included of the importance of the findings and their relevance to future understanding of disease processes, diagnosis or treatment?
No

Is the case presented with sufficient detail to be useful for other practitioners?
No

*Competing Interests:* No competing interests were disclosed.

I confirm that I have read this submission and believe that I have an appropriate level of expertise to state that I do not consider it to be of an acceptable scientific standard, for reasons outlined above.
This report "Case Report: Takotsubo cardiomyopathy in a postoperative patient without cardiological disease" is well written, and this reviewer understands what the authors tried to review with their unique case.

Points of review:
1. Describe the findings of the laparotomy. Chilaiditi's syndrome does not require a surgical treatment.
2. Add the information of cardiac enzyme (CK-MB).
3. Nitroglycerine is not appropriate to treat TC. Show the reason why authors applied this treatment.
4. Discussion is incoherent, each paragraph should include the topics authors would like to emphasize. The mixing of short and long paragraphs makes following the right discussion points difficult. Authors just addressed the knowledge which is already known in previous reports. Readers would like to know academic aspects from this case. To improve the quality of this case report, this reviewer recommends authors to reconsider the concept of this case.

Also, I recommend the manuscript be proofread.

Is the background of the case's history and progression described in sufficient detail? Yes

Are enough details provided of any physical examination and diagnostic tests, treatment given and outcomes? Yes

Is sufficient discussion included of the importance of the findings and their relevance to future understanding of disease processes, diagnosis or treatment? No

Is the case presented with sufficient detail to be useful for other practitioners? Yes

Competing Interests: No competing interests were disclosed.

Reviewer Expertise: Emergency medicine, Trauma, Intensive care

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard, however I have significant reservations, as outlined above.
Virgilio E Failoc-Rojas, Universidad Privada Norbert Wiener, Lima, Peru

Response to Reviewer 3

1. Describe the findings of the laparotomy. Chilaiditi’s syndrome does not require a surgical treatment.

Response: Thank you. We have described the findings of laparotomy. Indeed, Chilaiditi’s syndrome generally has conservative management. However, the main diagnosis was closed abdominal trauma, which did require immediate surgery.

1. Add the information of cardiac enzyme (CK-MB).

Response: Thank you. We have added the information of cardiac enzyme.

1. Nitroglycerine is not appropriate to treat TC. Show the reason why authors applied this treatment.

Response: Thank you. We have provided the reason why nitroglycerine was used.

1. Discussion: each paragraph should include the topics authors would like to emphasize. The mixing of short and long paragraphs makes following the right discussion points difficult.

Response: Thank you for your feedback. This section was revised accordingly.

1. Authors just addressed the knowledge which is already known in previous reports. Readers would like to know academic aspects from this case. To improve the quality of this case report, this reviewer recommends authors to reconsider the concept of this case.

Response: Thank you. We have provided more information on academic aspects from the case. The message of this case was reevaluated, and it was reinforced in the conclusion.

1. Also, I recommend the manuscript be proofread.

Response: Thank you. The manuscript was proofread as suggested.

Competing Interests: We declare no competing interests.

Reviewer Report 18 July 2022

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John D. Horowitz
Freemasons Centre for Male Health and Wellbeing, South Australian Health and Medical Research Institute, School of Medicine, The University of Adelaide, Adelaide, SA, Australia

This is a case report of a patient whose takotsubo syndrome (TTS: note the name) was diagnosed after an intra-abdominal operation on an elderly woman. There is a great deal wrong with the report, and I will try to cover some of it:-

1. It is not mentioned what were the findings at laparotomy. Was there, in fact, any intra-abdominal pathology?? If so, this is secondary TTS: if not, it is likely that the abdominal pain
reflected the onset of TTS of primary type.

2. The clinical details are deficient. It is not mentioned specifically, but I presume that the patient was seriously hypotensive. This occurs mainly because in the acute stages of TTS, there is inappropriate vasodilatation and extravasation of fluid from the vasculature to the interstitium. This was not mentioned. The patient was treated with pressor agents, despite the fact that TTS is caused by an aberrant response to released catecholamines, and by infused nitroglycerine, despite the fact that there is already increased tissue sensitivity to nitric oxide, together with a propensity towards nitrosative stress. The correct consideration would have been fluid administration as primary care: this would have averted renal functional deterioration.

3. TTS is neither a cardiomyopathy nor a transient disorder. It is inflammatory within the myocardium, transiting to energetic impairment, and eventually variable fibrosis. This was entirely omitted. The long-term prognosis is none too good either, partially because of an association with cancer. None of this was stated.

4. NT-proBNP is not a natriuretic peptide: it is actually inert. 35,000 represents the upper limit of most commercial assays: I suspect that the notation should be > 35,000.

5. The case is particularly relevant because it should have stimulated discussion on the nature of peri-operative myocardial injury in general: as the authors should know, this area remains controversial and tainted by (admitted) data falsification. There is increasing evidence that many of these cases are TTS rather than infarction. None of this was mentioned.

Is the background of the case's history and progression described in sufficient detail?
No

Are enough details provided of any physical examination and diagnostic tests, treatment given and outcomes?
No

Is sufficient discussion included of the importance of the findings and their relevance to future understanding of disease processes, diagnosis or treatment?
No

Is the case presented with sufficient detail to be useful for other practitioners?
No

Competing Interests: No competing interests were disclosed.

I confirm that I have read this submission and believe that I have an appropriate level of expertise to state that I do not consider it to be of an acceptable scientific standard, for reasons outlined above.
Response to Reviewer 2

1. It is not mentioned what were the findings at laparotomy. Was there, in fact, any intra-abdominal pathology?? If so, this is secondary TTS: if not, it is likely that the abdominal pain reflected the onset of TTS of primary type.

Response: Thank you. Findings on laparotomy were provided. As suggested by the report, TS was assumed to have appeared after surgery.

1. The clinical details are deficient. It is not mentioned specifically, but I presume that the patient was seriously hypotensive. This occurs mainly because in the acute stages of TTS, there is inappropriate vasodilatation and extravasation of fluid from the vasculature to the interstitium. This was not mentioned. The patient was treated with pressor agents, despite the fact that TTS is caused by an aberrant response to released catecholamines, and by infused nitroglycerine, despite the fact that there is already increased tissue sensitivity to nitric oxide, together with a propensity towards nitrosative stress. The correct consideration would have been fluid administration as primary care: this would have averted renal functional deterioration.

Response: Thank you for your comment. Hypotension was mentioned in the last sentence of the first paragraph in Case Report section. We agree on the suggested management of TS, we have noted this in the Discussion.

1. TTS is neither a cardiomyopathy nor a transient disorder. It is inflammatory within the myocardium, transiting to energetic impairment, and eventually variable fibrosis. This was entirely omitted. The long-term prognosis is none too good either, partially because of an association with cancer. None of this was stated.

Response: Thank you. As far as we know, the etiology and pathophysiology of TS remains poorly understood to date, reason for which there is still no consensus on the terminology to date [https://www.ncbi.nlm.nih.gov/pmc/articles/PMC7612566/]. However, we have followed the recent publications and decided to revise the term for Takotsubo syndrome. Also, we have revised the information on the prognosis as suggested.

1. NT-proBNP is not a natriuretic peptide: it is actually inert.35,000 represents the upper limit of most commercial assays: I suspect that the notation should be > 35,000.

Response: Thank you. We have revised the notation.

1. The case is particularly relevant because it should have stimulated discussion on the nature of peri-operative myocardial injury in general: as the authors should know, this area remains controversial and tainted by (admitted) data falsification. There is increasing evidence that many of these cases are TTS rather than infarction. None of this was mentioned.

Response: Thank you. We have dedicated this comment in a part of the discussion on diagnosis.

Competing Interests: We declare no competing interests.
Carlos Culquichicon  
Fred Hutch, Seattle, WA, USA

This case report presented a patient attending the emergency room due to an initial abdominal trauma. Days after the surgery, she developed Takotsubo syndrome as a complication but reversed in the discharge.

- Provide summarized details about the "electrocardiographic changes" in the abstract section.
- “The diagnosis of Takotsubo syndrome with cardiogenic shock and renal failure on hemodialysis was made”. It is unclear whether the Takotsubo diagnosis was made during or before hemodialysis.
- In the 3rd paragraph of the discussion section, the authors mention that some other cardiomyopathies were ruled out. It might be good to know which diseases were ruled out and the rationale.
- It would be great if an English-native clinical expert checks the accuracy of medical terminology.

Is the background of the case's history and progression described in sufficient detail?  
Yes

Are enough details provided of any physical examination and diagnostic tests, treatment given and outcomes?  
Yes

Is sufficient discussion included of the importance of the findings and their relevance to future understanding of disease processes, diagnosis or treatment?  
Yes

Is the case presented with sufficient detail to be useful for other practitioners?  
Yes

Competing Interests: No competing interests were disclosed.

Reviewer Expertise: Epidemiological methods

I confirm that I have read this submission and believe that I have an appropriate level of expertise to confirm that it is of an acceptable scientific standard.
Virgilio E Failoc-Rojas, Universidad Privada Norbert Wiener, Lima, Peru

Response to Reviewer 1

- Provide summarized details about the "electrocardiographic changes" in the abstract section.
  **Response: Thank you. We have added the suggested information.**

- "The diagnosis of Takotsubo syndrome with cardiogenic shock and renal failure on hemodialysis was made". It is unclear whether the Takotsubo diagnosis was made during or before hemodialysis.
  **Response: Thank you. We have revised the sentence for clarity.**

- In the 3rd paragraph of the discussion section, the authors mention that some other cardiomyopathies were ruled out. It might be good to know which diseases were ruled out and the rationale.
  **Response: Thank you. We have added the requested diseases and the rationale.**

- It would be great if an English-native clinical expert checks the accuracy of medical terminology.
  **Response: Thank you. The medical terminology was revised as suggested.**

**Competing Interests:** We declare no competing interests.

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