HHS Public Access

Author manuscript

Am J Med Case Rep. Author manuscript; available in PMC 2020 July 23.

Published in final edited form as:

Am J Med Case Rep. 2020 ; 8(10): 350–357.

Apical Takotsubo Cardiomyopathy in a COVID-19 Patient Presenting with Stroke: A Case Report and Pathophysiologic Insights

Pramod Theetha Kariyanna[1], **Harshith Priyan Chandrakumar**[1], **Apoorva Jayarangaiah**[2], **Abdullah Khan**[3], **Volodymyr Vulkanov**[4], **Michael Ashamalla**[1], **Moro O. Salifu**[1], **Samy I. McFarlane**[1,*]

[1]Department of Internal Medicine, State University of New York- Downstate Medical Center, Brooklyn, New York, U.S.A-11203

[2]Division of Hematology and Oncology, Department of Internal Medicine, Albert Einstein College of medicine/Jacobi Medical Center, Bronx, N.Y., U.S.A 10461

[3]Divisions of Cardiology, Department of Internal Medicine, Brookdale University Hospital and Research Center, Brooklyn, New York, U.S.A-11212

[4]Neurology, Department of Internal Medicine, Brookdale University Hospital and Research Canter, Brooklyn, New York, U.S.A-11212

Abstract

COVID-19 is a pandemic that started in Wuhan city, Hubei province in China in December 2019 and is associated with high morbidity and mortality. It is characterized by a heightened inflammatory and prothrombotic state that are known to cause various cardiovascular manifestations such as thromboembolism, acute coronary syndrome and stroke. We here present a 72-year-old woman with multiple cardiovascular risk factors and COVI 19 pneumonia who presented with acute ischemic stroke. She was also noted to have ST segment elevation myocardial infarction (STEMI) on the electrocardiogram however the imaging and clinical presentation was consistent with apical takotsubo cardiomyopathy. We here discuss the various pathophysiologic mechanisms by which COVID-19 can result in acute stroke. The patient likely developed takotsubo cardiomyopathy because of stroke and acute COVID-19 induced sympathetic stimulation and catecholamine surge. To the best of our knowledge this is the first case of apical variant of takotsubo cardiomyopathy in a COVID-19 report.

Keywords

COVID-19; takotsubo cardiomyopathy; stress cardiomyopathy; STEMI; stroke; cerebrovascular accident

*Corresponding author: smcfarlane@downstate.edu.

1. Introduction

COVID-19 (coronavirus disease of 2019) caused by the Severe Acute Respiratory Syndrome Coronavirus 2(SARS CoV-2) that started in Wuhan city, Hubei province in China by December 2019 [1], was officially declared as a pandemic in March by WHO [2]. As of April 24, 2020, according to the Johns Hopkins COVID-19 dashboard, there have been 2,735,117 confirmed cases and 192,019 deaths all over the world with 16,388 only in New york city [3]. Predominantly spread by respiratory droplets, the COVID-19 is similar in morphology to the previous pandemic viruses, namely severe acute respiratory distress syndrome (SARS) and middle eastern respiratory distress syndrome (MERS), but with a high contagious spread [4], COVID-19 could potentiate a prothrombotic state, causing arterial and venous thrombosis [5]. Cases of deep venous thrombosis, pulmonary embolism, ischemic and haemorrhagic stroke have been reported [5,6]. Here we present a 72-year-old female with altered mental status noted to have COVID-19 pneumonia and ST segment elevation myocardial infarction (STEMI) patient who was found to have an ischemic stroke, with a subsequent diagnosis of stress induced cardiomyopathy.

2. Case Presentation

A 72-year-old female with a past medical history of obesity, diabetes, hypertension, hyperlipidemia penicillin allergy was brought into the emergency room as a stroke notification after she was found at her home with altered mental status. Last known well time prior to presentation was 7 hours prior to presentation. National institute of health stroke scale (NIHSS) was 12 at presentation. As per the health care proxy the patient had dry cough and loss of appetite over the past 3-4 days. At presentation temperature was 98.6 degree Fahrenheit, heart rate at presentation was 98 beats per minute, blood pressure was 146/97 mm Hg, respiratory rate was initial in 32 but increased to 40-50’s, and patients saturation decreased from 89 to 56 on rebreather mask hence a decision was made to intubate the patient as she developed acute respiratory failure. Cardiopulmonary and abdominal examination did not reveal any abnormality. Patient had a right sided gaze. Table 1, Table 2, Table 3 and Table 4 summarized the laboratory tests at presentation revealed acute kidney injury and elevated inflammatory markers such as C reactive protein and ferritin. Also Troponin and natriuretic peptide were elevated. Abbott Real Time SARS-CoV-2 PCR assay using M2000 platform was positive for COVID-19 for nasopharyngeal swab. Chest X-ray showed diffuse bilateral infiltrates (Figure 1). Computer tomography of the head at presentation did not reveal acute stroke. Electrocardiography (EKG) revealed normal sinus rhythm, Q waves in V1-V2 leads suggestive of septal infarct and Q waves with ST segment elevation V3,V4,V5 and deep T wave inversion in V6 (Figure 2). Patient did not receive any thrombolytic therapy as she has passed the ideal duration for thrombolytic therapy. A repeat CT head demonstrated a subtle hypoattenuation in the right parietal lobe with loss of gray-white differentiation and sulcal effacement suggestive of acute infarct (Figure 3). A transthoracic echocardiography revealed diffuse hypokinesis with distinct regional wall motion abnormalities. There was apical dyskinesis or apical systolic ballooning suggestive of stress induce cardiomyopathy (takotsubo cardiomyopathy) (Figure 4, Figure 5 and Figure 6). Patient was conservatively managed for stroke with low dose aspirin 81 mg and high intensity statin therapy. Initially at presentation emergent coronary

angiogram was not performed as patients presentation was highly suspicious for stroke and the deep T wave inversions suggestive of an intracranial event. COVID 19 was managed with azithromycin and Plaquenil. Aztreonam and gentamicin was added for possible bacterial infection. Patient subsequently developed cardiogenic shock on day 4 of hospitalization and was started on multiple vasopressors and inotropic agents (vasopressin, dopamine, norepinephrine, epinephrine and dobutamine). She passed away from cardiac asystole despite resuscitative measures.

3. Discussion

COVID-19, caused by the SARS CoV-2 is a novel single stranded enveloped RNA virus belonging to the coronaviridae family. It is believed to have originated from bats, being passed on to an intermediate host (Malayan Pangolin) and then to humans [7]. COVID-19 has been observed to affect the upper and lower respiratory tract, heart, gastrointestinal tract, platelets and skin. A retrospective study reported the most common symptoms being fever (98%), fatigue (70%, dry cough (59%) and other viral flu-like symptoms including myalgia, diarrhea, headache vomiting [8]. The complications included development of acute respiratory distress syndrome, cardiac arrhythmias, septic shock, acute kidney injury, stroke, pulmonary embolism, Disseminated intravascular coagulation (DIC) [8,9,10]. There has also been reporting of fulminant myocarditis [11] and one prior case of Takotsubo cardiomyopathy in the setting of myocarditis [12].

The existence of comorbidities has been increasingly found in COVID-19 patients, with the presence more predominant with severe disease. In a cohort of 191 COVID-19 positive patients in Wuhan, 48% had any comorbidities with hypertension (HTN) seen in 30% and diabetes mellitus (DM) in 19% [13]. A larger cohort of 1099 patients from china confirmed the increased prevalence of HTN, DM, ischemic heart disease, cerebrovascular and renal disease in the severe disease subgroup [9]. A more recent single center study in New York City proved increased comorbidities in the subgroup receiving mechanical ventilation [14]. These patients particularly had a higher incidence of cardiovascular complications like acute myocardial injury denoted by elevated troponins, arrhythmias, heart failure, cardiomyopathy, incidences of cardiogenic shock, coagulopathies as well as thromboembolic events like pulmonary embolism and stroke [5,8,9,13,14,15,16].

The pathophysiologic mechanisms for cardiac manifestations and thrombotic events are not completely understood.

SARS CoV-2 is transmitted through respiratory droplets and direct contact with established community spread [17], with the virus remaining viable in aerosols for at least 3 hours and in plastic surfaces for upto 72 hours. Although the virus has been detected in gastrointestinal tract, saliva and urine, transmission through these routes needs more investigation [18]. Angiotensin converting enzyme 2 (ACE 2) has been found as the co-receptor for SARS CoV-2 [19] entered in conjunction with the host’s TMPRSS2 membrane protease which facilitates the virus entry [20]. The co-presence of these two entities significantly portends to viral proliferation and infectivity [21]. ACE2 and TMPRSS2 are distributed in lymphocytes, type 2 pneumocytes and bronchial epithelium of lung, gastrointestinal smooth muscle,

myocardium, vascular smooth muscle, neurons, liver and kidney [22]. The location of the receptors can provide insight on manifestation in different organ systems.

SARS CoV-2 virus binds through the spike protein S to the cellular receptor, ACE2.The virus then replicates, stimulating humoral and cellular immunity. It acts by disrupting the ACE2/angiotensin 1-7 axis. In the normal lung, Ang I is converted to Ang II by angiotensin converting enzyme(ACE) which acts on Ang II type 1 receptor to stimulate inflammation, fibrosis and increase in blood pressure. Ang II is degraded by ACE2 to Ang-(1-7) which exhibits anti-inflammatory, antiapoptotic, vasodilatory mechanisms demonstrating cardioprotection. SARS CoV-2, after binding to ACE2 internalizes and down regulates ACE2 resulting in unopposed Ang II accumulation and subsequent effects [23]. Loss of ACE2 can compromise cardiac function. Continued viral proliferation can lead to reduced ACE2 activity in lungs facilitating neutrophil infiltration [24]. Rapid viral replication in the early phase can result in massive epithelial and endothelial cell apoptosis and vascular leakage, triggering release of pro- inflammatory cytokines and chemokines [25].

Retrospective analysis has shown cases of cytokine storm with increased plasma concentrations of interleukins (IL), tumor necrosis factors(TNF) in ICU and severely infected patients than in non ICU and non severely infected patients. The plasma level of IL-6 increases in both mild and severe disease, but more significantly in critically ill patients [13,26,27,28]. These cytokines cause the endothelium to be dysfunctional creating a pro-adhesive and procoagulant state. Activated macrophages also release procoagulant factors such as plasminogen activator inhibitor - 1 (PAI-1) which is enhanced by reduction of ACE2 and predominance of Ang-II. This further enhances the prothrombotic state, often seen in scenarios with elevated IL-6 and D-dimer leading to microangiopathy and microthrombi [22].

Another mechanism contributing to cardiovascular and cerebrovascular events explained by Vetta et al [31] involve the destabilization of a pre-existing plaque caused by the persistent inflammatory state. This leads to rupture of fibrous cap, exposure of thrombogenic material and subsequent thrombotic occlusion of vessel. This inflammatory state further contributes to sympathetic stimulation, free radicals release and a state of increased thrombophilia [31].

The most common parameters consistent with hemostatic abnormalities include mild thrombocytopenia, increased D-dimer levels, increased fibrin degradation products, prolonged prothrombin time or activated partial thromboplastin time [10]. One study showed 71% patients meeting criteria for disseminated intravascular coagulation (DIC) [10]. Other studies showing 25-31% incidence of thrombotic complications despite systemic thromboprophylaxis [5,32]. Klok et al observed 184 patients of which 25 developed pulmonary embolism, 3 developed limb vein thrombosis and 3 with ischemic strokes. A single center retrospective study looking at acute cerebrovascular events showed 11 cases of acute ischemic stroke who were significantly older with cardiovascular risk factors and severe disease [5,6,32]. Severe forms of COVID-19 can lead to increased troponin, not necessarily due to acute coronary syndrome, but could be associated with non ischemic forms like myocarditis, stress cardiomyopathy, coagulopathy [12,29,30].

Takotsubo or stress induced cardiomyopathy is a clinical syndrome characterized by reversible left ventricular (LV) dysfunction often related to an emotional or physical stressful event preceding the syndrome or even without a trigger in some cases [33]. The typical pattern of regional LV wall motion abnormality is apical ballooning (dyskinesia/akinesia/hypokinesia) with basal hyperkinesis. [34] Stress induced cardiomyopathy has been reported secondary to acute critical illness, exacerbation of obstructive lung disease [35,36], acute ischemic or hemorrhagic stroke [37], epilepsy [38], head trauma [39], CKD [40], myasthenic crisis [41] commonly occuring in the setting of sympathetic hyperstimulation. Our patient had obesity, diabetes mellitus, hypertension, hyperlipidemia which has been correlated in literature [42]. In rare situations, Takotsubo has also been found to develop following respiratory tract infections, although the incidence rate is unclear [43]. Takotsubo, particularly secondary to an acute cerebrovascular event is better known as neurogenic stunned myocardium (NSM). Oftentimes, they mimic myocardial infarction with elevated cardiac biomarkers, transient LV hypokinesis and simultaneous coronary vasospasm, suggesting some degree of myocardial injury [44]. Myocarditis can falsely present as Takotsubo as well with ECG and biomarker changes, but presents with a global systolic dysfunction [12]. Coronary microcirculation is innervated by neurons originating in brain stem that mediate vasoconstriction, which could explain the neurogenic origin of myocardial stunning observed [34]. Studies have shown that stimulation or injury to the insular cortex can result in an increase in blood norepinephrine levels, hence sympathetic stimulation leading to LV dysfunction. [45,46] The influx of catecholamines cause cardiac injury through coronary vasospasm [47], increased myocardial oxygen demand [48] and direct toxic effects through calcium overload of the cardiac myocytes [49]. Takotsubo can present as typical apical LV segment dysfunction or atypical variants. The apical predilection has been attributed to a high β_2:β_1 receptor ratio [50] 10% of these patients can progress to cardiogenic shock [51].

In our patient with multiple traditional cardiovascular risk factors the acute ischemic is attributed to secondary to procoagulant state, stasis and endothelial damage from COVID-19. The developed stress cardiomyopathy/takotsubo cardiomyopathy is likely a result of stress, catecholamines and sympathetic overdrive from the ischemic stroke and ongoing COVID-19 infection. Physicians should also be aware of the differential diagnosis of ST segment elevation in the EKG in the setting of COVID-19 infection such as acute coronary syndrome [52], myocarditis [12], takotsubo cardiomyopathy [12], myopericarditis [53] and spontaneous coronary artery dissection [54]. Point of care ultrasound (POCUS) can be helpful in COVID-19 pandemic in early diagnosis and thus aid in early management of cardiovascular disease [55].

4. Conclusion

This unique case highlights the first noted presentation of a patient with COVID-19 pneumonia who presented with acute ischemic stroke. Incidentally also having LV dysfunction with Takotsubo Cardiomyopathy, complicated by cardiogenic shock and death. Takotsubo Cardiomyopathy, is known to be caused by physical stressors like stroke, sepsis, infections. But it is important to keep in mind the differentials of Takotsubo Cardiomyopathy, including acute coronary syndrome, myocarditis, pericarditis and coronary

dissection. To the best of our knowledge thi si the first case of typical apical variant of takotsubo cardiomyopathy in COVID 19 infection.

Acknowledgements

Tliis work is supported, in part, by the efforts of Dr. Moro O. Salifu M.D., M.P.H., M.B.A., M.A.C.P., Professor and Chairman of Medicine through NIH Grant number S21MD012474.

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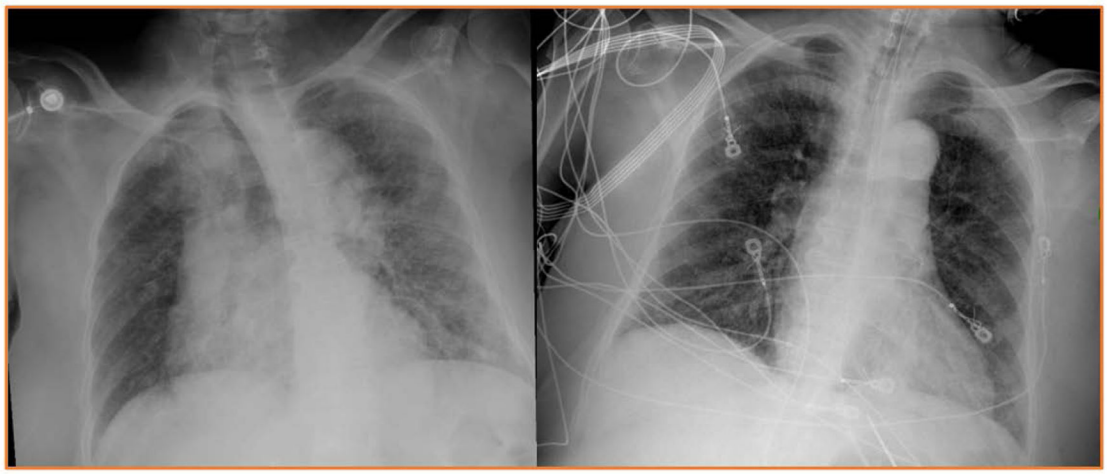

Figure 1.
Chest X-ray showing diffuse bilateral infiltrates at presentation (left) and after intubation (right)

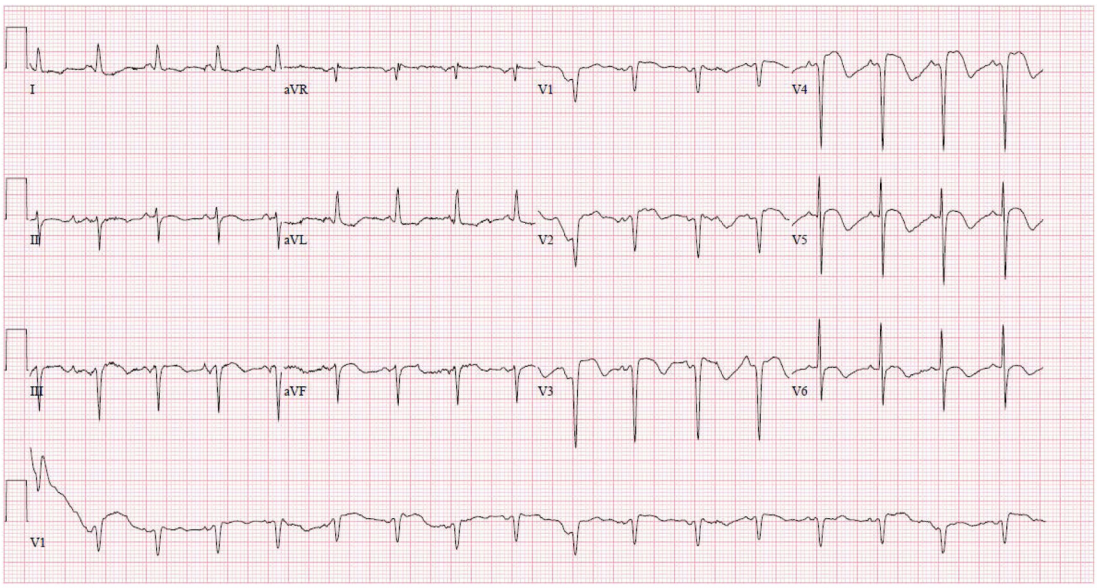

Figure 2.
EKG showing normal sinus tachycardia Q waves in V1-V2 leads suggestive of septal infarct and Q waves with ST segment elevation V3, V4, V5 and deep T wave inversion in V6

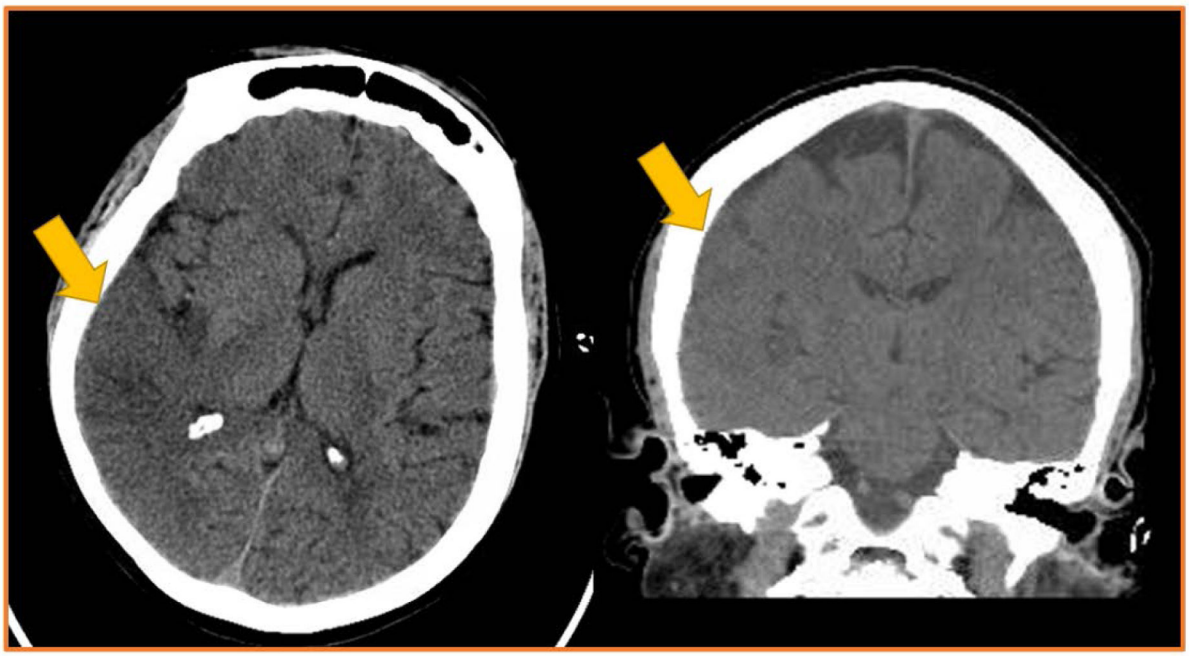

Figure 3.
CT head demonstrated a subtle hypoattenuation in the right parietal lobe with loss of gray-white differentiation and sulcal effacement suggestive of acute infarct (indicated by arrow)

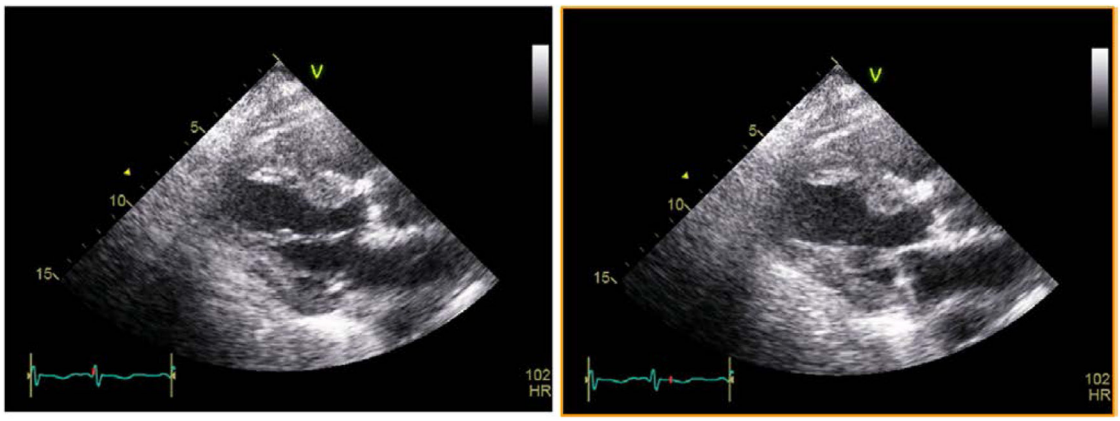

Figure 4.

Transthoracic echocardiography- parasternal long axis view showing Takotsubo cardiomyopathy. Note the anteroseptal wall in diastole (left) and ballooning of the anteroseptal wall in systole (right)

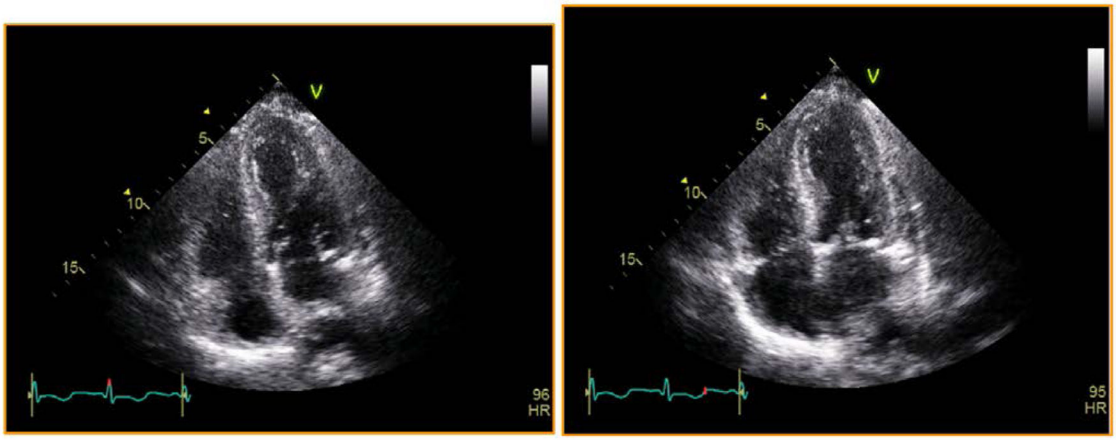

Figure 5.

Transthoracic echocardiography- four chamber view showing Takotsubo cardiomyopathy. Note the apical wall in diastole (above) and ballooning of the apical wall in systole (below). This is typical of apical takotsubo cardiomyopathy.

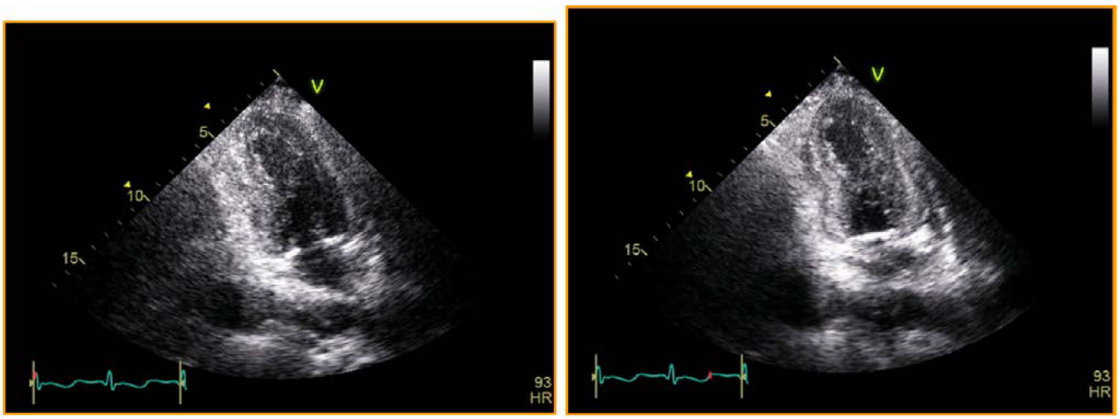

Figure 6.

Transthoracic echocardiography- two chamber view showing Takotsubo cardiomyopathy. Note the apical wall in diastole (above) and ballooning of the apical wall in systole (below). This is typical of apical takotsubo cardiomyopathy

Table 1.

Complete blood count at presentation

Complete blood count	Reference	At presentation
White blood cell count (10x3/uL)	4.10 - 10.10 10x3/uL	21.40
Neutrophils (%age)	44.5 - 73.4 %	92.4
Lymphocytes auto (%age)	17.8 - 42.0 %	4.4
Monocytes auto (%age)	5.7 - 11.2 %	2.5
Eosinophils Auto. (%age)	0.2 - 6.0 %	0.6
Basophill auto (%age)	0.3 - 1.1 %	0.2
Neutrophils Absolute (10x3/uL)	1.40 - 6.80 10x3/uL	18.10
Lymphocytes Absolute (10x3/uL)	1.10 - 2.90 10x3/uL	0.90
Monocytes Absolute (10x3/uL)	0.20 - 1.00 10x3/uL	0.50
Eosinophils absolute (10x3/uL)	0.00 - 0.40 10x3/uL	0.10
Basophil absolute (10x3/uL)	0.00 - 0.10 10x3/uL	0.00
Red blood cells (10x6/uL)	4.33 - 5.43 10x6/uL	2.95
Hemoglobin (g/dL)	12.9 - 16.7 g/dL	13.8
Hematocrit (% age)	40.0 - 47.0 %	42.6
Mean Corpuscular Volume (fL)	80.8 - 94.1 fL	106.1
Mean Corpuscular Hemoglobin(pg)	27.1 - 31.2 pg	34.5
Mean Corpuscular hemoglobin concentration (g/dl)	31.0 - 34.4 g/dL	32.5
Red cell distribution width (% age)	12.3 - 14.6 %	14.3
Mean Platelet Volume (fL)	7.9 - 11.0 fL	8.6
Platelets (10x3/uL)	153 - 328 10x3/uL	424

Table 2.

Comprehensive metabolic panel and inflammatory markers at presentation

Chemistry	Reference	At presentation
Glucose(mg/dL)	70 - 99 mg/dL	181
Blood Urea Nitrogen (mg/dL)	9.0 - 20.0 mg/dL	36
Creatine (mg/dL)	0.66 - 1.25 mg/dL	1.84
Sodium (mEq/L)	133 - 145 mEq/L	137
Potassium (mEq/L)	3.5 - 5.1 mEq/L	4.5
Chloride (mEq/L)	98 - 107 mEq/L	98
Calcium (mg/dL)	8.4 - 10.5 mg/dL	8.7
Anion Gap (mEq/L)		14.00
Anion Gap (mmoL/L) with K	7.00 - 17.00 mmoL/L	18.50
Total Protein (g/dL)	6.3 - 8.2 g/dL	7.4
Albumin (g/dL)	3.5 - 5.0 g/dL	3.1
Total Bilirubin (mg/dL)	0.2 - 1.3 mg/dL	1.0
Aspartate transaminase (U/L)	21 - 72 U/L	49
Alanine aminotransferase (U/L)	17 - 59 U/L	77
Alkaline Phosphatase(U/L)	38.0 - 126.0 U/L	87.0
Serum Bicarbonate. Co2 (mEq/L)	22 - 30 mEq/L	25
C reactive protein	0.50 - 1.00 mg/dL	27
Ferritin	11-264 ng/mL	476 → 652

Table 3.

Coagulation parameters at presentation

Coagulation parameters	Reference range	At presentation
Prothrombin time	10.3-13.7 seconds	15.6
INR	0.7-1.2	1.31
Partial thromboplastin time	23.5-35.5 seconds	30.7
D-Dimer	0-230 ng/ml	6518

Table 4.

Cardiac biomarkers at presentation

	Reference range	At presentation
Troponin I (ng/mL)	0.012 - 0.034 ng/mL	3.930 → 4.250
P-Natriuretic Peptide (pg/mL)	11.1 - 125.0 pg/mL	17800