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Rare Association of Takotsubo Cardiomyopathy with Right Bundle Branch Block in the Dual Setting of Asthma Exacerbation and Psychiatric Illness

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Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
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Conflict of interest: None declared

Patient: Male, 58-year-old
Final Diagnosis: Takotsubo cardiomyopathy
Symptoms: Dyspena
Medication: —
Clinical Procedure: —
Specialty: Cardiology

Objective: Rare co-existance of disease or pathology

Background: Takotsubo cardiomyopathy is characterized by a transient left ventricular dysfunction without obstructive coronary artery disease that mimics an acute myocardial infarction. The electrocardiogram findings of Takotsubo cardiomyopathy usually present with ST-segment elevation or depression, T-wave inversion, left bundle branch block or high-grade atrioventricular block.

Case Report: This is a report of a case of a 58-year-old male diagnosed with Takotsubo cardiomyopathy that occurred in the setting of an acute asthma exacerbation and psychiatric exacerbation with novel electrocardiogram findings of right bundle branch block. Transthoracic echocardiogram showed a preserved ejection fraction with left ventricular apical ballooning and hyperkinesis of the basal segments. The nuclear stress test showed a fixed perfusion defect at the apical segment, but the patient refused further testing such as coronary angiography. The patient was managed medically, and a repeat echocardiogram done after 8 weeks from discharge showed a complete resolution of the apical ballooning.

Conclusions: It is important to recognize that patients with psychiatric illness and asthma exacerbation are predisposed to develop Takotsubo cardiomyopathy. It is also reasonable to suspect Takotsubo cardiomyopathy in the presence of new electrocardiogram findings aside from those typically seen in acute myocardial infarction, especially if it is associated with apical ballooning.

MeSH Keywords: Asthma • Bundle-Branch Block • Psychotic Disorders • Takotsubo Cardiomyopathy

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/920461

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Background

Stress cardiomyopathy is reported among elderly females who present with anginal pain after a severe emotional or physical stressor with an incidence of 1% to 2% [1]. It is typically associated with ischemic electrocardiogram (ECG) findings similar to an acute myocardial infarction, with ST segment elevation being the most common (43.7%), followed by ST depression (7.7%), QT interval prolongation, T wave inversion or abnormal Q waves [1]. Some cases of left bundle branch block (LBBB) or high-grade atrioventricular block are also noted [1]. We present this case report of a male patient with underlying psychiatric illness who developed stress cardiomyopathy with atypical right bundle branch block (RBBB) that was triggered by combined exacerbation of his underlying psychiatric illness and asthma.

Case Report

A 58-year-old male with known schizoaffective disorder, hypertension, and asthma presented with acute worsening of shortness of breath. Initial vitals were within normal limits. On evaluation, the initial ECG showed a RBBB and left posterior fascicular block (Figure 1) that was not present on his previous ECG (Figure 2). The initial troponin I was elevated (1.8 ng/mL, normal < 0.04 ng/mL) and echocardiogram unremarkable. Other laboratory investigations were unremarkable. The patient was managed as a case of non-ST elevation myocardial infarction with asthma exacerbation. The following day, the patient exhibited increasingly agitated behavior with worsening of the asthma exacerbation accompanied by new T wave inversions (TWI) on the anterolateral leads (Figure 3) and rising troponins (4.9 ng/mL) (Table 1). He denied any chest pain, dyspnea, or palpitations at the time the troponins peaked. On examination, bilateral extensive wheezing was noted. After ruling out organic causes such as acute neurologic or metabolic changes for his behavioral changes, the patient received lorazepam to control his agitation. A bedside echocardiogram was done due to the rising troponins with ischemic ECG changes, which showed left ventricular apical ballooning. Due to the high suspicion of stress cardiomyopathy, he received lorazepam 2 mg intravenous (IV) every 8 hours to prevent further episodes of agitation and low dose beta-agonist nebulization for acute exacerbation of asthma. A formal echocardiogram was done the following day, which showed an ejection fraction of >55%, and apical ballooning with hyperkinesis of the basal segments (Figure 4). A nuclear stress test (NST) was done which showed a moderate fixed perfusion defect on the apical segment (Figure 5). Left heart catheterization with coronary angiography was planned to evaluate for underlying coronary artery disease, but the patient refused any invasive procedures. The patient was discharged on aspirin 81 mg/day, atorvastatin 40 mg/day, lisinopril 2.5 mg/day, and metoprolol succinate

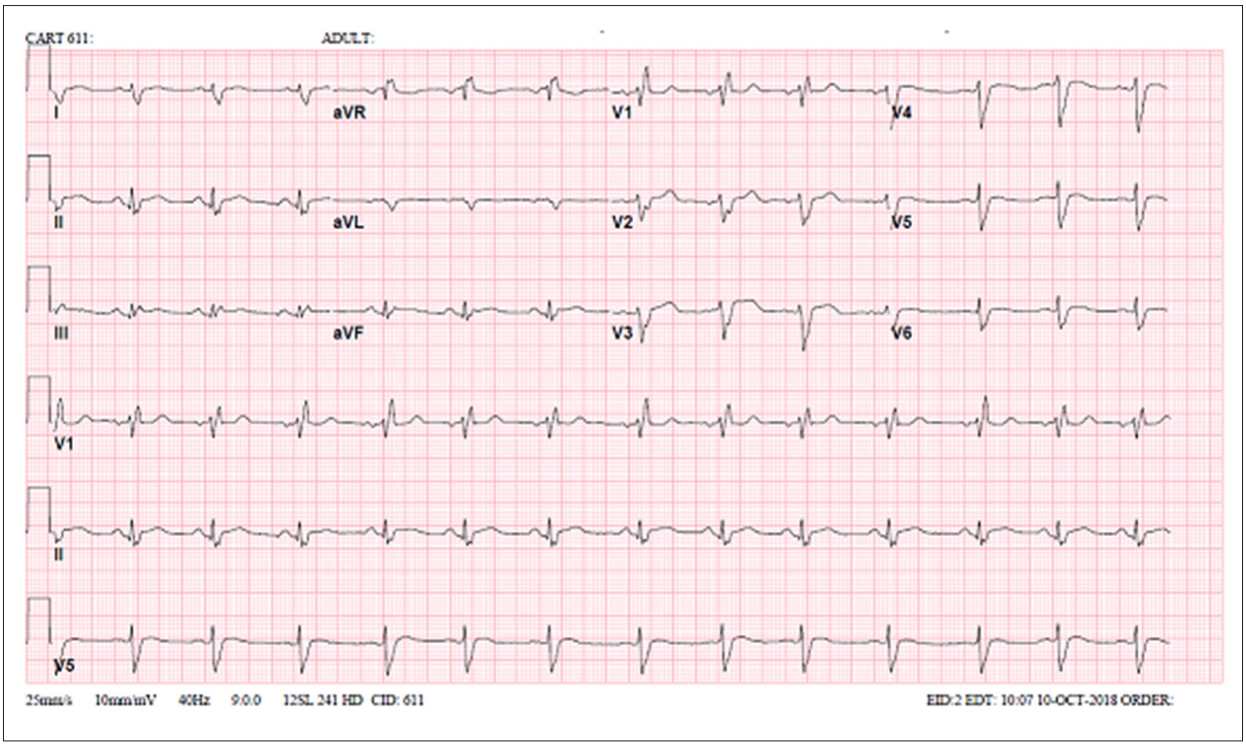

Figure 1. Electrocardiogram on admission showing new onset right bundle branch block and left posterior fascicular block, rate 80 bpm, PR 146 ms, QTc 447 ms (October 6, 2018 12: 30 PM).

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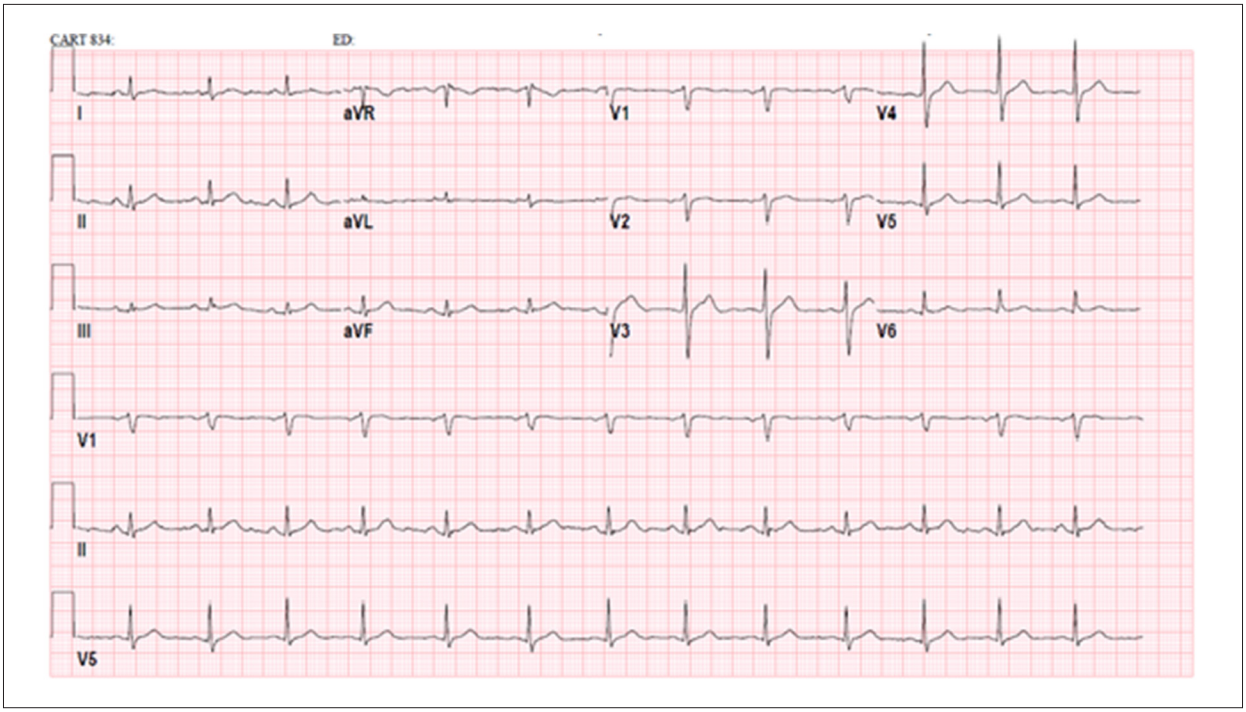

Figure 2. Previous electrocardiogram showing normal sinus rhythm, rate of 81 bpm, PR interval 150 ms, QTc 420 ms (July 2017).

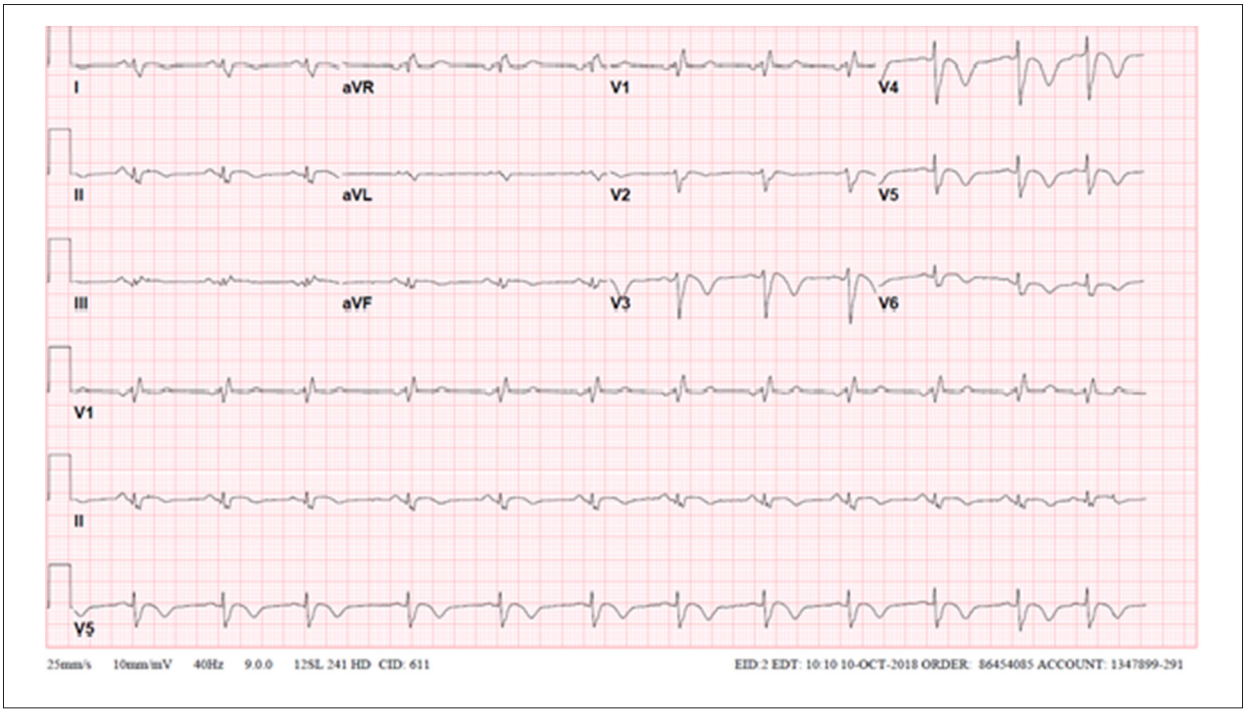

Figure 3. Electrocardiogram showing new T wave inversions in the anterolateral leads when the troponins peaked at 3.9 ng/mL during the acute episode of agitation and asthma exacerbation. (Oct 7, 2018 2 PM).

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Table 1. Trend of Troponin-I levels.

Time from initial event	Troponin I (ng/ml)
0 hour	1.8
4 hours	1.8
10 hours	2.6
17 hours	3.5
26 hours (peak agitation)	4.9
32 hours	3.5
38 hours	3.9
44 hours	2.0
50 hours	1.4
68 hours	0.67
92 hours	0.32

50 mg/day. A repeat echocardiogram was done 8 weeks after, which showed complete resolution of the apical ballooning.

Discussion

Stress cardiomyopathy, also known as Takotsubo cardiomyopathy or apical ballooning syndrome is characterized by a transient left ventricular dysfunction that may mimic an acute myocardial infarction but without evidence of obstructive coronary artery disease [2–4]. Imaging shows hypokinesis of the left ventricular mid and apical segments with hyperkinesis of the basal segments leading to a distal ballooning of the apex [3,4]. In most cases, the regional wall abnormality extends beyond the territory perfused by a single epicardial coronary artery [4]. Stress cardiomyopathy typically occurs among post-menopausal women who present with anginal pain. Some patients

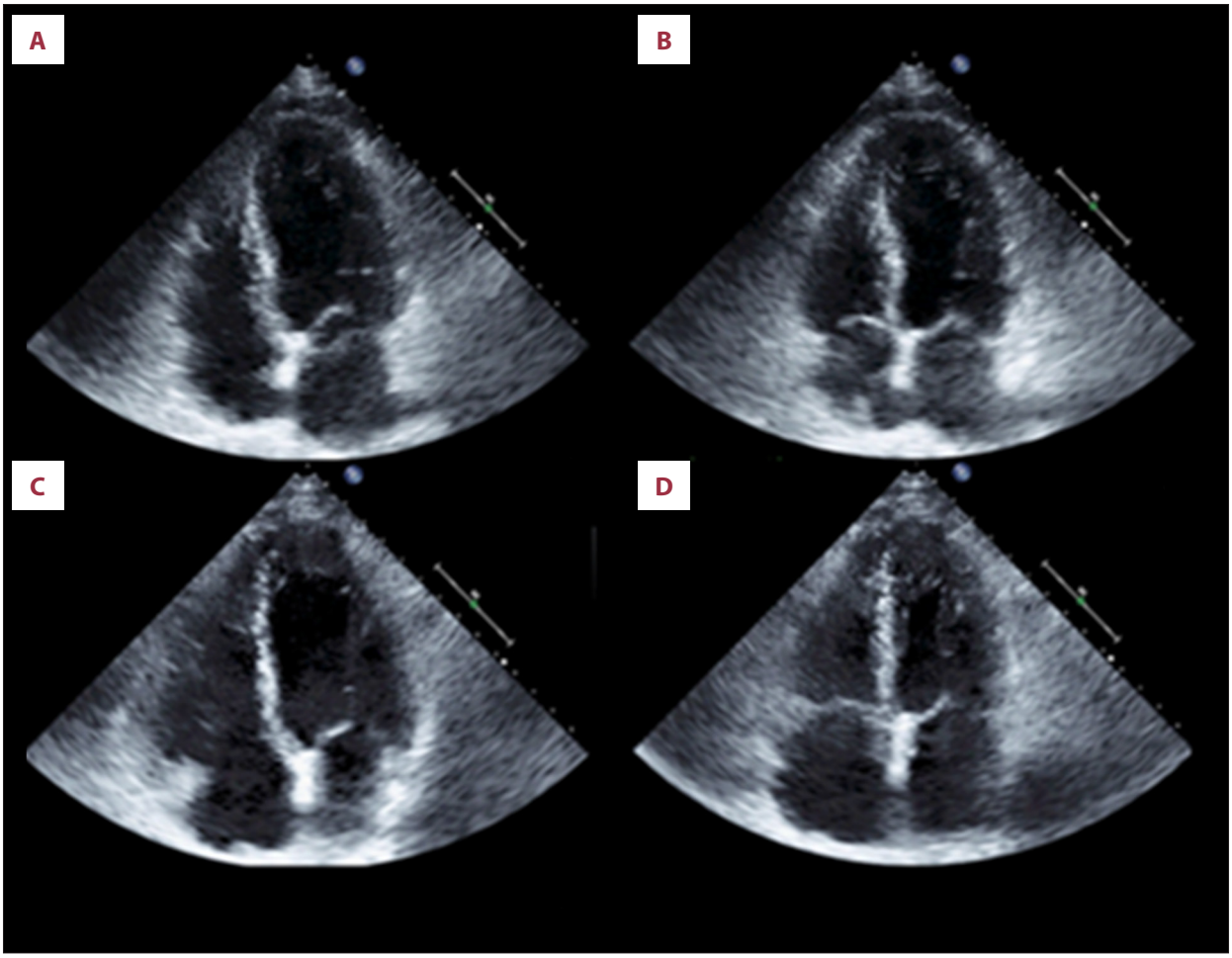

Figure 4. Initial echocardiogram showing apical ballooning during diastole (**A**) and systole (**B**) in the 4-chamber view. Repeat echocardiogram 8 weeks after discharge showing resolution of the apical ballooning during diastole (**C**) and systole (**D**) in the 4-chamber view.

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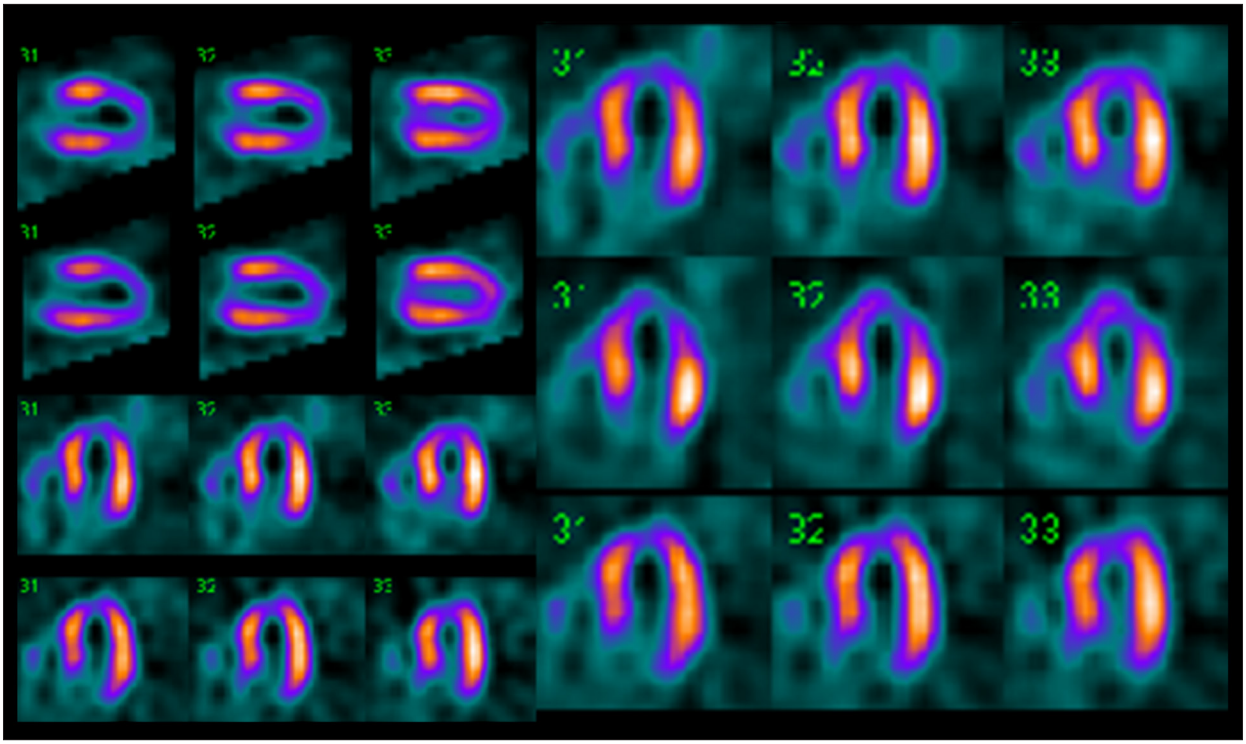

Figure 5. The nuclear stress test using ^{99mc}TC sestamibi scan showing the fixed perfusion defect on the apical segment.

also present with dyspnea, syncope, or even shock in the setting of an acute episode of severe emotional or physical stress [2,3,5,6]. Typical ECG findings include changes that can also be seen in myocardial infarction such as ST elevation (STe), ST depression (STd), T wave inversion (TWI), prolonged QT interval, left bundle branch block or high degree atrioventricular block. But the presence of STe without reciprocal STd or TWI is more prevalent in stress cardiomyopathy compared to myocardial infarction [7,8]. To our knowledge, this is the first case of stress cardiomyopathy that is associated with new onset RBBB and left posterior fascicular block, along with typical findings of TWI on the ECG that developed in the dual setting of asthma exacerbation and psychiatric disorder.

Retrospective studies have shown an association between stress cardiomyopathy and psychiatric illness. Around one third of patients with stress cardiomyopathy had a psychiatric comorbidity, such as depression, anxiety or schizophrenia among others [9–11]. Exacerbations of psychiatric illness have also been reported as a trigger for stress cardiomyopathy [9]. The psychiatric illness was managed accordingly, and the cardiomyopathy resolved after 3 to 4 weeks [9–11].

Other studies have also shown stress cardiomyopathy in the setting of an acute asthma exacerbation or after repeated use of beta2-agonist inhalation [10–17]. Most of the cases involved elderly women with a history of asthma presenting with dyspnea rather than chest pain [12–19]. Only 1 case involved a male patient who presented with asthma exacerbation complicated with stress cardiomyopathy after frequent inhalation of steroids and long acting beta2-agonist [17]. There was resolution of the cardiomyopathy after 2 to 3 weeks among the cases reported.

The exact mechanism of the pathophysiology of stress cardiomyopathy is not yet fully understood. Due to its association with severe emotional or physical stress, it has long been postulated that a surge of catecholamines from increased sympathetic activity can induce coronary microvascular spasm and myocardial dysfunction [20,21]. The transient ventricular dysfunction occurs as a result of catecholamine-induced myocardial stunning or myocardial toxicity [22,23].

The role of catecholamines in the brain-heart axis is thought to account for the association of psychiatric disorder with stress cardiomyopathy [24–26]. Psychiatric patients have higher catecholamine levels and have a higher cardiac sympathetic sensitivity through reduced catecholamine uptake [27–29]. This may explain the predisposition of patients with psychiatric disorder to develop stress cardiomyopathy.

Asthmatic patients are also predisposed to develop stress cardiomyopathy since asthma exacerbation can also cause elevated catecholamine levels in response to increased sympathetic activity [30,31]. Repeated use of beta2-agonists for management of asthma is another potential trigger for stress

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cardiomyopathy as well. *In vitro* studies have shown that the apical cardiomyocytes have more beta2 receptors and are more sensitive to epinephrine compared to basal cardiomyocytes [21]. As in the case of our patient, he was also predisposed to develop stress cardiomyopathy secondary to his underlying psychiatric illness, the asthma exacerbation and repeated use of beta2-agonists.

The initial management of stress cardiomyopathy is similar to an acute myocardial infarction until obstructive coronary artery disease has been ruled out. But in our case, we were not able to effectively rule out coronary obstruction through coronary angiogram. We suggest using alternative methods such as cardiac nuclear imaging techniques if the patient is not willing to undergo coronary angiogram or if contrast is contraindicated in the setting of renal failure. Both single photon emission computed tomography (SPECT, using ^{201}thallium chloride or ^{99m}technetium sestamibi) which provides semi-quantitative information and position emission tomography (PET, using ^{13}N-ammonia ^{82}Rubidium) which offers quantitative measurements, have been used in stress cardiomyopathy for assessment of perfusion, metabolism, and innervation. The "myocardial thinning" in the apical segment may lead to a reduction in isotope counts because of the partial volume effect, which may mimic reduction of perfusion on SPECT, but following correction for this factor on PET, blood flow in the thinned apical regions is intact while the normally functioning basal segments show hyper-perfusion [8].

Once acute myocardial infarction has been ruled out, the management of stress cardiomyopathy involves addressing the offending factor and managing for heart failure such as beta blockers and angiotensin converting enzyme inhibitors (ACEIs) or angiotensin receptor blockers (ARBs) [8]. The use of ACEIs or ARBS is associated with improved mortality benefit, whereas beta-blockers did not show any survival benefit after 1 year [2]. The triggering event for the cardiomyopathy in this case was controlled with benzodiazepines for his agitation and salbutamol nebulization for his asthma exacerbation. The frequency of salbutamol nebulization was decreased after his asthma exacerbation was stabilized to prevent further exacerbation of his cardiomyopathy. He was then started on metoprolol succinate and lisinopril for management of heart failure.

The prognosis is excellent, with most patients recovering completely within 4 to 8 weeks, as in this case [32]. However, it can still cause severe cardiac complications such as life-threatening arrhythmias, acute heart failure, and cardiac rupture among others [33,34]. Recurrence may recur in 1.5% to 3.5% of the cases, and may be related to underlying psychiatric disorders [35].

Conclusions

Echocardiogram remains a useful test in diagnosing stress cardiomyopathy since it is non-invasive and can show the transient apical ballooning that is characteristic of this condition. However, ECG findings should not be limited to STe, STd, TWI, or LBBB in considering this disease. It is reasonable to suspect stress cardiomyopathy in the presence of any new ECG findings associated with apical ballooning, especially if it occurs in the setting of an acute stressor. Other echocardiographic findings include atypical variants of regional wall motion abnormalities like basal, mid-ventricular, focal or diffuse. Predisposing factors should also be considered especially among patients with underlying psychiatric illness. The judicious use of beta-agonists in asthma exacerbation is another important factor to consider to prevent further worsening of the cardiomyopathy. Future studies should also investigate the diagnostic value of cardiac nuclear imaging as an alternative method to evaluate for transient perfusion defects in cases where patients are unable to do coronary angiogram.

Conflicts of interest

None.

References:

1. Kurowski V, Kaiser A, Hof KV et al: Apical and midventricular transient left ventricular dysfunction syndrome (Tako-Tsubo cardiomyopathy) frequency, mechanisms, and prognosis. Chest, 2007; 132: 809–16
2. Templin C, Ghadri JR, Diekmann J et al: Clinical features and outcomes of Takotsubo (stress) cardiomyopathy. N Eng J Med, 2015; 373: 929–38
3. Tsuchichashi K, Ueshima K, Uchida T et al: Transient left ventricular apical ballooning without coronary artery stenosis: A novel heart syndrome mimicking acute myocardial infarction. Angina pectoris-myocardial infarction investigations in Japan. J Am Coll Cardiol, 2001; 38(1): 11–18
4. Prasad A, Lerman A, Rihal CS: Apical ballooning syndrome (Takotsubo or stress cardiomyopathy): A mimic of acute myocardial infarction. Am Heart J, 2008; 155(3): 408–17
5. Sharkey SW, Lesser JR, Zenovich AG et al: Acute and reversible cardiomyopathy provoked by stress in women from the United States. Circulation, 2005; 111: 472–79
6. Akashi YJ, Goldstein DS, Barbaro G, Ueyama T: Takotsubo cardiomyopathy; A new form of reversible heart failure. Circulation, 2008; 118: 2754–62
7. Frangieh AH, Obeid S, Ghadri JR et al: ECG criteria to differentiate between Takotsubo (stress) cardiomyopathy and myocardial infarction. J Am Heart Assoc, 2016; 5(6): pii: e003418
8. Ghadri JR, Wittstein IS, Prasad A et al: International expert consensus document on Takotsubo syndrome (part II): Diagnostic workup, outcome, and management. Eur Heart J, 2018; 39(22): 2047–62
9. Nayeri A, Rafla-Yuan E, Farber-Eger E et al: Pre-existing psychiatric illness is associated with increased risk of recurrent Takotsubo cardiomyopathy. Psychosomatics, 2017; 58(5): 527–32

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10. Corrigan FE, Kimmel MC, Jayaram G: Four cases of Takotsubo cardiomyopathy linked with exacerbations of psychiatric illness. Innov Clin Neurosci, 2011; 8(7): 50–53
11. El-sayed AM, Brinjikji W, Salka S: Demographic and comorbid predictors of stress (takotsubo) cardiomyopathy. Am J Cardiol, 2012; 11(9): 1368–72
12. Stanojevic DA, Alla VM, Lynch JD, Hunter CB: Case of reverse takotsubo cardiomyopathy in status asthmaticus. South Med J, 2010; 103(9): 964
13. Mendoza I, Novaro GM: Repeat recurrence of Takotsubo cardiomyopathy related to inhaled beta-2-adrenoceptor agonists. World J Cardiol, 2012; 4: 211–13
14. Venditti F, Bellandi B, Parodi G: Fatal Tako-Tsubo cardiomyopathy recurrence after β2-agonist administration. Int J Cardiol, 2012; 161: e10-1
15. Patel B, Assad D, Wiemann C et al: Repeated use of albuterol inhaler as a potential cause of Takotsubo cardiomyopathy. Am J Case Rep, 2014; 15: 221–25
16. Marmoush FY, Barbour MF, Noonan TE et al: Takotsubo cardiomyopathy: A new perspective in Asthma. Case Rep Cardiol, 2015; 2015: 640795
17. Saito N, Suzuki M, Ishii S et al: Asthmatic attack complicated with takotsubo cardiomyopathy after frequent inhalation of inhaled corticosteroids/long-acting beta2-adrenoceptor agonists. Intern Med, 2016; 55: 1615–20
18. Parsa SA, Khaheshi I, Memaryan M, Naderian M: Takotsubo cardiomyopathy following recurrent doses of albuterol due to asthma attack: A very rare case report. Future Cardiol, 201; 12(6): 609–12
19. Kotsiou OS, Douras A, Makris D et al: Takotsubo cardiomyopathy: A known unknown foe of asthma. J Asthma, 2017; 54(8): 880–86
20. Wittstein IS, Thiemann DR, Lima JA et al: Neurohormonal features of myocardial stunning due to sudden emotional stress. N Engl J Med, 2005; 352(6): 539–48
21. Paur H, Wright PT, Sikkel MB et al: High levels of circulation epinephrine trigger apical cardiodepression in a β2-adrenergic receptor/Gi-dependent manner: A new model of Takotsubo cardiomyopathy. Circulation, 2012; 126: 697–706
22. Gianni M, Dentali F, Grandi AM et al: Apical ballooning syndrome or Takotsubo cardiomyopathy: A systematic review. Eur Heart J, 2006; 27: 1523–29
23. Nef HM, Möllmann H, Kostin S et al: Tako-Tsubo cardiomyopathy: Intraindividual structural analysis in the acute phase and after functional recovery. Eur Heart J, 2007; 28: 2456–64
24. Samuels MA: The brain-heart connection. Circulation, 2007; 116(1): 77–84
25. Suzuki H, Matsumoto Y, Kaneta T et al: Evidence for brain activation in patients with Takotsubo cardiomyopathy. Circ J, 2014; 78(1): 256–58
26. Ziegelstein R: Depression and Takotsubo cardiomyopathy. Am J Cardiol, 2010; 105(2): 281–82
27. Fell D, Derbyshire DR, Maile CJ et al: Measurement of plasma catecholamine concentrations. An assessment of anxiety. Br J Anaesth, 1987; 57(8): 770–74
28. Wyatt RJ, Portnoy B, Kupfger DJ et al: Resting plasma catecholamine concentrations in patients with depression and anxiety, Arch Gen Psychiatry, 1971; 24(1): 65–70
29. Barton DA. Dawood T, Lamber EA et al: Sympathetic activity in major depressive disorder: Identifying those at cardiac risk. J Hypertens, 2007; 25: 2117–24
30. Barnes PJ: Endogeneous plasma adrenaline in asthma. Eur J Respir Dis, 1983; 64: 559–63
31. Ind PW, Causon RC, Brown MJ, Barnes PJ: Circulating catecholamines in acute asthma. Br Med J, 1985; 290: 267–69
32. Singh K, Carson K, Usmani Z et al: Systematic review and meta-analysis of incidence and correlates of recurrence of Takotsubo cardiomyopathy. Int J Cardiol, 2014; 174(3): 696–701
33. Migliore F, Zorzi A, Peruzza F et al: Incidence and management of life-threatening arrhythmias in Takotsubo syndrome. Int J Cardiol, 2013; 166: 261–63
34. Donohue D, Movahed MR: Clinical characteristics, demographics and prognosis of transient left ventricular apical ballooning syndrome. Heart Failure Rev, 2015; 10(4): 311–16
35. Christensen TE, Ahtarovski KA, Anderson H et al: Takotsubo cardiomyopathy: A case of extremely fast recovery described by multimodality cardiac imaging. J Nucl Cardiol, 2012; 19: 1240–42

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