CASE REPORT

Bedside Cardiac Ultrasound to Aid in Diagnosing Takotsubo Cardiomyopathy

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A case of a 64-year-old patient presenting with new-onset chest pain and shortness of breath illustrates the utility of bedside cardiac ultrasound.

Cardiac ultrasound is among the many beneficial applications of point-of-care (POC) ultrasound in the ED. This modality can prove extremely beneficial in evaluating the critically ill patient. For example, POC cardiac ultrasound not only permits the emergency physician (EP) to diagnose a pericardial effusion and cardiac tamponade, but also perform a pericardiocentesis.[1] The EP can also employ beside ultrasound to estimate an ejection fraction (EF) almost as well as cardiology services,[2] look for signs of right-heart strain in patients with pulmonary embolism (PE),[3] and guide fluid management in patients who have septic shock.[4] In addition to only taking a few minutes to perform, POC cardiac ultrasound can also drastically

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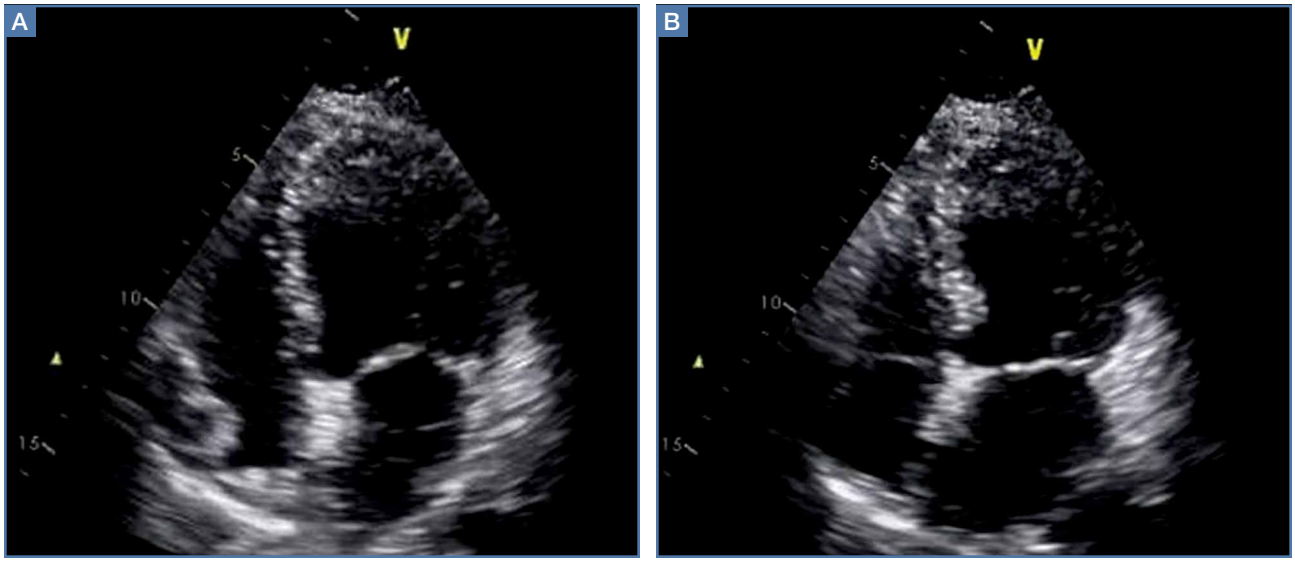

Figure. Apical four-chamber ultrasound views of the patient's heart in (**A**) diastole and (**B**) systole demonstrate severely decreased ejection fraction with apical hypokinesis, no pericardial effusion, and a normal-sized right ventricle.

change the course of management in some patients. Our case illustrates the use of POC ultrasound to diagnose Takotsubo cardiomyopathy in a 64-year-old patient and guide management when she became unstable prior to cardiac catheterization.

Case

A 64-year-old white woman with a medical history of diabetes, obesity, and nephrolithiasis presented to the ED with chest pain and shortness of breath, which she stated had begun earlier in the day. The patient's chest pain did not intensify upon exertion, but the shortness of breath worsened when she was in the supine position.

Three months prior, the patient had also presented to our ED with chest pain. Evaluation during that visit included a negative stress echocardiogram with an EF of 55%. At this second visit, an electrocardiogram (ECG) showed new T-wave inversions in the anterior, lateral, and inferior leads. Vital signs at presentation were: blood pressure, 107/63 mm Hg; heart rate, 100 beats/min; respiratory rate, 18 breaths/min; and temperature, 97.9°F. Oxygen saturation was 97% on room air when patient was sitting upright, but decreased to 90% when she was supine. A chest X-ray showed left basilar atelectasis with a trace effusion. Laboratory evaluation was remarkable for the following: troponin I, 2.99 ng/mL; D-dimer, 294 ng/mL; and brain natriuretic peptide, 559 pg/mL.

Given the patient's vital signs and positive troponin I level, a computed tomography (CT) scan was ordered to assess for a PE. This was done despite the patient's negative D-dimer results, as it was felt that she was not low-risk for PE. At the same time the CT scan was ordered, a POC cardiac ultrasound was performed to assess for signs of right heart strain. Ultrasound showed a severely decreased EF of approximately 20% with apical hypokinesis, no pericardial effusion, and a normal-sized right ventricle (**Figure**).

Based on the ultrasound findings and a normal EF 3 months prior, there was concern for Takotsubo cardiomyopathy. The patient was further questioned as to the events surrounding the onset of her chest pain. She informed the EP the pain started when she learned that she might be evicted from her home.

The CT scan was negative for PE. The consulting cardiologist was informed of the results of the ultrasound findings, and the patient was given aspirin, heparin, morphine, and furosemide, and was admitted to the cardiac progressive unit. She was also initially given morphine for pain management, but due to intolerance, she was switched to nitroglycerin.

During the first evening of her inpatient stay, the patient experienced acute changes in her chest pain that resulted in activating the rapid response team. Secondary to the information gathered in the ED, the patient was managed conservatively and was evaluated by a physician extender who repeated laboratory studies, provided supplemental potassium and magnesium, and ordered another ECG in consultation with the cardiologist (who was caring for the patient via telephone). In the morning, the patient continued to have chest pain, and a repeat ECG showed worsening of previous T-wave inversions. Based on these findings, the cardiologist ordered cardiac catheterization.

On hospital day 2, the cardiologist performed another echocardiogram, which confirmed the low EF of 20% with severe global hypokinesis with sparing of the basal segments. Cardiac catheterization showed no significant disease (20% lesion in the mid-left anterior descending artery) with the left ventriculogram showing an EF of 10%, cardiac output of 3.7, and cardiac index of 1.8, confirming the diagnosis of Takotsubo cardiomyopathy. The patient remained in the hospital for a total of 8 days while awaiting a life vest; however, a repeat echocardiogram on hospital day 8 showed an EF of 55%.

Discussion

Takotsubo cardiomyopathy is an acute, stress-induced cardiomyopathy that was first described in Japan in the early 1990s.[5] It is thought to be due to catecholamine-induced dysfunction from a stressful event,[6-8] such as the death of a loved one, which is why it is often referred to as "broken heart syndrome." However there are case reports highlighting other causes of Takotsubo cardiomyopathy, such as cocaine use,[9] scuba diving,[10] and diabetic ketoacidosis combined with hypothermia.[11]

Patients with Takotsubo cardiomyopathy will frequently have ECG abnormalities, including ST-segment elevation or depression, or T-wave changes; troponin levels also may be elevated. The majority of patients (>80%) are postmenopausal women, typically aged 50 to 75 years.[6,12] Echocardiogram findings in Takotsubo cardiomyopathy show significant left ventricular (LV) dysfunction or regional dysfunction that is not in one coronary artery distribution.[12,13] There will often be apical dilation or ballooning with dyskinesia but more preserved function at the base and normal dimensions.[14,15] A negative cardiac catheterization or catheterization in the absence of significant disease is required to confirm the diagnosis.[16] The LV function usually returns to baseline in 1 to 4 weeks, but there can be recurrence in some patients.[6,17] The condition is also associated with a large burden of morbidity and mortality.[6,18] In a case series by Gopalakrishnan et al[6] of 56 patients, there was an 8.9% in-hospital mortality rate and an additional 17.9% out-of-hospital mortality rate even in patients in whom LV function had returned to normal.

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In a review by Gianni et al,[19] 4.2% of patients with Takotsubo cardiomyopathy present with or go into cardiogenic shock at some point during admission, and up to 2% of patients who present with acute myo-

cardial infarction have Takotsubo cardiomyopathy. Patients can go into cardiogenic shock due to depressed EF or LV outflow tract obstruction from hyperkinesis of the basilar segments. Some of these patients may be sent directly to the catheter laboratory based on ST elevations on ECG, in which case the diagnosis is made there. Our patient, however, did not have ST elevation and later became unstable on the floor. Citro et al[20] suggest that a patient with a predisposition for Takotsubo cardiomyopathy (eg, postmenopausal patients, those who experienced a trigger event), in the right clinical setting and without ST-segment elevation on ECG, could be managed more conservatively with delayed cardiac angiography or CT angiography (CTA) evaluation of the coronary arteries (sparing the patient an invasive procedure)—as long as ultrasound was consistent with typical Takotsubo cardiomyopathy findings. However, CTA is still needed to make the diagnosis.

At this time, Takotsubo cardiomyopathy should remain an important part of the differential diagnosis for emergency patients who have chest pain—especially for postmenopausal women with a history of significant stressor—as early recognition can lead to better patient care.

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Conclusion

This case highlights the importance of POC ultrasound in the management of patients in the ED and after admission. The care of our patient was enhanced by the ability to take a real-time look at her EF and cardiac function at the time of admission through bedside ultrasound. This information guided her management and optimized stabilization.

References

1. Goodman A, Perera P, Mailhot T, Mandavia D. The role of bedside ultrasound in the diagnosis of pericardial effusion and cardiac tamponade. *J Emerg Trauma Shock*. 2012;5(1):72-75. doi:10.4103/0974-2700.93118.
2. Unlüer EE, Karagöz A, Akoğlu H, Bayata S. Visual estimation of bedside echocardiographic ejection fraction by emergency physicians. *West J Emerg Med*. 2014;15(2):221-226. doi:10.5811/westjem.2013.9.16185.
3. McConnell MV, Solomon SD, Rayan ME, Come PC, Goldhaber SZ, Lee RT. Regional right ventricular dysfunction detected by echocardiography in acute pulmonary embolism. *Am J Cardiol*. 1996;78(4):469-473.
4. Coen D, Cortellaro F, Pasini S, et al. Towards a less invasive approach to the early goal-directed treatment of septic shock in the ED. *Am J Emerg Med*. 2014;32(6):563-568. doi:10.1016/j.ajem.2014.02.011.
5. Dote K, Sato H, Tateishi H, Uchida T, Ishihara M. [Myocardial stunning due to simultaneous multivessel coronary spasms: a review of 5 cases.] *J Cardiol*. 1991;21(2):203-214.

6. Gopalakrishnan M, Hassan A, Villines D, Nasr S, Chandrasekaran M, Klein LW. Predictors of short- and long-term outcomes of Takotsubo cardiomyopathy. *Am J Cardiol*. 2015;116(10):1586-1590. doi:10.1016/j.amjcard.2015.08.024.
7. Paur H, Wright PT, Sikkel MB, et al. High levels of circulating epinephrine trigger apical cardiodepression in a β2-adrenergic receptor/Gi-dependent manner: a new model of Takotsubo cardiomyopathy. *Circulation*. 2012;126(6):697-706. doi:10.1161/CIRCULATIONAHA.112.111591.
8. Wittstein IS, Thiemann DR, Lima JA, et al. Neurohumoral features of myocardial stunning due to sudden emotional stress. *N Engl J Med*. 2005;352(6):539-548. doi:10.1056/NEJMoa043046.
9. Butterfield M, Riguzzi C, Frenkel O, Nagdev A. Stimulant-related Takotsubo cardiomyopathy. *Am J Emerg Med*. 2015;33(3):476.e1-e3. doi:10.1016/j.ajem.2014.08.058.
10. Baber A, Nair SU, Duggal S, Bhatti S, Sundlof DW. Stress cardiomyopathy caused by diving: case report and review of the literature. *J Emerg Med*. 2016;50(2):277-280. doi:10.1016/j.jemermed.2015.09.045.
11. Katayama Y, Hifumi T, Inoue J, Koido Y. A case of Takotsubo cardiomyopathy induced by accidental hypothermia and diabetic ketoacidosis. *BMJ Case Rep*. 2013;2013:1-3. doi:10.1136/bcr-2012-008143.
12. Bybee KA, Kara T, Prasad A, et al. Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. *Ann Intern Med*. 2004;141(11):858-865.
13. Virani SS, Khan AN, Mendoza CE, Ferreira AC, de Marchena E. Takotsubo cardiomyopathy, or broken-heart syndrome. *Tex Heart Inst J*. 2007;34(1):76-79.
14. Okura H. Echocardiographic assessment of takotsubo cardiomyopathy: beyond apical ballooning. *J Echocardiogr*. 2016;14(1):13-20. doi:10.1007/s12574-015-0271-3.
15. Naser N, Buksa M, Kusljugic Z, Terzic I, Sokolovic S, Hodzic E. The role of echocardiography in diagnosis and follow up of patients with takotsubo cardiomyopathy or acute ballooning syndrome. *Med Arh*. 2011;65(5):287-290.
16. Ono R, Falcão LM. Takotsubo cardiomyopathy systematic review: Pathophysiologic process, clinical presentation and diagnostic approach to Takotsubo cardiomyopathy. *Int J Cardiol*. 2016;209:196-205. doi:10.1016/j.ijcard.2016.02.012.
17. Opolski G, Budnik M, Kochanowski J, Kowalik R, Piatkowski R, Kochman J. Four episodes of takotsubo cardiomyopathy in one patient. *Int J Cardiol*. 2016;203:53-54. doi:10.1016/j.ijcard.2015.10.048.
18. Templin C, Ghadri JR, Diekmann J, et al. Clinical features and outcomes of Takotsubo (stress) cardiomyopathy. *N Engl J Med*. 2015;373(10):929-938.
19. Gianni M, Dentali F, Grandi AM, Sumner G, Hiralal R, Lonn E. Apical ballooning syndrome or takotsubo cardiomyopathy: a systematic review. *Eur Heart J*. 2006;27(13):1523-1529. doi:10.1093/eurheartj/ehl032.
20. Citro R, Lyon AR, Meimoun P, et al. Standard and advanced echocardiography in Takotsubo (stress) cardiomyopathy: clinical and prognostic implications. *J Am Soc Echocardiogr*. 2015;28(1):57-74. doi:10.1016/j.echo.2014.08.020.