Clinical Case Reports

Open Access

CASE REPORT

Acute gastritis-induced Takotsubo's cardiomyopathy

Pedro A. Villablanca, Shashvat Sukhal, Asimul Ansari & Dergham Mohammed

John H. Stroger, Jr. Hospital of Cook County, Chicago, 60612, Illinois

Correspondence
Pedro A. Villablanca, John H. Stroger, Jr. Hospital of Cook County, 1900 West Polk Street, 15th Floor, Chicago 60612, IL. Tel: (312) 864-7229; Fax: (312) 864-9725; E-mail: pedro_a_villablanca@rush.edu

Funding Information
No funding information provided.

Received: 21 July 2013; Revised: 8 August 2013; Accepted: 10 October 2013

Clinical Case Reports **2013; 1(2): 91–95**

doi: 10.1002/ccr3.32

Key Clinical Message

A 50-year-old lady presented with epigastralgia, electrocardiogram (ECG) showed T-wave inversions and the echocardiogram low ejection fraction (EF) with apical ballooning. An esophagogastroduodenoscopy (EGD) revealed gastritis. She recovered with proton pump inhibitors treatment. This is the first case that describes gastritis-induced stress cardiomyopathy. Clinicians should be aware of Takotsubo's cardiomyopathy (TCM) as a possible complication of gastritis.

Keywords

Acute gastritis, Takotsubo's cardiomyopathy.

Case Report

Ms. JD, a 50-year-old lady with past medical history of alcoholic hepatitis, gastroesophageal reflux disease, depression, peripheral neuropathy, and hypertension, presented to the emergency room after 2 days of vomiting and burning epigastric abdominal pain. Her last drink was 2 weeks ago. She denied any chest pain, and her baseline exercise tolerance was five to six blocks, four flights of stairs. Review of systems was otherwise negative.

On admission, she was afebrile with a blood pressure of 94/55 mmHg, heart rate 134 breaths/min, and oxygen saturation 95% on room air. Physical examination was unremarkable, except for dehydration and epigastric tenderness. Laboratory results showed an elevated white blood cell count of 14.2 × 103 cells/cubic millimeter, mild elevations in levels of lipase to 101 units/L (reference range five to 55 units/L), alanine transaminase to 74 units/L (reference range five to 35 units/L), and aspartate transaminase (AST) to 56 units/L (reference range 0–40 units/L). A computed tomography (CT) of the abdomen, on admission, was essentially normal. Notably, no pancreatic inflammation of edema was seen. An electrocardiogram (ECG), however, showed diffuse anterolateral deep T-wave inversions and prolonged QT interval, which were new compared with previous ECGs (Fig. 1). Cardiac biomarkers were positive with a troponin-I value of 0.27 ng/mL (reference range <0.034 ng/mL). Troponin trended down to 0.21 ng/mL and then to 0.083 ng/mL after 24 h. She was managed with bowel rest, intravenous hydration, and analgesia. A repeat CT of the abdomen carried out for persisting abdominal pain showed gastric wall thickening consistent with gastritis. A transthoracic echocardiogram [1] showed an ejection fraction (EF) of 40% with severe hypokinesis of the apical septal, apical lateral, and apical wall(s) with apical ballooning. She had a cardiac catheterization carried out the next day showing essentially normal coronary arteries (Figs. 2 and 3). EGD was performed showing gastritis of the antrum and fundus. Gastritis-induced TCM was suspected, as the patient had no other recent stressors. She was discharged in stable condition on drug therapy with pantoprazole, carvedilol, and enalapril. One month later, the patient was completely asymptomatic and doing well. A repeat ECG (Fig. 1) was completely normal, and a repeated transthoracic echocardiogram showed normal EF without wall motion abnormalities.

Discussion

TCM, also known as stress-induced cardiomyopathy or apical ballooning syndrome is a relatively new but increasingly reported entity. It is characterized by acute onset of symptoms and electrocardiographic alterations that mimic myocardial infarction (MI) with transient but completely reversible left ventricular dysfunction, usually

© 2013 The Authors. *Clinical Case Reports* published by John Wiley & Sons Ltd.
This is an open access article under the terms of the Creative Commons Attribution-NonCommercial License, which permits use, distribution and reproduction in any medium, provided the original work is properly cited and is not used for commercial purposes.

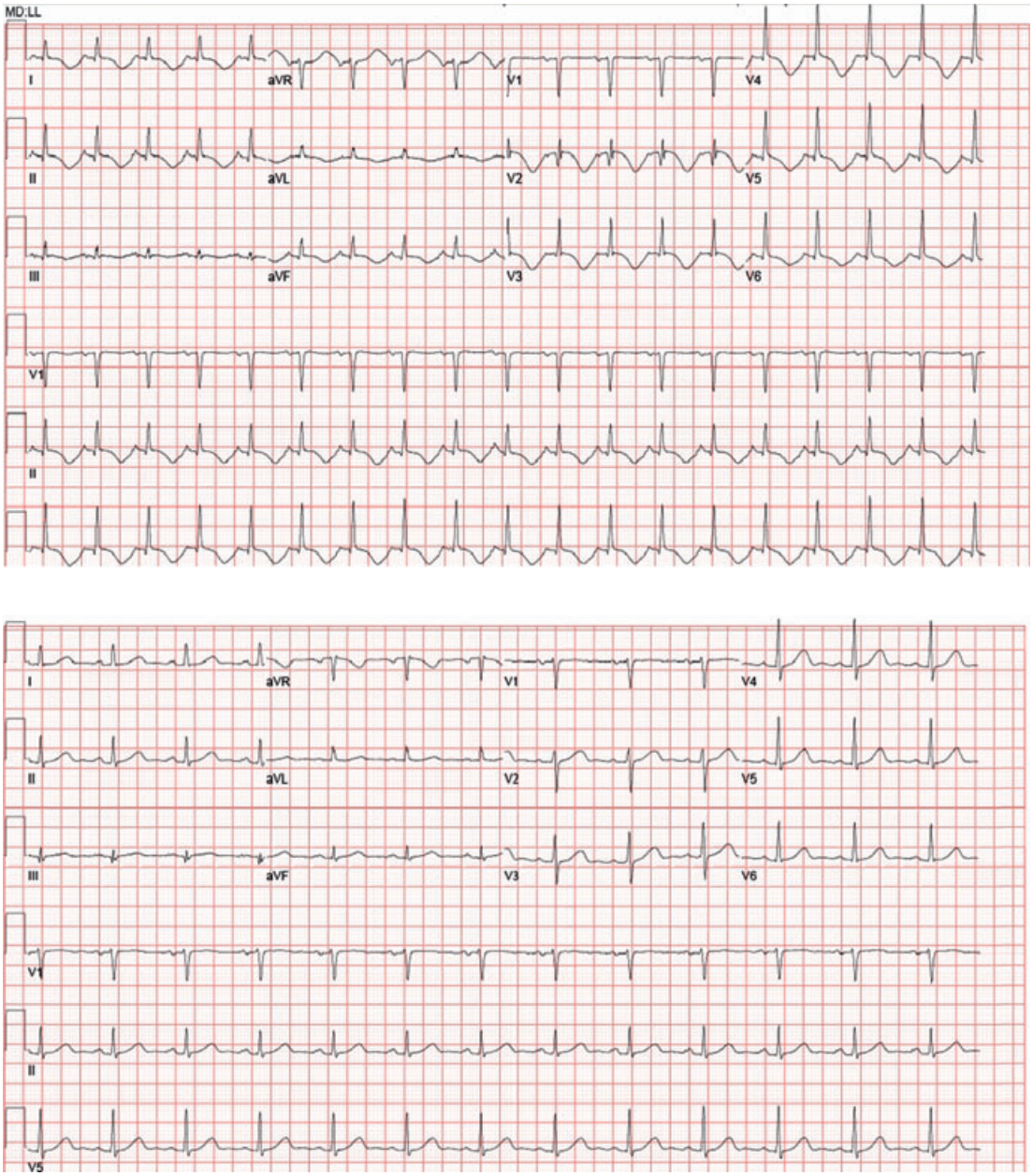

Figure 1. ECG on admission (upper panel) and repeat ECG after 8 weeks (lower panel).

following physical or emotional stress [2], but with demographic and comorbid predictors differing substantially from those of MI [3]. This syndrome presents a diagnostic challenge, given the wide spectrum of responsible physiological stressors. Numerous cases of gastrointestinal diseases or procedures have been described including recurrent vomiting, achalasia, upper digestive tract bleeding, cholecystitis, pancreatitis, cancers, all acting as triggers [4–10], but to the best of our knowledge, this patient is the first reported case of gastritis-induced TCM in literature to date.

TCM was first described in Japan and derives its name from "Takotsubo" – an octopus trapping jar, which resembles the apical akinetic heart of TCM [11]. The incidence of TCM is increasing and it approximately accounts for 2.2% of all the admissions presumed to have a diagnosis of acute MI [12]. Prevalence in the United States is about 0.02% of all hospitalizations, mostly in elderly women with history of smoking, alcohol abuse, anxiety states, and hyperlipidemia [13]. Commonly, these patients present with shortness of breath, chest pain, palpitations, diaphoresis, nausea, vomiting, or syncope. They may have ECG

© 2013 The Authors. *Clinical Case Reports* published by John Wiley & Sons Ltd.

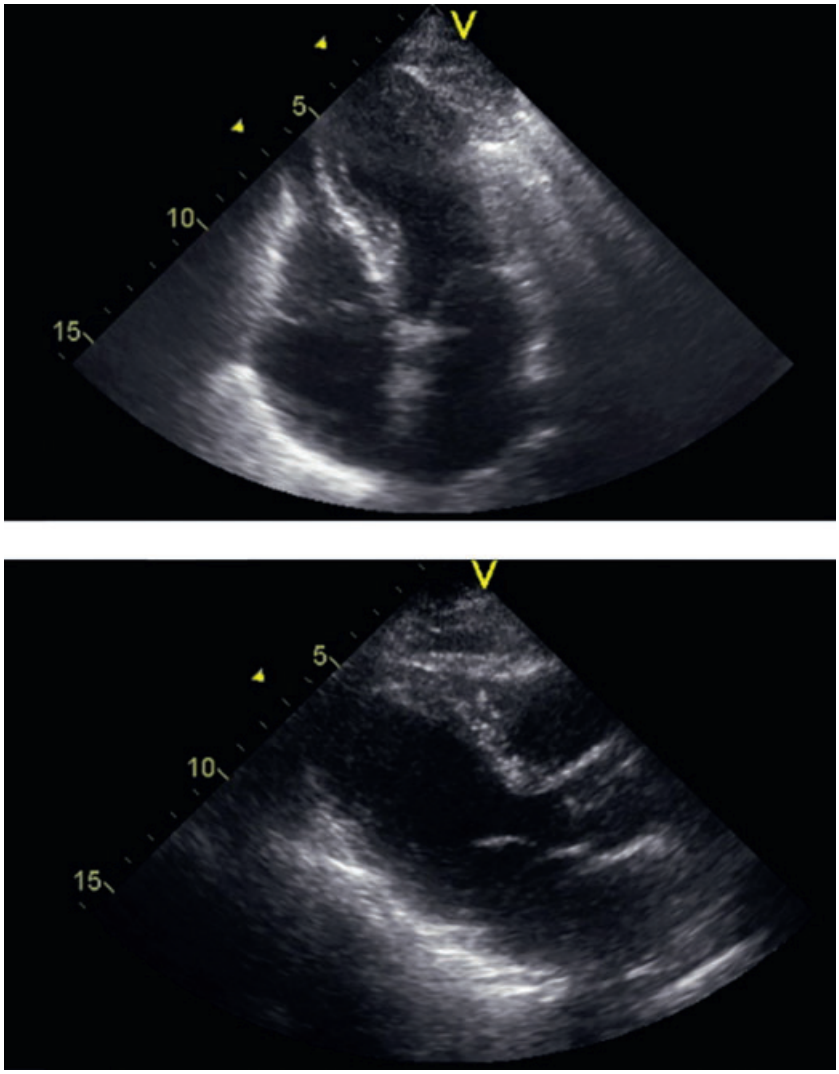

Figure 2. Echocardiogram view showing apical ballooning.

abnormalities such as, transient minimal ST-segment elevation in the precordial leads V1 through V4 and deep, symmetric T-wave inversions that involve most, if not all, precordial and limb leads with associated QT interval prolongation. On coronary angiography, either no angiographic detectable coronary lesions, or nonobstructive coronary disease is found [14]. Most patients with TCM exhibit mild elevation in levels of troponin, with absence of the typical trend in serial measurements as seen with acute coronary syndrome (ACS) [15].

Echocardiography and/or ventriculography classically reveal extensive apical and/or midventricular akinesia or hypokinesia, but with basal function preserved or hyperkinetic. Apart from the apical involvement, new variants affecting midventricular and basal segments of left ventricle have also been described. Repeat assessment 6–8 weeks later showed complete recovery of left ventricular function in terms of wall motion and LVEF. Criteria proposed by the Mayo Clinic Group are most often used for diagnosis [14].

The pathophysiological mechanisms proposed have been related to transient catecholamine surges, with either multivessel epicardial spasm or microvascular coronary spasm and possible direct myocyte injury [16–18]. Wittstein et al. [19] compared plasma catecholamine concentrations in patients with TCM and acute MI with Killip class III on presentation and found that levels of catecholamines were three times higher in TCM. There are no randomized treatment trials for this condition; however, initial diagnosis and management for an ACS are justified, including antiplatelet, anticoagulation, and urgent coronary angiography. After the diagnosis of TC has been established, therapy is mainly supportive. Beta-blockers (BB) and angiotensin converting enzyme inhibitors

© 2013 The Authors. *Clinical Case Reports* published by John Wiley & Sons Ltd.

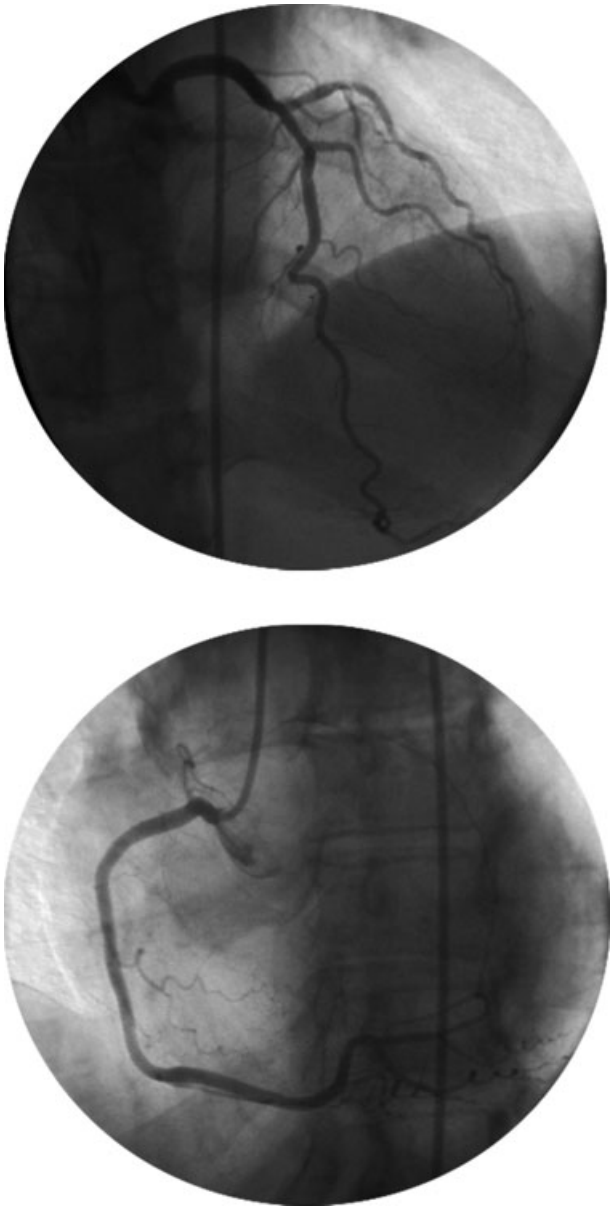

Figure 3. Cardiac catheterization with normal left and right coronary arteries.

should be started until recovery of cardiac function [20]. Mechanical circulatory support is preferred in cases of hypotension or cardiogenic shock [21]. Beta-blockers should be given as indefinite therapy in the absence of contraindications to help prevent future attacks in stressful situations [22].

Conclusions

Although various gastrointestinal conditions can trigger stress-induced cardiomyopathy, ours is the first gastritis-induced reported case. Clinicians should be aware of TCM as a possible complication of acute gastritis and providers should consider this possibility in patients with chest pain, ECG changes, or left ventricular dysfunction with typical echocardiographic appearance in patients with acute gastritis.

Conflict of Interest

None declared.

References

1. Meissner, I., J. P. Whisnant, B. K. Khandheria, P.C. Spittell, W. M. O'Fallon, R. D. Pascoe, et al. 1999. Prevalence of potential risk factors for stroke assessed by transesophageal echocardiography and carotid ultrasonography: the SPARC study. Stroke Prevention: Assessment of Risk in a Community. Mayo Clin. Proc. 74:862–869.
2. Gianni, M., F. Dentali, A. M. Grandi, G. Sumner, R. Hiralal, and E. Lonn. 2006. Apical ballooning syndrome or takotsubo cardiomyopathy: a systematic review. Eur. Heart J. 27:1523–1529.
3. El-Sayed, A. M., W. Brinjikji, and S. Salka. 2012. Demographic and co-morbid predictors of stress (takotsubo) cardiomyopathy. Am. J. Cardiol. 110:1368–1372.
4. Awais, M., R. A. Hernandez, and D. S. Bach. 2008. Takotsubo cardiomyopathy triggered by severe vomiting. Am. J. Med. 121:e3–e4.
5. Cheezum, M. K., S. L. Willis, S. P. Duffy, F. J. Moawad, J. D. Horwhat, L. L. Huffer, et al. 2010. Broken pancreas, broken heart. Am. J. Gastroenterol. 105:237–238.
6. Coutance, G., E. Cauderlier, R. Gloro, and F. Labombarda. 2010. Tako-tsubo cardiomyopathy triggered by severe achalasia. Rev. Esp. Cardiol. 63:747–748.
7. Durning, S. J., J. M. Nasir, J. M. Sweet, and L. J. Cation. 2006. Chest pain and ST segment elevation attributable to cholecystitis: a case report and review of the literature. Mil. Med. 171:1255–1258.
8. Gangadhar, T. C., E. Von der Lohe, S. G. Sawada, and P. R. Helft. 2008. Takotsubo cardiomyopathy in a patient with esophageal cancer: a case report. J. Med. Case Rep. 2:379.
9. Elikowski, W., M. Malek, W. Witczak, D. Wroblewski, T. Kozlowski, and M. Dziarmaga. 2011. [Takotsubo cardiomyopathy as a consequence of gastrointestinal disorder–a case preceded by exacerbation of gastroesophageal reflux disease]. Pol. Merkur. Lekarski 31:227–232.
10. Bibiano Guillén, C., M. T. García Sanz, F. J. Serantes Pombo, and M. J. Vázquez Lima. 2008. Transient apical dyskinesia associated with upper digestive tract bleeding. Emergencias 20:291–296.

© 2013 The Authors. *Clinical Case Reports* published by John Wiley & Sons Ltd.

11. Kawai, S., H. Suzuki, H. Yamaguchi, K. Tanaka, H. Sawada, T. Aizawa, et al. 2000. Ampulla cardiomyopathy ('Takotusbo' cardiomyopathy)–reversible left ventricular dysfunction: with ST segment elevation. Jpn. Circ. J. 64:156–159.
12. Bybee, K. A., A. Prasad, G. W. Barsness, A. Lerman, A. S. Jaffe, J. G. Murphy, et al. 2004. Clinical characteristics and thrombolysis in myocardial infarction frame counts in women with transient left ventricular apical ballooning syndrome. Am. J. Cardiol. 94:343–346.
13. Deshmukh, A., G. Kumar, S. Pant, C. Rihal, K. Murugiah, and J. L. Mehta. 2012. Prevalence of Takotsubo cardiomyopathy in the United States. Am. Heart J. 164: 66–71.
14. Madhavan, M., and A. Prasad. 2010. Proposed Mayo Clinic criteria for the diagnosis of Tako-Tsubo cardiomyopathy and long-term prognosis. Herz 35:240–243.
15. Ramaraj, R., V. L. Sorrell, and M. R. Movahed. 2009. Levels of troponin release can aid in the early exclusion of stress-induced (takotsubo) cardiomyopathy. Exp. Clin. Cardiol. 14:6–8.
16. Kurisu, S., I. Inoue, T. Kawagoe, M. Ishihara, Y. Shimatani, S. Nakamura, et al. 2004. Time course of electrocardiographic changes in patients with tako-tsubo syndrome: comparison with acute myocardial infarction with minimal enzymatic release. Circ. J. 68:77–81.
17. Abe, Y., M. Kondo, R. Matsuoka, M. Araki, K. Dohyama, and H. Tanio. 2003. Assessment of clinical features in transient left ventricular apical ballooning. J. Am. Coll. Cardiol. 41:737–742.
18. Mann, D. L., R. L. Kent, B. Parsons, and G. Cooper IV. 1992. Adrenergic effects on the biology of the adult mammalian cardiocyte. Circulation 85:790–804.
19. Wittstein, I. S., D. R. Thiemann, J. A. Lima, K. L. Baughman, S. P. Schulman, G. Gerstenblith, et al. 2005. Neurohumoral features of myocardial stunning due to sudden emotional stress. N. Engl. J. Med. 352:539–548.
20. Richard, C. 2011. Stress-related cardiomyopathies. Ann. Intensive Care 1:39.
21. Madhavan, M., C. S. Rihal, A. Lerman, and A. Prasad. 2011. Acute heart failure in apical ballooning syndrome (TakoTsubo/stress cardiomyopathy): clinical correlates and Mayo Clinic risk score. J. Am. Coll. Cardiol. 57:1400–1401.
22. Nef, H. M., H. Mollmann, and A. Elsasser. 2007. Tako-tsubo cardiomyopathy (apical ballooning). Heart 93:1309–1315.

© 2013 The Authors. *Clinical Case Reports* published by John Wiley & Sons Ltd.