

Case report

A rare presentation of stress cardiomyopathy in a patient with ulcerative colitis


Nimesha SNH¹, Jayasena H² ¹University Medical Unit, Colombo North Teaching Hospital, Ragama, Sri Lanka²Faculty of Medicine, General Sir John Kotelawala Defence University, Sri Lanka

Article Information

Corresponding Author

H Jayasena

Email: hiru65@hotmail.com

 <https://orcid.org/0000-0001-5175-0803> <https://doi.org/10.4038/sljms1.v1i1.8>

Received 03 June 2024

Revised version 13 June 2024

Accepted 19 July 2024

Conflicts of Interest: None

Abstract

Stress cardiomyopathy is a reversible cause of acute left ventricular dysfunction that can cause disease-related complications in the acute phase. Due to overlapping symptoms and investigation findings, stress cardiomyopathy can be misdiagnosed as an acute myocardial infarction. Hence correct identification of the condition is of great importance. It is increasingly being described with several medical conditions, which are associated with increased catecholamine surge like inflammatory bowel disease. Although the disease is reversible, due to the disease-related severe complications it is important to promptly identify and treat accordingly to prevent morbidity and mortality.

Keywords: chest pain, stress cardiomyopathy, ulcerative colitis,

Introduction

Stress cardiomyopathy or “Takotsubo cardiomyopathy” is a syndrome characterized by a transient systolic dysfunction of the heart mainly affecting the left ventricle, in the absence of obstructive coronary arterial disease. Whilst at most cardiac dysfunction is regional, it can also be global. Usually, the regional wall motion abnormality extends beyond the territory perfused by a single epicardial coronary artery.¹ Even though the incidence of stress cardiomyopathy is unknown, there are several reported cases of stress-related cardiomyopathy, especially in intensive care units.²

In stress cardiomyopathy acute left ventricular dysfunction occurs with or without apical ballooning on echocardiogram usually in a patient without any cardiac risk factors, which subsequently completely resolves within one to four weeks.³ It is found to be more predominant in females, with men being affected less than 10%.^{1,4} Although the pathogenesis of stress cardiomyopathy is poorly understood, it is postulated that a catecholamine excess results in these transient changes.¹ The long-term prognosis of the disease is generally good even though there are life-threatening complications during the acute

Cite this article as: Jayasena H, et al. A rare presentation of stress cardiomyopathy in a patient with ulcerative colitis. SLJMS 2024; 1(1): 46-48.



This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License, which permits unrestricted use, distribution and reproduction in any medium provided the original author and source are credited.

phase.¹ The recurrence rate is less than 10%.⁵ The clinical manifestation of the condition is like that of a myocardial infarction. Hence, patients present with chest pain, acute dyspnoea, arrhythmia, and autonomic symptoms. In most cases, it is difficult to differentiate acute myocardial infarction from stress cardiomyopathy, therefore are managed in the same manner. However, it is important to note that stress cardiomyopathy is transient and does not require long-term management as for acute coronary syndrome. Hence it prevents patients from undergoing unnecessary and prolonged treatment as well as follow-up for a long-term illness.

Ulcerative colitis (UC) is one of the two main forms of inflammatory bowel disease (IBD), which targets the gastrointestinal tract, the other being Crohn's disease.⁶ Both diseases are relatively common in developed countries.⁷ Stress cardiomyopathy associated with UC is a rare combination where only a few cases have been documented.

Here, we discuss a rare case of a patient with acute severe exacerbation associated with UC.

Case report

A 42-year-old married Sinhalese lady who was previously well, presented with an episode of severe exacerbation of UC. She had initially presented to a tertiary care centre a month prior with mucous, bloody diarrhoea of increasing frequency, where she was diagnosed with UC. There she was treated with high doses of intravenous steroids and intravenous cyclosporin but had failed to improve.

On this admission, she was found to be febrile with a pulse rate of 120 beats per minute and a blood pressure of 130/90 mmHg. She appeared unwell, pale and dehydrated. On abdominal examination, there was generalized tenderness throughout the abdomen, without any guarding or organomegaly. An erect abdominal x-ray showed dilated cecum at around 4 cm. Her serological investigations revealed a normal white cell count of $8.74 \times 10^6/\text{mL}$, but haemoglobin was reduced at 8.5 g/dL and CRP level was raised at 83 mg/L. A colonoscopy found the colonic mucosa to be diffusely inflamed and ulcerated up to the cecum, with a MAYO endoscopy score of 3, indicating severe inflammation.

She received intravenous steroids with steroid enema and high-dose mesalazine initially, along with intravenous broad-spectrum antibiotics and intravenous saline. Her haemodynamic status was maintained with intravenous fluids and blood replacement, with no requirement for inotropes at any point. However, despite the treatment she continued to experience persistent symptoms 4-6 times of mucous, bloody diarrhoea with fever and tachycardia.

She was frequently teary with low mood and was diagnosed with moderate depression following a psychiatry review.

On the 6th day of admission, the patient developed a sudden onset of dyspnoea with orthopnoea, which was associated with mild central chest pain without radiation. There were bi-basal fine crepitations heard on chest examination. The electrocardiogram (ECG) showed the presence of lateral T wave depressions in V4 to V6 chest leads which was initially normal at presentation. She was also found to have a positive Troponin I level of 0.978 ng/mL. A transthoracic echocardiogram (TTE) showed the presence of dilated cardiomyopathy with severe left ventricular dysfunction and apical ballooning with an ejection fraction of 25%. There was no myocarditis or pericarditis noted. A coronary angiogram was not performed owing to acute gastrointestinal bleeding. The patient was medically managed in the intensive care unit owing to worsening clinical status. For underlying UC, she received her first dose of infliximab 300 mg with a plan to repeat infusions at 2, 6 and 8 weeks. Gradually, the tachycardia settled with the improvement of her bowel symptoms. She remained afebrile. A repeat TTE done on day five in the intensive care unit showed complete resolution of earlier changes with a healthy ejection fraction of 60%. There appeared to be no ischemic changes or wall motion abnormalities present. Given the fact that ejection fraction and hypokinesia improved quickly, it was deemed that underlying stress cardiomyopathy was the likely cause for transient ischaemia.

Despite use of biological therapy, she failed to respond to medical treatment. Hence, following a multidisciplinary team discussion, she underwent a total proctocolectomy without any cardiological consequences. The patient was soon mobilized and was discharged home after one week and arranged follow-up at the gastroenterology clinic.

Discussion

Stress cardiomyopathy is frequently documented to be associated with intense physical and emotional stress.² The possibility of stress cardiomyopathy should be suspected in all adults presenting with features of acute coronary syndrome, especially when the clinical manifestation, clinical picture and ECG findings are not compatible with the level of elevation of cardiac biomarkers like Troponin I as in the case. The most common presentation of stress cardiomyopathy is retrosternal chest pain with other symptoms being shortness of breath, syncope, arrhythmia, acute mitral regurgitation, and sudden cardiac death.^{1,2}

Stress cardiomyopathy is diagnosed using the Mayo Clinic diagnosing criteria and all four criteria should be present along with a clear stressful trigger to make the diagnosis.¹

1. In transient left ventricular systolic dysfunction (hypokinesia, akinesia, or dyskinesia) the wall motion abnormalities are typically regional rarely involve focal and global types frequently but are not always associated with a stressful trigger.
2. Absence of obstructive coronary disease or angiographic evidence of acute plaque rupture.
3. New electrocardiographic abnormalities or modest elevation in the cardiac Troponin.
4. Absence of pheochromocytoma or myocarditis.

In our patient during the hospital admission, she had features of acute heart failure with dynamic ECG changes, positive Troponin I with TTE finding of global hypokinesia, and severe left ventricular dysfunction that improved completely after five days. There was no clinical or echocardiographic evidence of myocarditis or pericarditis. There was no collateral history or other evidence to suggest pheochromocytoma. The need for an urgent angiogram in this patient was deemed low as she had continued acute gastrointestinal bleeding, which is a relative contraindication to angiogram, as well as her cardiac symptoms resolved along with her ECG and echocardiogram changes spontaneously in less than 21 days.¹ In the above case, the patient had a background acute severe exacerbation of UC that was ongoing for nearly four weeks before admission. Additionally, she was diagnosed with moderate depression later. Whilst IBD has multiple extraintestinal manifestations, affective disorders (especially among women) also feature highly.⁸ It is thought that in those with anxiety or depression, the catecholamine effects are pronounced.⁹ Hence it is possible that the prolonged course of illness, together with poor mental health, may have contributed to the patient developing stress cardiomyopathy. Thus, we could make the clinical diagnosis of stress cardiomyopathy also known as Takotsubo cardiomyopathy, associated with severe exacerbation of UC in this patient.

There are increasing numbers of patients being diagnosed with IBD at present. Therefore, an uncommon presentation such as this must be suspected in those presenting with IBD and chest pain. In fact, there are several similarities between the two conditions of stress cardiomyopathy and IBD. Both have endothelial dysfunction and associated affective disorders.¹⁰ Hence having UC with increased catecholamine surge can precipitate an event of stress cardiomyopathy in susceptible individuals.¹⁰ However, it is also worth appreciating that the atherosclerosis risk is also high among patients with chronic illness. Thus, they are anyway at a high risk of having true myocardial infarction. Currently, it is important to identify the uncommon complications of the diseases such as stress cardiomyopathy, when treating patients so that prevention, timely identification, and the ideal management of the conditions can be carried out.

Conclusions

In patients who are in acute medical stress situations such as severe exacerbation of UC, presence of chest pain, shortness of breath with acute ECG changes, and positive Troponin I, the possibility of stress cardiomyopathy must be considered.

Author Contribution

All authors equally contributed to the study.

References

1. Hurst RT, Prasad A, Askew JW, et al. Takotsubo cardiomyopathy: a unique cardiomyopathy with variable ventricular morphology. *JACC: Cardiovascular Imaging* 2010; **3**(6): 641-9.
2. Desmet WJ, Adriaenssens BF, Dens JA. Apical ballooning of the left ventricle: first series in white patients. *Heart*. 2003; **89**(9): 1027-31.
3. Bybee KA, Kara T, Prasad A, et al. Systematic review: transient left ventricular apical ballooning: a syndrome that mimics ST-segment elevation myocardial infarction. *Annals of Internal Medicine* 2004; **141**(11): 858-65.
4. Prasad A, Lerman A, Rihal CS. Apical ballooning syndrome (Tako-Tsubo or stress cardiomyopathy): a mimic of acute myocardial infarction. *American Heart Journal* 2008; **155**(3): 408-17.
5. Loftus CG, Loftus Jr EV, Harmsen SW, et al. Update on the incidence and prevalence of Crohn's disease and ulcerative colitis in Olmsted County, Minnesota, 1940-2000. *Inflammatory Bowel Diseases* 2007; **13**(3): 254-61.
6. Hanauer SB. Inflammatory bowel disease: epidemiology, pathogenesis, and therapeutic opportunities. *Inflammatory Bowel Diseases*. 2006; **12**(suppl_1): S3-9.
7. Gutierrez CH, Aude W. Clinical Features and Outcomes Of Takotsubo (Stress) Cardiomyopathy In South Texas Hispanics. *Journal of the American College of Cardiology* 2020; **75**(11_Supplement_1): 1067.
8. Vlachos II, Barbatis C, Tsopanomichalou M, Abou-Assabeh L, et al. Correlation between depression, anxiety, and polymorphonuclear cells' resilience in ulcerative colitis: the mediating role of heat shock protein 70. *BMC Gastroenterology*. 2014; **14**(1): 1-0.
9. Nutt D, Demyttenaere K, Janka Z, et al. The other face of depression reduced positive affect: the role of catecholamines in causation and cure. *Journal of Psychopharmacology* 2007; **21**(5): 461-71.
10. Mirijello A, D'Errico MM, Curci S, et al. Takotsubo Syndrome and Inflammatory Bowel Diseases: Does a Link Exist?. *Digestive Diseases*. 2020; **38**(3): 204-10.