Development of Takotsubo Cardiomyopathy in 46-Year-Old With Refractory Crohn's Disease

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Abstract
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Keywords
Takotsubo cardiomyopathy, Crohn's disease, inflammatory bowel disease

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**Development of Takotsubo Cardiomyopathy in 46-Year-Old With Refractory Crohn's Disease**

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**Abstract**

Rampant autoimmune disease has the potential to ravage the entirety of the body in a systemic fashion. Rarely, it has been reported for Takotsubo cardiomyopathy (TCM) as a result of refractory inflammatory bowel disease. Balancing the comprehensive cares required to support both the cardiovascular system and treat the underlying autoimmune condition care present unique challenges. Here, we describe a patient who developed late-onset Takotsubo cardiomyopathy after prolonged systemic stress as a result of uncontrolled Crohn’s disease. While this patient unfortunately did succumb to the disease process, it is our hope that highlighting these rare cases may progress care for future patients.

**Keywords**

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**Introduction**

Crohn’s disease is an idiopathic inflammatory bowel disease (IBD) characterized by transmural inflammation of the gastrointestinal tract, along with a variety of extraintestinal manifestations. Cardiovascular manifestations of Crohn’s disease include coronary artery disease, myocardial infarct, thrombomembolism, valvular disease, arrhythmias, and both ischemic and non-ischemic cardiomyopathies.1,2 IBD-induced systemic inflammation contributes to atherogenic processes and underlies the increased incidence of acute cardiac dysfunction in these patients.2,4 Specifically, the incidence of coronary artery disease has been reported to be as much as 9% greater in patients with IBD as compared to the general population.1 Furthermore, the incidence of acute myocardial infarct can be 20% greater in patients with IBD compared to the general population.1 Autoimmune disease has been shown to be a non-genetic etiology of non-ischemic cardiomyopathy.2 Takotsubo cardiomyopathy (TCM) is an acute non-ischemic cardiomyopathy characterized by left ventricular systolic dysfunction and cardiac biomarker elevation triggered by significant psychological stress.2 TCM remains a poorly understood atypical cardiomyopathy, and its potential association with autoimmune diseases (such as IBD) necessitates further exploration. Here, we detail the presentation of TCM in a patient with prolonged course of refractory Crohn’s disease.

**Case**

The patient was a 46-year-old female with a past medical history of uncontrolled Crohn’s disease. At the time of presentation, she was being treated with vedolizumab due to incomplete disease control after treatment with budesonide and mesalazine in 2019. She had a history of poor disease control, characterized by abdominal pain, frequent stooling, blood per rectum, and weight loss. Notably, she had a self-reported history of tachycardia, with a resting heart rate typically between 90-110s beats per minute. The patient had a complex surgical history including ileocelecectomy for management of Crohn’s disease, followed by small bowel obstruction requiring enterolysis and ileostomy, with eventual ileostomy reversal. Her post-surgical course was complicated by sepsis. The patient continued to be managed on vedolizumab, budesonide, and mesalazine after reporting significant abdominal pain 1-year post-ileocecectomy and ileostomy. She ultimately required right-to-left transureterouterostomy as a result of intraoperative injury to the right ureter with multiple failed attempts are repair. This was further complicated by recurrent urinary tract infections, which in turn required multiple nephrostomy tubes and chronic antibiotic suppression. This ultimately led to the decision to pursue a retroperitoneal right nephrectomy after a short interval of good control of her Crohn’s symptoms, when her fecal calprotectin entered a normal range.

The postoperative course following right nephrectomy was complicated by small bowel obstruction and complex peritonitis due to a combination of intraabdominal adhesions and the long-standing Crohn’s disease. This required multiple trips to the operating room for exploration, adhesiolysis, additional bowel resection, and ileostomy; the course was further complicated intestinal leak requiring placement of multiple drains by interventional radiology as the patient was left with a frozen abdomen. The patient slowly made progress and was stable for placement to a rehabilitation facility by postoperative day 36. Unfortunately, she had unexpected decompensation on postoperative day 38, with hypotension to systolic blood pressure of 90, and an increase in her baseline tachycardia to a heart rate of 140, at which point an electrocardiogram was obtained revealing an ST segment elevation myocardial infarction. Troponin was trended to a peak of near 9000 ng/L, and lactate was elevated at 7.7 mmol/L. Echocardiography revealed global hypokinesis with an ejection fraction of 10-15%. Supportive measures were performed as the patient had progressive hemodynamic deterioration, and she was taken for left heart catheterization. No actionable stenosis was noted, and the concern for TCM was raised. In light of this acute onset cardiac dysfunction, the family ultimately made the decision to proceed with comfort measures.

**Discussion**

Here, we describe a 46-year-old female with longstanding refractory Crohn’s disease, who underwent right nephrectomy in attempted definitive management of intractable urinary tract infections secondary to ureteral stenosis. Her postoperative course was complicated by sepsis and the ultimate development of TCM. The in-hospital mortality rate for patients who develop TCM is 4.2%, with mortality being driven by critical illness in over 80% of cases.3 Risk factors for TCM-induced sudden cardiac death include QT-interval prolongation.8 Crohn’s disease has been associated with the development of acute cardiovascular disorders, including ischemic and non-ischemic cardiomyopathy. In patients with Crohn’s disease, atherogenesis is mediated by IBD-induced endothelial dysfunction, endotoxin and inflammatory mediator dysregulation, coagulation cascade abnormalities, and disturbed lipoprotein profiles, leading to ischemic cardiomyopathies.4 Autoimmune disorders are a non-genetic etiology of non-ischemic cardiomyopathy. The risk of developing non-ischemic cardiomyopathies has been found to increase following Crohn’s related hospitalization. Furthermore, patients with autoimmune disease have an increased risk of developing cardiomyopathy if hospitalized for more than 7 days.3 The pathogenesis of Takotsubo cardiomyopathy is not well-understood, but one theory involves extreme physical or psychological stressors that cause an excess of catecholamines, leading to microvascular dysfunction or direct myocardial
This patient’s acute illness and extensive surgical course likely precipitated an increase in circulating catecholamines that may have contributed to the development of TCM. While rare, the development of atypical acute cardiac decompensation in the setting of IBD exacerbation similar to this case has been documented. Baseline cardiac anomalies such as tachycardia in patients with chronic or critical illness should be considered as a potential harbinger of propensity to develop TCM in a system-stressing critically ill state.

References


