Case Report

Takotsubo cardiomyopathy following laparoscopic port placement in a patient with ovarian cancer⁎,☆,☆☆

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Introduction

Takotsubo cardiomyopathy (TC), also referred to as transient left ventricular apical ballooning syndrome or stress-induced cardiomyopathy, is characterized by an acute left ventricular wall dysfunction with apical left ventricular ballooning. The disease is associated with both postmenopausal status and preceding emotional and physical stress (Gianni et al., 2006).

No universal consensus has been reached on the diagnostic criteria for TC. The most commonly used criteria were proposed by the Mayo Clinic in 2004 and later were modified in 2008. Patients must satisfy all criteria. They include the following: 1) transient hypokinesis, akinesis or dyskinesis in the left ventricular mid segments with or without apical involvement; regional wall motion abnormalities that extend beyond a single epicardial vascular distribution; and frequently, but not always a stressful trigger; 2) the absence of obstructive coronary disease or angiographic evidence of acute plaque rupture; 3) new ECG abnormalities or modest elevation in cardiac troponin; 4) and the absence of pheochromocytoma and myocarditis (Akashi et al., 2008).

Classically, TC patients present with acute chest pain and dyspnea, symptoms that mimic acute coronary syndrome (ACS) (Gianni et al., 2006). New electrocardiographic (EKG) abnormalities, most commonly ST-segment elevations in the precordial leads, frequently are observed at time of presentation, as are minor elevations in cardiac enzymes (Gianni et al., 2006). Despite these symptoms and findings, patients with TC differ from those with ACS because no significant obstructive coronary artery disease is detected on angiography (Gianni et al., 2006).

Commonly, severe psychological or physical stressors precede symptoms of TC. In a systematic review of all cases published to date, 27% of patients diagnosed with TC reported a preceding emotional stressor, while 38% of patients identified a physical stressor (Gianni et al., 2006). Additionally, composite reviews demonstrate an 82–100% female predominance with mean age ranges of 58–77 years (Gianni et al., 2006). Although the exact mechanism remains unknown, the finding of elevated catecholamine levels has prompted the theory that exaggerated sympathetic stimulation plays a key role in the myocardial stunning and subsequent cardiac dysfunction observed in TC (Wittstein et al., 2005).

The majority of TC resolves spontaneously within a week and typically patients fully recover, with an in-hospital mortality rate as low as 1–3% (Gianni et al., 2006). Although uncommon, life-threatening complications of this condition, including cardiogenic shock and systemic embolization, can occur during the acute state (Y-Hassan and Shahgaldi, 2011). We report a case of TC complicated by systemic embolization and ultimately death in a postmenopausal gynecologic oncology patient following a minor laparoscopic procedure.

Case

A 53 year-old postmenopausal woman with Stage IIIc, Grade 3, clear cell ovarian cancer with a history of an optimal surgical cytoreduction procedure four weeks prior, presented for laparoscopic intraperitoneal port placement. The patient had no prior history of hypertension, coronary artery disease or any cardiovascular dysfunction. Her preoperative electrocardiogram revealed no abnormalities and she had no previous echocardiograms. An uncomplicated subclavian intravenous port placement occurred two days prior.

The patient underwent an uncomplicated laparoscopic intraperitoneal port placement under general anesthesia. She received medical and mechanical thromboembolism prophylaxis. Her immediate post-operative recovery was notable only for nausea for which she remained hospitalized overnight. On post-operative day (POD) 1, the patient became tachypneic, tachycardic, and hypoxic with mental status changes and confusion. An arterial blood gas revealed respiratory alkalosis. An
EKG demonstrated sinus tachycardia with new 1.5 mm ST-segment elevations in precordial leads V3–V6, evolving to 2.5 mm inferiorly compared with her pre-operative EKG. Given concern for an acute coronary syndrome versus pulmonary embolus, the patient was transferred to the intensive care unit.

Chest X-ray revealed no evidence of pulmonary edema, pneumothorax or widened mediastinum. Echocardiogram showed a dilated left ventricle with a decreased ejection fraction (EF) of 15–20% with wall-motion abnormalities, concerning for a dilated cardiomyopathy. Cardiac enzymes were elevated with CK 509 U/L (normal 26–140 U/L), CK-MB 35.0 ng/ml (normal 0–5 ng/ml), and troponin I 19.89 ng/ml (normal 0.0–0.4 ng/ml). No obstructive atherosclerotic disease was found during cardiac catheterization. However, left ventriculography confirmed apical ballooning of the left ventricle with a left ventricular ejection fraction of 10–15% and a cardiac index of 1.6 L/min/m². Pulmonary angiography excluded a pulmonary embolus. The possible explanations for her dilated cardiomyopathy included a pulmonary embolus and Takotsubo’s cardiomyopathy. Given the classic TC findings on echocardiogram, her new EKG changes, presence of a stressful trigger, and no evidence of coronary artery disease or pulmonary embolus, the patient was diagnosed with TC. With concern for impending cardiogenic shock, an intra-aortic balloon pump was placed to augment her cardiac function and she was started on heparin, clopidogrel, and aspirin for anticoagulation.

Following aortic balloon pump placement, the patient required vasopressor support. By POD 6, she was weaned from hemodynamic support and the intra-aortic balloon pump was removed. Her echocardiogram demonstrated an improved ejection fraction of 40%, but persistent left ventricular segmental wall-motion abnormalities.

Despite improvement in her cardiac function, the patient developed heparin-induced thrombocytopenia and disseminated intravascular coagulopathy. Imaging revealed disseminated thromboemboli, which ultimately caused hemorrhagic stroke with subsequent herniation. Life sustaining care was withdrawn and followed by death on POD 12.

Comment

Although TC is most prevalent in postmenopausal women presenting with acute emotional or physical stress, only a single case report exists in the gynecologic literature (Griffith et al., 2010) and to our knowledge there is only a single case describing a gynecologic oncology patient (Shah and Wallis, 2010).

Reports describe several different causes of severe physical and emotional stress precipitating TC (Gianni et al., 2006). Perioperative physical stresses related to cyoreductive surgery as well as intravenous and intraperitoneal port placements may have contributed to our patient’s development of TC; however, emotional stress related to both her cancer diagnosis and treatment was also likely a contributor.

Patients diagnosed with ovarian cancer have been shown to have significantly high rates of anxiety and depression (Bodurka-Bevers et al., 2000). Aside from the high likelihood of cancer recurrence, other stressors can include changes in their home and family life and fear of disease or death. Prior retrospective analysis of patients diagnosed with TC found that 68% had a prior diagnosis of either anxiety or depression, which was significantly higher than patients diagnosed with myocardial infarction. This suggests that patients with chronic psychological stress are at risk of developing this condition, which is then triggered by an acute stressor (Wittstein et al., 2005).

In prior systematic reviews, 88–100% of reported cases of stress-induced cardiomyopathy occur in postmenopausal women (Gianni et al., 2006). The pathophysiology behind the female predominance has yet to be identified. A current hypothesis suggests that the lack of estrogen in the postmenopausal state creates an increase in sensitivity to circulating catecholamines and a decrease in production of cardio-protective substances (Kubo et al., 2010). This is of interest in our gynecologic oncology population; many of the patients are either menopausal or become menopausal due to surgical interventions. Additionally, Burgdorf and colleagues report an incidence of cancer in 23.6% of patients in their cohort of 191 patients with TC. This exceeds the expected prevalence of cancer in age-matched populations and suggests malignancy may contribute to the pathophysiology of TC (Burdorf et al., 2008).

Although most patients recover from stress-induced cardiomyopathy, our patient’s cancer-related hypercoagulable state and development of HIT contributed to her thromboemboli formation and ultimately her death. Gynecologic oncology patients appear to represent an at-risk population for the development of TC. These patients are postmenopausal and exposed to both chronic and acute physical and emotional stress. As such, TC is potentially under-reported and is an important condition to consider in patients in this population who present with severe cardiopulmonary dysfunction.

Written informed consent was obtained from the patient’s spouse for publication of this case report. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Conflict of interest statement

The authors declare that there are no conflicts of interest.

References


