Acute gastritis-induced Takotsubo’s cardiomyopathy

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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 $14.2 \times 10^3$ cells/cubic millimeter, mild elevations in levels of lipase to 101 units/L (reference range 5 to 55 units/L), alanine transaminase to 74 units/L (reference range 5 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 tended 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...
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

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

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