Case Reports

Necrotic Bowel Induces Takotsubo-Like Myocardial Injury

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Although chest pain in association with ST-segment electrocardiographic deviation is often indicative of cardiac ischemia, it has also been associated with noncardiac conditions. The case of a 63-year-old woman that we report here is extraordinary because her presentation of "acute abdomen" did not appear severe enough to warrant urgent surgical intervention, whereas the chest pain and electrocardiographic changes (supported by rising troponin levels) created strong clinical suspicion of acute coronary syndrome. Was the evidence of cardiac ischemia a primary event, or was it a sequela of an acute surgical condition? Noncardiac surgical cases associated with evidence of myocardial injury can be extremely challenging from a diagnostic and management perspective. We believe that the accuracy of the clinical diagnosis is crucial to a well-considered approach. ***(Tex Heart Inst J 2014;41(6):638-40)***

Patients who report chest pain receive priority attention from emergency department (ED) providers—especially when that pain is associated with electrocardiographic (ECG) changes and with cardiac biomarkers consistent with acute coronary syndrome (ACS). In these cases, a consulting cardiologist needs to quickly decide whether to transfer the patient for invasive heart catheterization. We report a challenging ED clinical situation in which cardiac injury and "acute abdomen" occurred simultaneously. Despite time constraints, a thorough and comprehensive patient evaluation must be performed in order to avoid unnecessary invasive interventions.

Key words: *Abdomen, acute; bowel, necrotic; diagnosis, differential; echocardiography; electrocardiography/abnormalities; radiography, abdominal; small bowel/obstruction; takotsubo cardiomyopathy; troponin/abnormalities*

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Case Report

In July 2012, a 63-year-old woman presented at our emergency room with abdominal pain that had started several hours earlier. An abdominal computed tomogram showed signs of colonic thickening, thereby raising suspicion of ischemic colitis. As evaluated by a surgeon, the patient's clinical status (no guarding or abdominal rebounding), hemodynamic results, and laboratory values (normal lactic acid) did not warrant emergent surgical management. Therefore, the patient was at first treated in the ED via supportive measures that included pain management and the administration of fluids and antibiotics. Six to seven hours later, she reported chest pain. Cardiac enzyme tests, including retroactive measurements of troponin T from the first laboratory draw, showed abnormal levels of troponin T, rising from 0.13 to 0.23 ng/mL (negative <0.03 ng/mL), together with a normal creatine kinase level and an abnormal creatine kinase-MB fraction at 10.5 ng/mL (normal, <5 ng/mL). An ECG showed a 1- to 2-mm ST elevation in leads V_2 and V_3, which was sufficiently suspect to trigger an urgent cardiology consultation to evaluate the possible need for cardiac catheterization in advance of abdominal surgery.

Laboratory data showed a white-cell count of 8.5 $\times 10^9$/L, a hematocrit of 36.5%, and a platelet count of 191 $\times 10^9$/L. Two serial measurements of lactic acid were normal. Abdominal computed tomography with contrast agent revealed mildly dilated fluid-filled loops of small bowels, possible small-bowel obstruction secondary to adhesions, and a small area of nondistended small bowel between 2 distended loops.

At our bedside evaluation, the patient was very uncomfortable because of diffuse severe pain in the mid-epigastric and lower sternal areas, associated with diaphoresis and nausea. Vital signs were a temperature of 98.3 °F, a heart rate of 90 beats/min, a respiratory rate of 18 breaths/min, an oxygen saturation of 98% on 2 L/min oxygen

therapy through a nasal cannula, and a blood pressure of 90/56 mmHg, down from 130/70 mmHg at first presentation in the ED (although the blood pressure had remained fairly stable in the last hour, while the patient received the 2nd liter of normal saline solution intravenously). The first ECG at 3:25 PM (Fig. 1) showed sinus rhythm, a heart rate of 80 beats/min, significant loss of R waves, a Q wave in lead V_3, and borderline (<1 mm) ST-elevation in leads V_2 and V_3. These later abnormalities had not been present in the patient's ECG 2 years earlier. The 2nd ECG, performed at 11:25 PM (Fig. 2), when the heart rate was up to 92 beats/min, showed slightly more prominent ST elevation, Q waves in leads V_2 and V_3, and developing new T-wave inversions extending from leads V_2 and V_4, possibly indicative of dynamic ischemic changes. It is of interest that there were no reciprocal changes in the inferior leads and that the QTc interval had increased from 418 ms on admission to 470 ms. Upon our first evaluation, we concluded that the ECG abnormalities did not fulfill the criteria for ST-elevation myocardial infarction, although we were concerned that the evolving pattern and rising troponin level might be indicative of an underlying ACS.

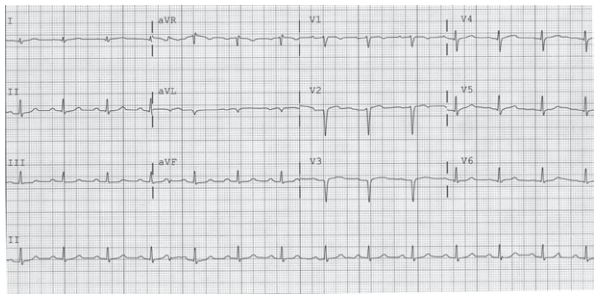

Fig. 1 *Electrocardiogram upon the patient's emergent presentation with abdominal pain shows borderline ST-elevation in leads V_2 and V_3 and significant loss of R waves in the precordial leads, not present in an electrocardiogram 2 years earlier.*

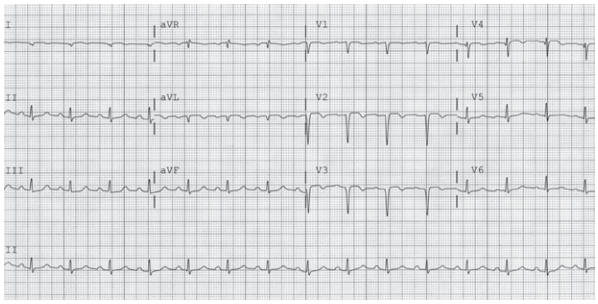

Fig. 2 *Electrocardiogram 8 hours after presentation, during patient's chest pain, shows slightly more prominent ST-segment elevation (≤1 mm), Q waves in leads V_2 and V_3, and the development of new T-wave inversion extending from leads V_2 to V_4, without reciprocal changes in the inferior leads. The QTc interval has increased to 470 ms, from 418 ms 8 hours earlier.*

We decided to perform bedside echocardiography to evaluate the patient for acute and specific wall-motion abnormalities. The results showed severe hypokinesis of the mid-distal left ventricular wall with preserved contractility of the basal portion, which was inconsistent with ischemia in a specific territory of any main coronary artery (Fig. 3). The likelihood of stress-induced takotsubo cardiomyopathy (TCM) was high in this woman who was undergoing severe physiologic stress while displaying these left ventricular wall-motion abnormalities and ECG changes. Although we were aware of the limitations of a bedside noninvasive study in evaluating coronary artery disease, our recommendation was not to pursue cardiac catheterization, but to treat the primary problem, the acute abdomen. The patient was admitted to the surgical intensive care unit (ICU) with co-management by a surgery and cardiology team that closely monitored clinical, hemodynamic, and laboratory data. The 3rd set of cardiac enzyme tests, at 4:30 AM, showed troponin slightly up (to 0.29 ng/mL) and the creatine kinase-MB fraction up (to 15 ng/mL), with normal creatine kinase. The ECG at 6:25 AM —15 hours after the first one—showed a prolongation of QTc up to 500 ms.

Continual dialogue among the physicians who cared for the patient throughout the night indicated their ongoing concern in regard to her management—specifically, they discussed whether she should first have received surgery or cardiac catheterization, and the urgency of that decision. In the early morning, the surgical attending physician thought that we were dealing with a surgical abdomen and decided to take the patient to the operating room. Abdominal exploration revealed approximately 2 feet of necrotic small bowel, herniated and encapsulated by an adhesive band. This was resected without major sequelae. The patient recovered well during a 5-day ICU stay. While she was in the

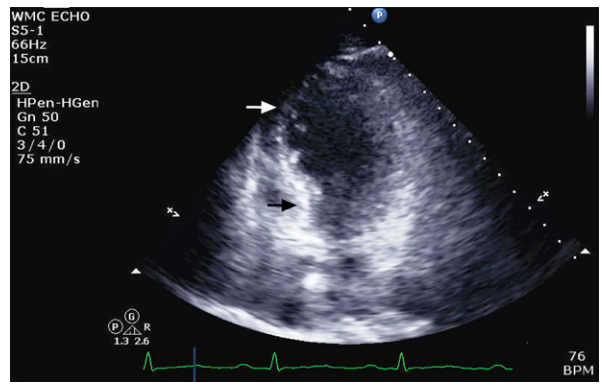

Fig. 3 *Two-dimensional bedside echocardiogram (4-chamber apical view) shows balloon-like configuration of severely hypokinetic mid-distal left ventricular wall (white arrow) with preserved contractility of the basal portion (black arrow) at end-systole.*

Supplemental motion image is available for Figure 3.

ICU, a series of echocardiograms supported our initial impression of TCM. Once stable, 7 days after her initial presentation, the patient underwent a diagnostic heart catheterization that did not reveal significant coronary lesions. The patient's cardiac condition has since remained stable. Subsequent echocardiograms, the last performed in July 2014, have shown normalization of her left ventricular dysfunction.

Discussion

The literature offers several cases of surgical conditions that mimic ECG changes classic for myocardial infarction.[1-4] Although rare, there are also reports of patients who have presented with abdominal pain complicated by TCM.[5-7] This case was a challenge from both the diagnostic and management perspectives.

On the one hand, the primary condition (small-bowel obstruction) did not present with classic "surgical" clinical features, and the ECG changes (together with the troponin elevations) further hindered its prompt recognition and management as a surgical condition. On the other hand, a causal relationship between possible ischemic colitis and myocardial injury was probably not a consideration, especially because significant hypotension was not present. The literature provides evidence that a distinct group of patients with ischemic colitis and simultaneous myocardial infarction have significantly more sequelae and worse in-hospital prognoses.[8] Thus, it becomes important to make a distinction between myocardial injury in association with stress (as in our case) and myocardial infarction in association with thrombus (a much worse prognosis).

Our case is a very rare presentation of small-bowel strangulation by adhesions, which caused bowel-wall infarction and induced TCM in a setting of extreme physical stress. It should be noted that the ECG changes in this 63-year-old woman—including T-wave evolution, borderline ST-segment elevation in leads V_2 and V_3 in the absence of inferior reciprocal changes or Q waves and QTc prolongation—were in line with the latest published criteria for differentiating TCM from acute myocardial infarction.[9-11] Accurate and prompt diagnosis has substantial prognostic implications in view of the evidence that, in TCM patients, the underlying critical illness is the main driver of mortality rates.[12] A recent case report heightens the importance of physicians' awareness of TCM sequelae in hypovolemic patients: the implication for aggressive fluid resuscitation is obvious.[13] The pathophysiologic mechanism of TCM development in our patient was very probably the catecholamine surge in the setting of acute abdomen and hypovolemia.

Our case is helpful in raising awareness of surgical conditions that are complicated by typical features of "classic" myocardial ischemia. This case proves how essential bedside echocardiographic evaluation has become for acute management, especially in the ED. This rather quick and noninvasive diagnostic evaluation prevented the patient from undergoing an invasive heart catheterization with its attendant substantial sequelae and morbidity burden, given the patient's underlying condition.

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