

A soccer mom with chest pain: Spontaneous coronary artery dissection in a young woman

BENJAMIN BLACKWOOD, MD; STEFI LEE, MD; OTTO LIEBMANN, MD; WILLIAM BINDER, MD, MA, FACEP

*From the Case Records of the Alpert Medical School
of Brown University Residency in Emergency Medicine*

DR. BENJAMIN BLACKWOOD: Today's patient is a 45-year-old woman who presents to our emergency department with acute onset of substernal chest pain. The patient was watching her son's soccer game when she abruptly stood up from a sitting position to cheer. She had immediate onset of a squeezing chest tightness and pressure that radiated to her left arm and was associated with nausea and mild shortness of breath. She described the pain initially as an 8/10. The discomfort receded over time, but never completely abated, and about 2 hours after onset she came to the emergency department because she felt that her discomfort, which was now a 3/10, was unusual and persistent.

The patient stated that she regularly exercised and jogged several miles 3-4 times per week. She had a history of hypertension, anxiety, and a total abdominal hysterectomy for uterine fibroids and adenomyosis. She was currently taking amlodipine and estrogen replacement therapy. She was a teacher, did not use tobacco or drugs, and was allergic to penicillin and sulfa medications. She did report multiple stressors in her life and stated that her mother had a myocardial infarction in her 60s.

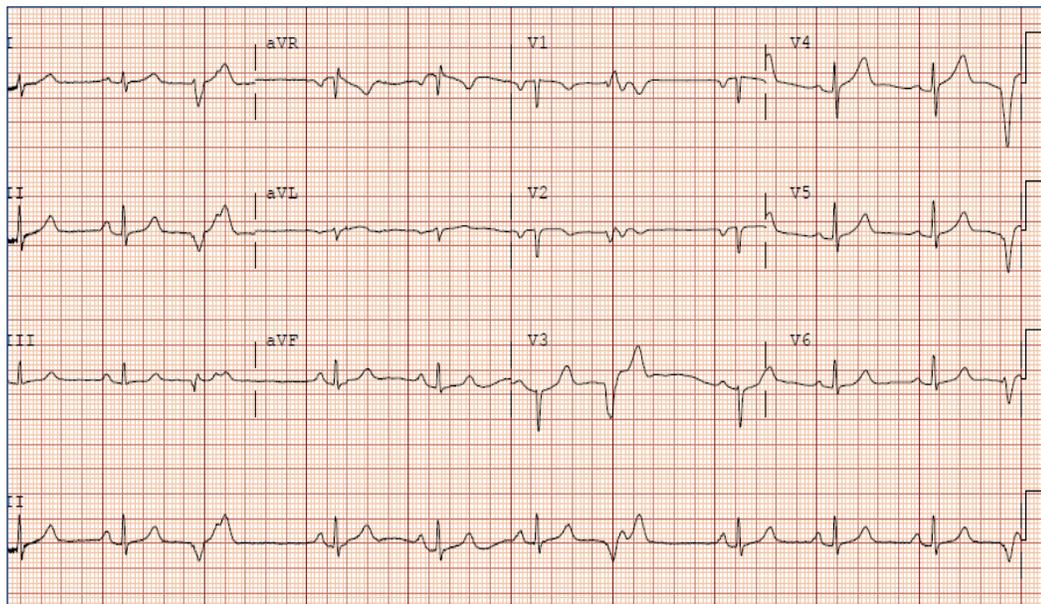
Upon arrival the patient had a blood pressure of 162/99,

pulse of 68, and otherwise had normal vital signs. She looked quite well but stated she still had 1/10 residual chest discomfort. Her physical exam was significant for clear lungs, a normal s1s2 with occasional irregular beats, and strong 2+ pulses bilaterally in the carotid, radial, femoral, and dorsal pedis locations. Her abdominal, musculoskeletal, and neurologic exams were unremarkable.

Due to the nature of her complaint an ECG was performed at triage.

DR. ANDREW NATHANSON: This is an abnormal ECG. What was your interpretation and what were your interventions?

DR. BLACKWOOD: The patient was in sinus rhythm with a rate at around 60. She had multiple ventricular premature complexes and she had an abnormal R wave progression. The patient's q wave in lead V2 was concerning for a completed ischemic event. Because of the patient's chief complaint and her abnormal ECG, an aspirin was ordered and she was placed on telemetry. A chest x-ray was unremarkable, the CBC revealed a mildly elevated white blood cell count of 12.4, she had normal electrolytes, and a d-dimer was within normal limits. Her troponin was elevated at 22.2 ng/ml (range .006-.060 ng/ml).



DR. LISA MERCK: The troponin is markedly elevated. While this is likely a myocardial infarction, what other diagnoses did you include in your differential?

DR. BLACKWOOD: The cardiac troponin, found in both the sarcomere and the cytosol of cardiomyocytes, is released into circulation as a byproduct of irreversible myocardial cell injury. Necrosis of the cardiomyocyte can be due to a number of processes. Our

patient had sudden onset of pain and consequently we were also concerned about an acute aortic dissection, which is noted to have a troponin elevation in almost 20% of cases. (1) Additionally, acute pulmonary embolism commonly results in an elevated troponin due to a combination of factors, including right ventricular strain. (2,3) While lower on our differential, we were unable to use the Pulmonary Embolism Rule-Out Criteria (PERC) rule due to her use of exogenous estrogen. Consequently, we obtained a CT, which demonstrated both a normal aorta, a normal appearing heart, and was negative for a significant segmental pulmonary embolism. (4,5) Other causes of an elevated troponin in a non-ischemic event include heart failure, cardiac inflammatory syndromes such as myocarditis, endocarditis, and pericarditis, as well as infectious and autoimmune processes, trauma, chemotherapy, and a number of other diseases and syndromes. (6) These causes were not considered relevant to our patient's presentation.

DR. LAWRENCE PROANO: Given this data, it appears that the patient was indeed having a myocardial infarction. What are the indications for going directly to the catheterization lab for this patient?

DR. BLACKWOOD: Patients who are having an ST elevation myocardial infarction (STEMI) should go to the catheterization lab emergently as outcomes are improved for these patients. Data suggests that patients with a non-ST elevation myocardial infarction (NSTEMI) can proceed to the catheterization lab urgently (24 hours and longer) unless they demonstrate an emergent need, defined as hemodynamic instability, a witnessed arrest, mechanical complications such as a valvular insufficiency, acute LV dysfunction and heart failure, sustained ventricular tachycardia, or dynamic ST-T wave changes. Additionally, patients with a rising troponin and stuttering chest pain and unstable angina need urgent catheterization and revascularization. (7, 8)

DR. THOMAS GERMANO: What was your initial treatment for this patient's acute MI?

DR. BLACKWOOD: The patient had already received an aspirin at the triage area of the emergency department. After her CT, she received clopidogrel and was begun on an intravenous heparin drip. A bedside echocardiogram was performed which demonstrated a reduced left ventricular ejection fraction (40%), as well as anterior, anteroseptal, apical and inferoseptal hypokinesis.

DR. ALISON MACGREGOR: This patient had only 1 cardiac risk factor—hypertension—and was on unopposed estrogen. It seems unusual that she would have an ischemic event. What do you think led to her myocardial infarction?

DR. OTTO LIEBMANN: While coronary artery disease due to atherosclerosis is the most frequent cause of a myocardial ischemic event, there are gender-based differences in both the presentation and cause of a myocardial infarction.

Plaque rupture is less common in women than in men, while plaque erosion is seen more frequently in women, and particularly in younger women. Non-atherosclerotic disease is increasingly recognized as a cause of myocardial ischemia. In patients with an acute coronary syndrome, up to 10% of patients undergoing coronary angiography are negative for obstructive epicardial coronary artery disease due to atherosclerosis or a "culprit" lesion. (9) A number of clinical entities causing non atherosclerotic MI exist including coronary arteritis from infectious and /or connective tissue disorders, coronary aneurysm, congenital abnormalities, substance abuse (cocaine), fibrous proliferation following transplantation and cardiac surgery, Takotsubo cardiomyopathy, as well as systemic metabolic disorders. (10) Additionally, spontaneous coronary artery dissection (SCAD), vasospastic angina, and coronary microvascular dysfunction are increasingly recognized as causes of ischemia in women without evidence of obstructive epicardial CAD. (9) Given the young age of this patient, the sudden onset of pain, and a lack of multiple CAD risk factors, we were concerned about Takotsubo's cardiomyopathy, SCAD, and, while less likely, vasospastic angina.

DR. JORDAN WOLFE: What transpired overnight and did the patient go to the catheterization lab?

DR. STEFI LEE: The patient was pain free when she arrived in the CCU. She had another episode of squeezing chest pain overnight without ECG changes and received nitroglycerin and lorazepam with good effect. Her troponin peaked at 72 ng/ml and by the following morning had dropped to 38 ng/ml. A left heart catheterization the following morning revealed a dissection of the left anterior descending (LAD) artery with a 99% mid LAD stenosis and TIMI 1 flow. Intravascular ultrasound revealed an intramural hematoma. The patient's other coronary arteries demonstrated only minor irregularities. The patient received 2 drug eluting stents (DES) with excellent results (TIMI 3 flow).

DR. ELIZABETH SUTTON: This patient had minimal cardiac risk factors. Do you think a case such as this can have an impact on risk-stratification tools used in the emergency department?

DR. LIEBMANN: This is an important consideration. In one recent retrospective study, 75% of patients with SCAD had one or fewer atherosclerotic disease risks factors. While our patient's ECG was abnormal, frequently ECGs in SCAD are non-specific and initial troponin levels are negative. Commonly used risk-stratification tools such as the HEART score or the Emergency Department Assessment of Chest Pain score could underestimate a young person's risk for disease. (11)

DR. WILLIAM BINDER: A spontaneous coronary artery dissection (SCAD) is an unusual cause of a myocardial infarction. What are the causes of coronary artery dissection and how often does it occur?

DR. BLACKWOOD: Spontaneous coronary artery dissection was first reported in 1931, and until the past decade was considered to be a rare diagnosis. New techniques used during coronary imaging, including intravascular ultrasound and optical coherence tomography, has led to an increased frequency of the diagnosis. (9) SCAD has a reported prevalence of 0.2% - 4.0% of all patients undergoing coronary angiography and has been reported in approximately 10% of women under 50 who present with ACS or AMI (with one Japanese study suggesting 20% of women under 50 had the disorder). (9,12,13,14) SCAD accounts for over 40% of cases of myocardial ischemia in pregnancy, and elevated estrogen and progesterone levels are believed to create both a hypercoagulable state as well as impair the integrity of vessel walls. (15, 16)

Triggers for SCAD include anything that can lead to increased shear stress and elevated blood pressure, including intense emotions, exercise, and the Valsalva response. It is felt that catecholamine surge may lead to increased shear stress as well as injury to the vascular intima. (9) Underlying causes include an association with fibromuscular dysplasia which is noted in up to 80% of patients with SCAD. (16)

DR. LAURA MCPEAKE: Today's patient had an LAD dissection. Is there a predilection for a particular coronary artery in SCAD?

DR. LIEBMAN: Spontaneous coronary artery dissection can occur in any of the epicardial arteries. Reports suggest that the LAD is most commonly involved, followed by the left circumflex and right coronary artery. (9) Multiple arterial lesions is unusual. Importantly, recurrence is not insignificant – up to 15% of patients have another spontaneous dissection within 2 years, and up to 25% will develop a recurrence within 5 years. (16)

DR. BRUCE BECKER: What was this patient's outcome?

DR. LEE: The patient did well and was discharged on hospital day 4 on dual antiplatelet therapy with aspirin and clopidogrel. Her ejection fraction was 45% and it was expected that it would improve. She was placed on atorvastatin and metoprolol, as well as amlodipine to ameliorate possible coronary artery spasm. She was seen 6 weeks post PCI and was doing well, with minimal atypical chest pain, and with plans to initiate cardiac rehabilitation. Further work-up to diagnose concomitant fibromuscular dysplasia using CT and MR angiography in her renal and cerebral vasculature was deferred for the time being, but will be considered at a later date.

References

1. Rapezzi C, Longhi S, Graziosi M, Biagini E, et al. Risk factors for diagnostic delay in acute aortic dissection. *Am J Cardiol.* 2008; 102: 1399-1406.
2. Kline JA, Hernandez-Nino J, Rose G, Norton HJ, Carmargo C. Surrogate markers for adverse outcomes in normotensive patients with pulmonary embolism. *Crit Care Med.* 2006; 34: 2773-2780.

3. Pruszczyk P, Bochowicz A, Torbicki A, Szulc M, et al. Cardiac troponin T monitoring identifies high-risk group of normotensive patients with acute pulmonary embolism. *Chest.* 2003; 123: 1947-1952.
4. Kline JA, Courtney DM, Kabrhel C, Moore CL, Smithline HA, Plewa MC, et al. Prospective multicenter evaluation of the pulmonary embolism rule-out criteria. *J Thromb Haemost JTH.* 2008; 6: 772-780. Doi:10.1111/j.1538-7836.2008.02944.x.
5. Freund Y, Rousseau A, Guyot-Rousseau F, Claessens YE et al. PERC rule to exclude the diagnosis of pulmonary embolism in emergency low-risk patients: study protocol for the PROPER randomized controlled study. *Trials.* 2015; 16: 537
6. Kelley, W. E., J. L. Januzzi, R. H. Christenson. "Increases of Cardiac Troponin in Conditions Other than Acute Coronary Syndrome and Heart Failure." *Clinical Chemistry* 55.12 (2009): 2098-2112.
7. Rab T, Kern KB, Tamis-Holland JE, Henry TD, et al. Cardiac Arrest: A Treatment Algorithm for Emergent Invasive Cardiac Procedures in the Resuscitated Comatose Patient. *J Am Coll Cardiol.* 2015; 66: 62-73.
8. Amsterdam EA, Wenger NK, Brindis RG, Casey DE Jr, et al. 201 AHA/ACC guideline for the management of patients with non-ST-elevation acute coronary syndromes: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation.* 2014; 130: 2354-12394.
9. Ahmed B, Creager MA. Alternative Causes of Myocardial Ischemia in Women: An update on Spontaneous Coronary Artery Dissection, Vasospastic Angina, and Coronary Microvascular Dysfunction. *Vascular Medicine.* 2017; 22: 146-160.
10. Bastante T, Rivero F, Cuesta J, Benedicto A, et al. Nonatherosclerotic Causes of Acute Coronary Syndrome: Recognition and Management. *Current Cardiology Reports.* 2014; 16: 543 doi:10.1007/s11886-014-0543-y
11. Lindor RA, Tweet MS, Goyal KA, Lohse CM, et al. Emergency Department Presentation of Patients with Spontaneous Coronary Artery Dissection. *Journal of Emergency Medicine.* 2017; 52: 286-291.
12. Mehta L, Beckie T, DeVon H, Grines CL, Krumholz HM, et al. Acute Myocardial Infarction in Women: A Scientific Statement from the American Heart Association. *Circulation.* 2016; 133: 916-947.
13. Vanzetto G, Berger-Coz E, Barone-Rochette G, Chavanon O, Bouvaist H, Hacini R, Blin D, Machecourt J. Prevalence, therapeutic management and medium-term prognosis of spontaneous coronary artery dissection: results from a database of 11,605 patients. *Eur J Cardiothorac Surg.* 2009;35:250-254 doi: 10.1016/j.ejcts.2008.10.023
14. Nakashima T, Noguchi T, Haruta S. Prognostic impact of spontaneous coronary artery dissection in young female patients with acute myocardial infarction: A report from the Angina Pectoris-Myocardial Infarction Multicenter Investigators in Japan. *Int J Cardiology.* 2016; 207: 341-348.
15. Sheikh A, Sullivan M. Pregnancy-related spontaneous coronary artery dissection: Two case reports and a comprehensive review of literature. *Heart Views.* 2012; 13: 53-65.
16. Saw J, Mancini GBJ, Humphries KH. Contemporary Review on Spontaneous Coronary Artery Dissection. *Cardiology.* 2016; 68: 297-312.

Authors

Benjamin Blackwood, MD, PGY3, Department of Emergency Medicine, Alpert Medical School of Brown University.

Steff Lee, MD, PGY-3, Department of Internal Medicine, Alpert Medical School of Brown University.

Otto Liebmann, MD, Assistant Professor of Emergency Medicine, Alpert Medical School of Brown University.

William Binder, MD, MA, FACEP, Associate Professor of Emergency Medicine, Alpert Medical School of Brown University .

Correspondence

william_binder@brown.edu