A 48-year-old woman with a history of hypertension and mild asthma has been transferred to the medical service because of an abnormal postoperative ECG. She had been admitted 2 weeks earlier to the gynecology-oncology service for local recurrence of a previously resected uterine sarcoma and underwent laparotomy for debulking of the pelvic mass and resection of the rectosigmoid colon. She did well until postoperative day 14, when sudden chest pain and dyspnea developed.

Blood pressure is now 150/88 mm Hg, heart sounds are normal, lungs are clear, and abdominal findings are normal. Serum potassium level is 3.8 mEq/L; magnesium, 1.7 mEq/L. An arterial blood gas measurement on room air reveals a pH of 7.4; partial pressure of oxygen, 57 mm Hg; partial pressure of carbon dioxide, 39 mm Hg; and serum bicarbonate, 25 mEq/L. White blood cell count is 19,400/L with a left shift.

Her postoperative (A) and preoperative (B) ECGs are shown here. 1. What abnormality is evident on the postoperative ECG? 2. Is the preoperative ECG normal? 3. Are the postoperative ECG findings related to the patient's recent chest pain and dyspnea? WHAT'S WRONG: 1. What abnormality is evident on the postoperative ECG?

The patient's ECG shows normal sinus rhythm with a prolonged corrected QT interval (QTc) of 625 milliseconds (normal QTc, less than 440 milliseconds) and inferior and anterolateral T-wave abnormalities. The QT interval, which varies inversely with the heart rate, is measured from the start of the QRS complex to the end of the T wave. It includes the total duration of ventricular activation and recovery. The QTc is defined as the QT interval divided by the square root of the R-R interval (measured in seconds); it is the best reflection of the ventricular repolarization time. 2. Is the preoperative ECG normal? No. The preoperative ECG also shows a prolonged QTc of 516 milliseconds and similar inferolateral T-wave abnormalities. 3. Are the postoperative ECG findings related to the patient's recent chest pain and dyspnea?

Although a long QT interval may indicate myocardial ischemia—which was a concern in this patient—it has many possible causes. The long QT syndrome (LQTS) is the phenotypic description of a heterogeneous group of disorders characterized by a prolonged QT interval. The syndrome can be inherited or acquired. Causes of acquired LQTS include medications (ie, class I or III antiarrhythmics and some antibiotics and psychotropic drugs), electrolyte abnormalities, and bradycardia. Because a prolonged QT interval was evident on all of our patient's ECGs, the etiology is most likely to be congenital and unrelated to her recent chest pain and dyspnea. Recognition of a prolonged QT interval is important, because this seemingly benign ECG finding places a patient at risk for torsades de pointes, or “twisting of the points,” a potentially fatal form of ventricular tachycardia. Once a prolonged QT interval is identified, correct all electrolyte abnormalities, especially hypokalemia, hypomagnesemia, and hypocalcemia; discontinue all potentially offending medications; and monitor the patient closely via telemetry. If all secondary causes have been ruled out, consider congenital LQTS. Congenital LQTS is caused by genetic mutations that encode for abnormal cardiac ion channels. In these situations, it is important to obtain a detailed family history, because LQTS puts patients at risk for sudden death. Our patient had no family history of cardiac disease or sudden death, which
suggests a sporadic or recessive mutation. In patients in whom congenital LQTS is suspected, genetic testing can confirm one of the 7 identified forms of this syndrome. **FURTHER WORKUP--AND A NEW TWIST**
The patient is transferred to the coronary care unit. A contrast CT angiogram of the chest shows no evidence of pulmonary embolism, but Doppler studies reveal a left lower extremity deep venous thrombosis. Low molecular weight heparin is started, and a Greenfield filter is placed in the inferior vena cava.
Serial enzyme determinations rule out myocardial infarction. An echocardiogram reveals mildly decreased left ventricular systolic function and mild to moderate mitral regurgitation. A stress test, performed 23 months before admission, had found only mild anteroseptal ischemia and normal left ventricular function. Thus, no specific cause of the patient's chest pain is found.
Her condition improves, and she is transferred back to the gynecologyoncology service. Two days after the transfer, the patient has a cardiac arrest, and the following rhythm is recorded.

1. **What is this rhythm?**
   The rhythm shown is torsades de pointes. This specific type of polymorphic ventricular tachycardia occurs in the setting of a prolonged QT interval. In patients with this arrhythmia, the QRS axis swings from a positive to a negative direction in a single lead.

2. **What should be done next to treat the patient?**
   Emergent treatment of torsades de pointes is critical, because the rhythm can quickly degenerate into ventricular fibrillation. Magnesium sulfate, 2 g, given as an intravenous bolus, followed by 1 to 3 g/h, is an effective intervention. Other therapies that accelerate the patient's heart rate shorten the QT interval and can terminate torsades de pointes. Isoproterenol, 2 to 8 g/min, can also be used. However, the safest and most effective treatment is placement of a temporary pacemaker, which "overdrive paces" the ventricle and shortens repolarization time.

Secondary causes of LQTS can induce torsades de pointes when the baseline QT interval is already prolonged. At the time of the arrhythmia, our patient's potassium and magnesium levels were low, which probably reduced the threshold for torsades de pointes. **OUTCOME AND FOLLOW-UP**
During her episode of torsades de pointes, the patient receives 2 shocks of 360 joules and 2 g of intravenous magnesium sulfate. Her ECG shows normal sinus rhythm (Figure); however, the pattern is T-wave alternans, which is characterized by changes in contour, amplitude, or polarity of the T wave that appear regularly--usually every other beat--without changes in the cycle length. Recently, T-wave alternans has been implicated as a predictor of susceptibility to malignant ventricular arrhythmias in patients with cardiomyopathy, previous myocardial infarction, or LQTS. The prognostic value of T-wave alternans has not been assessed in patients with congenital or acquired LQTS.

The patient is transferred to the coronary care unit. Her electrolytes are aggressively replenished, and she is monitored by telemetry for the next 48 hours with no further events. Subsequent ECGs continue to show intermittent T-wave alternans and a prolonged QT interval. An automatic cardioverter-defibrillator is implanted. Noninvasive testing indicates that the device is functioning well, and the patient is discharged with prescriptions for metoprolol, furosemide, and dalteparin. Routine follow-up with her gynecologist and cardiologist is scheduled.

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