

Ventricular Tachycardia, Myocardial Infarction, and Prolonged QT Interval: A Complication of Bronchoscopy

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This report details a unique case of ventricular tachycardia, myocardial infarction, and prolonged QT interval, all of which were precipitated during flexible fiberoptic bronchoscopy in a woman with no prior history of cardiac disease. Fiberoptic bronchoscopy was performed for the evaluation of an asymptomatic right upper lobe pulmonary nodule and bilateral upper lobe pulmonary infiltrates. These complications have not been previously reported with bronchoscopy in patients with a healthy heart. Flexible fiberoptic bronchoscopy is widely used in the management of patients with bronchopulmonary disease.^{1,2}

CASE PRESENTATION

A 41-year-old woman with no prior history of cardiac disease is admitted to the hospital for evaluation of an asymptomatic right upper lobe pulmonary nodule and bilateral upper lobe pulmonary infiltrates. The pulmonary nodule was initially detected on a chest radiograph, which was performed as a routine procedure before her surgery for removal of a fibroid tumor in her uterus. A follow-up computed tomography scan of the chest showed bilateral upper lobe infiltrates. The patient denies chest pain, shortness of breath, palpitation, syncope, seizure, cough, sputum production, night sweats, weight loss, or fever.

Patient History

The patient's medical history is insignificant for any prior cardiac or pulmonary diseases. She had a 20 pack-year history of cigarette smoking, but she quit smoking 2 years before presentation. The patient is a clerical worker. She has not traveled recently and has

no known history of exposure to tuberculosis nor to silica or rock dust of toxic material. The patient has a history of allergic reaction to intravenous pyelography dyes. She denies any family history of seizure, syncope, or sudden cardiac death. She denies drug addiction, and she was not taking any medications prior to her admission.

Physical Examination and Pertinent Laboratory Findings

Baseline physical examination shows an afebrile, well-nourished woman without any acute distress. Her blood pressure is 110/68 mm Hg, pulse is 72 bpm, and respiratory rate is 15 breaths/min. Cardiac monitoring during the physical examination shows normal sinus rhythm without any premature beats. Neck examination is unremarkable. The patient's lungs are clear with good bilateral air entry. Cardiac examination reveals no murmurs or extra sounds. The remainder of the physical examination is unremarkable.

The patient's serum potassium level is 4.1 mEq/L; magnesium, 2.2 mg/dL; and calcium, 9.2 mg/dL.

Calculation of the QT and QTc Intervals

The QT interval, reflecting the duration of depolarization and repolarization, is measured from the beginning of the QRS complex to the end of the T wave. The end of the T wave is defined by the return of the

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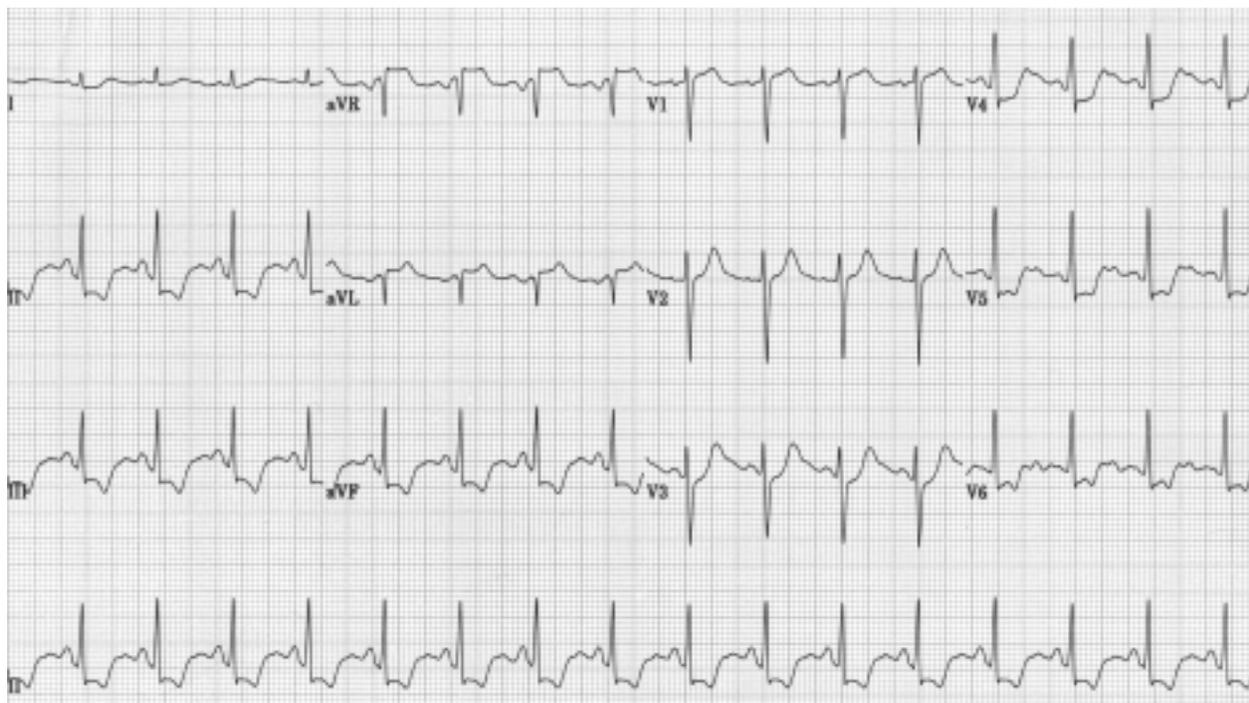


Figure 1. A 12-lead electrocardiogram performed immediately after bronchoscopy shows sinus tachycardia with a 1-mm ST segment elevation in lead aVL; a 3-to-4-mm segment depression in leads II, III, aVF, and V₄₋₆; and normal QT and QTc intervals.

end of the T wave to the isoelectric baseline (TP baseline). The QTc, which is the QT interval corrected for heart rate, is calculated by the most commonly used formula, suggested by Bazett:³

$$QTc = QT/\sqrt{(RR)}$$

Flexible Fiberoptic Bronchoscopy and Clinical Course

The patient undergoes flexible fiberoptic bronchoscopy using standard protocol. A total dose of 3 mg of midazolam is administered intravenously for sedation. The patient also receives an intramuscular injection of 50 mg of meperidine and 0.4 mg of atropine. Lidocaine (2% viscous) is used at the airway as topical anesthesia prior to bronchoscopy.

The procedure is completed, and, immediately after the bronchoscope is removed from the tracheobronchial tree, the patient has stridor associated with hypoxemia. Her oxygen saturation on pulse oxymetry drops to 68%. Her stridor is relieved by racemic epinephrine. Immediately thereafter, the patient has a short episode of supraventricular tachycardia at a rate of 160 bpm, which then converts into ventricular tachycardia at a rate of 180 bpm; her blood pressure is 180/100 mm Hg. The ventricular tachycardia lasts for approximately 3 minutes and is followed by hypoten-

sion (blood pressure, 78/40 mm Hg). Shortly thereafter, the patient's rhythm spontaneously converts to normal sinus, with ventricular bigeminy for approximately 4 to 5 minutes.

A 12-lead electrocardiogram (ECG) taken immediately after bronchoscopy shows sinus tachycardia with a rate of 100 bpm and 1-mm ST elevation in lead aVL, as well as marked ST depression of 3 to 4 mm in leads II, III, aVF, and V₄₋₆ (**Figure 1**). The QT and QTc intervals are 316 ms and 407 ms, respectively. The patient complains of the sensation of chest heaviness during as well as after the episode, which is partially relieved by two tablets of sublingual nitroglycerin (0.4 mg each).

The patient is started on aspirin, diltiazem, steroids, and diphenhydramine and is transferred to the coronary care unit. Based on her enzymatic criteria—a serial increase and then decrease of cardiac enzymes—the patient is diagnosed with myocardial infarction. Her electrolytes remain within normal limits. Serial ECGs do not demonstrate development of Q waves. The QT and QTc intervals show gradual prolongation to a maximum of 544 ms and 612 ms, respectively. Coronary angiography shows normal coronary arteries and normal left ventricular function.

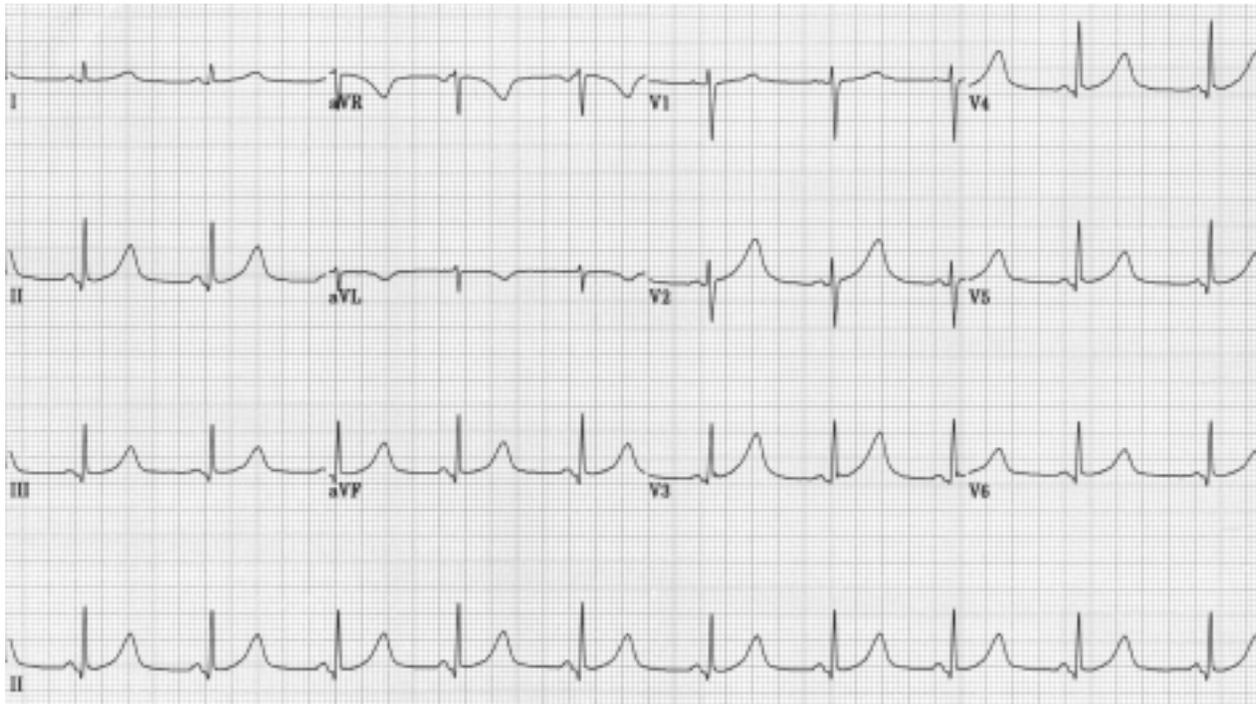


Figure 2. A 12-lead electrocardiogram performed 2 weeks following the bronchoscopy and adverse events shows normal sinus rhythm and prolonged QT and QTc intervals.

Follow-up at 2 Weeks

At follow-up 2 weeks after surgery, a 12-lead ECG is performed on the patient. The ECG shows persisting T-wave inversion in lead aVL and prolonged QT (542 ms) and QTc (545 ms) intervals (**Figure 2**). The subsequent course is uneventful.

DISCUSSION

Adverse Effects of Bronchoscopy

Several accounts of the risks and adverse effects of bronchoscopy of the airway pathways are available.^{1,2} This case report illustrates an unusual case of ventricular tachycardia, electrocardiographic and enzymatic evidence of myocardial infarction, and persisting prolonged QT interval following bronchoscopy in a relatively young patient without a prior history of cardiovascular disease.

Mechanism of Myocardial Injury

Several possible mechanisms could have caused the myocardial injury in this patient.

Acute myocardial ischemia. The cause of myocardial injury presumably was acute myocardial ischemia during a period of severe airway obstruction and stridor, possibly associated with an allergic reaction and/or mechanical manipulation.

Hypoxemia. Hypoxemia has been identified as a frequent complication in the setting of bronchoscopy.² The possibility of a repolarization abnormality as a consequence of myocardial insult from myocardial hypoxia during bronchoscopy has recently been emphasized in another case report.⁴ In that case,⁴ neither the ECGs nor the cardiac enzymes showed evidence of myocardial infarction, despite the development of unusually tall T waves in anterior leads during bronchoscopy.

Myocardial hypoperfusion. Another contributing factor to the development of myocardial injury for the patient in this case report may have been myocardial hypoperfusion during ventricular tachycardia, possibly induced by exogenous epinephrine inhalation as well as endogenous catecholamines released during the procedure.

Coronary artery spasm. An additional factor contributing to the clinical presentation of this dramatic cardiac event may have been a coronary artery spasm, which is an uncommon response induced by bronchoscopy. Histamine released during an allergic reaction has been reported to provoke coronary artery spasm in swine models of localized atherosclerosis^{5,6} as well as in a human patient.⁷ Yasue et al⁸ have postulated that the increased parasympathetic tone may paradoxically

activate the α -adrenergic receptors present in the large coronary arteries, causing vasoconstriction and decreasing coronary blood flow. Yasue et al^{8,9} and other researchers¹⁰ have also pharmacologically induced coronary artery spasm by administration of parasympathetic drugs and have relieved these spasms with atropine. The patient in this case report received atropine and opiate alkaloids before the procedure. Of note, however, is the fact that atropine administration itself has actually induced coronary artery spasm.¹¹ Endogenous histamine is shown to be released by a wide variety of pharmacologic agents, including opiate alkaloids.¹²

Causes of Prolonged QT Interval

In this patient, the QT and QTc intervals were 316 ms and 407 ms, respectively, on the initial ECG performed soon after the bronchoscopy. When a patient's heart rate is between 45 and 115 bpm, the normal limits of QT may vary between 460 and 300 ms;¹³ many clinical studies that involve the QT interval do not consider the interval to be abnormally long unless the QTc is greater than 440 ms.¹⁴ Subsequently, the QT and QTc intervals showed gradual prolongation to a maximum of 544 ms and 612 ms, respectively, in this patient.

The most common causes of acquired long QT syndromes are class I antiarrhythmic drugs (eg, quinidine, procainamide, disopyramide) and electrolyte disorders (eg, hypokalemia, hypomagnesemia, and hypocalcemia).¹⁵ This patient did not have any electrolyte abnormalities that could predispose her to the QT prolongation. The ventricular tachycardia that developed following bronchoscopy resolved spontaneously, and the patient did not receive any antiarrhythmic medications that could explain prolongation of QT interval. Extreme bradycardia is known to lead to QT prolongation; however, the patient's heart rate never dropped below 60 bpm, despite the fact that she received diltiazem, a calcium antagonist. Bepridil, which blocks the outward delayed rectifier current (I_k), is the only known calcium antagonist that leads to an acquired prolonged QT interval.¹⁶ The antihistamine administered to the patient for the first 24 hours (for treatment of possible allergic reaction) is very unlikely to have prolonged her QT interval.

The QT prolongation in this patient was apparently associated with myocardial ischemia and infarction. The QT interval gradually became prolonged and subsequently decreased as the acute process of myocardial infarction and its healing occurred. The ventricular tachycardia was not polymorphic but monomorphic in nature, which is unrelated to QT prolongation. The QT interval and QT dispersion (ie, the difference

between the minimal and maximal QT interval) is known to be prolonged in patients with myocardial infarction¹⁷⁻¹⁹ as well as in subjects with coronary artery spasm, such as in variant angina.¹⁹

Morbidity and Mortality with Bronchoscopy

The morbidity and mortality associated with bronchoscopy is very low.^{1,2,20} Bradycardias, ventricular premature complexes, supraventricular tachycardias, ventricular tachycardias, cardiac arrest, and deaths have all been described.^{2,20,21} The arrhythmias and tachycardia observed during bronchoscopy have been associated with general anesthesia, tetracaine administration, atropine injection before bronchoscopy, hypoxia, and unsuspected acute myocardial infarction.²⁰⁻²⁴ Transient ST segment depression of greater than 1 mm and bundle branch block have been described in elderly patients undergoing fiberoptic bronchoscopy.²⁵ To the knowledge of these authors, no other reports have been published of ventricular tachycardia with myocardial infarction and prolonged QT interval in a patient with a previously healthy heart as a complication related to bronchoscopy.

SUMMARY

This unique case presents the potential risk of ventricular tachycardia with myocardial infarction as a complication associated with bronchoscopy, even in patients without cardiac disease. Physicians should be aware of this rare, but potentially life-threatening, complication of bronchoscopy. ECG monitoring during bronchoscopy is mandatory (even in relatively young patients without prior history of cardiovascular disease) for the early detection and appropriate management of hemodynamically unstable cardiac arrhythmias. A 12-lead ECG is recommended following fiberoptic bronchoscopy. **HP**

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