Case Report

Complete Heart Block in a Patient with Acute Cholangitis Caused by the Cardiobiliary Reflex

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Highlights

- Cardiobiliary reflex can cause bradyarrhythmia in biliary disease.
- Complete heart block may occur with acute cholangitis after cholecystectomy.
- The cardiobiliary reflex should be considered after exclusion of other causes of bradyarrhythmia.

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ABSTRACT

Background: Gallbladder and biliary tract diseases may manifest as cardiac symptoms, ischemic electrocardiographic changes, and arrhythmias. Albeit rare, cases of bradyarrhythmia caused by the cardiobiliary reflex have been reported in the context of acute cholecystitis. The case discussed herein emphasizes the cardiobiliary reflex as a possible cause of bradyarrhythmia in patients with gallbladder and biliary tract diseases.

Keywords: Arrhythmia; Cope; Electrocardiogram; Cardiobiliary; Bradycardia

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Introduction

Complete heart block (CHB) is a life-threatening clinical condition characterized by atrioventricular node dysfunction, often because of age-related degeneration, coronary artery disease, or cardiac surgery, resulting in an inability to conduct electrical impulses from the atria to the ventricles. Gallbladder and cholangial diseases can mislead clinicians through their symptoms, ischemia-like electrocardiographic (ECG) changes, and arrhythmias. In 1971, O'Reilly and Krauthamer[1] first reported that patients with cholecystitis may have sinus bradycardia via the cardiobiliary reflex. A literature search identified few reported cases of bradycardia in patients with cholecystitis but no reported cases of CHB associated with cholangitis and biliary tree involvement. Nonetheless, considering the frequent occurrence of bradycardia and arrhythmias in daily clinical practice and the prevalence of gallbladder and cholangial diseases in the wider community, this case is presented to avoid overlooking the relationship between these 2 disease processes.

Case Presentation

A 67-year-old woman who resided in a nursing home presented to the emergency department with nausea and fever for 2 days. She had not experienced syncope or lightheadedness, nor did she have risk factors such as diabetes, hypertension, or smoking. She was taking fesoterodine (8 mg once daily) for neurogenic bladder. She had undergone partial gastrectomy 7 months earlier and cholecystectomy 5 months earlier. On presentation, her blood pressure was 150/70 mm Hg, and her heart rate was 40 beats/min. Her oxygen saturation was normal, and her body temperature was 38.0 °C. Initial blood tests revealed an elevated white blood cell count of 18 000/cm^3, alkaline phosphatase level of 682 U/L (upper limit of normal [ULN], 105 U/L), gamma-glutamyl transferase level of 166 U/L (ULN, 42 U/L), direct bilirubin level of 1.13 mg/dL (ULN, 0.3 mg/dL), total bilirubin level of 1.88 mg/dL (ULN, 1 mg/dL), and C-reactive protein level of 23 mg/L (ULN, 5 mg/L). Potassium level was 4.4 mmol/L, and thyroid test results were normal. Troponin test results were negative. ECG at presentation revealed CHB (Figure 1), and echocardiography showed no additional pathologic findings.

The patient was admitted to the coronary care unit (CCU) with a diagnosis of CHB. Gastroenterology was consulted, and intravenous ceftriaxone and hydration were initiated. On the third day of her stay in the CCU, a ventricular tachycardia storm occurred. A temporary transvenous pacemaker wire was inserted. The patient was then transferred to the catheterization laboratory, where conventional selective coronary angiography was performed to assess for myocardial ischemia as a possible cause. The angiogram revealed no significant lesions. Gastroenterology was consulted again, and magnetic resonance cholangiopancreatography (MRCP) was recommended. MRCP revealed dilated intrahepatic and extrahepatic biliary ducts (Video 1; Figure 2).

Simultaneously, *Klebsiella pneumoniae* was grown in blood cultures. Because of increased cholestatic enzyme levels and worsening right upper quadrant pain and jaundice, percutaneous cholangiostomy was performed. Cholestatic enzyme levels and symptoms gradually regressed. On account of the prolonged duration of the cholangitis episode, the patient remained dependent on the pacemaker. A decision was made to implant a permanent pacemaker and remove the temporary transvenous pacemaker wire. Subsequently, the patient was referred to a hepatobiliary surgical center to undergo revision of the prior cholecystectomy.

The occurrence of ventricular tachycardia storm coincident with the diagnosis of CHB suggested that the arrhythmia was of recent onset. This also coincided with the diagnosis of cholangitis. Because no other cause of CHB was identified, the heart block was attributed to the cardiobiliary reflex.

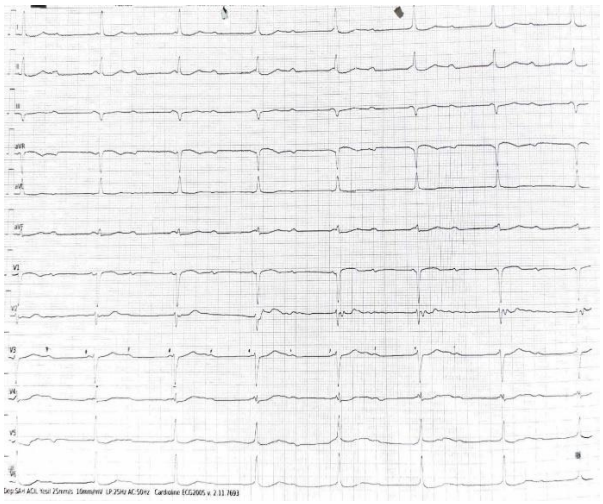


Figure 1. Electrocardiogram at presentation demonstrating complete heart block

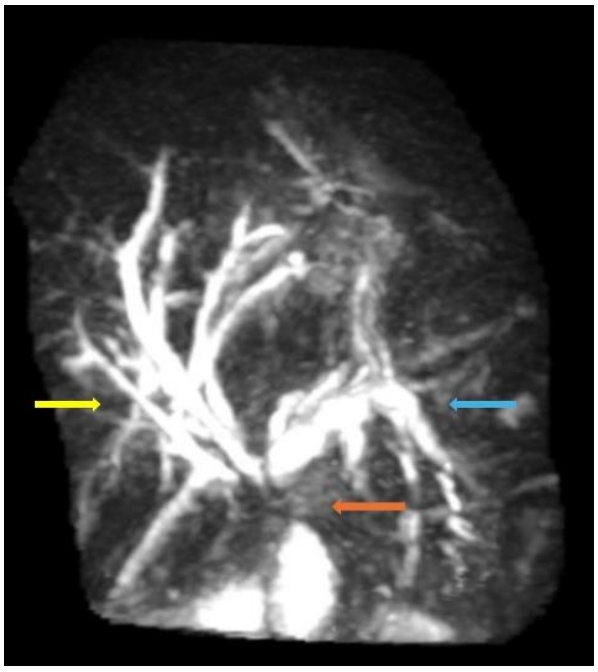


Figure 2. Magnetic resonance cholangiopancreatography image demonstrating biliary duct dilatation associated with cholangitis.
Yellow arrow indicates the dilated right intrahepatic biliary duct; blue arrow, the dilated left intrahepatic biliary duct; and orange arrow, the stenotic segment.

Discussion

Acute cholangitis, an infection of the biliary tract, is typically caused by biliary duct obstruction. Choledocholithiasis and neoplasia are the 2 primary causes. Radiologic imaging is drawn upon when acute cholangitis is clinically suspected, in addition to clinical diagnosis based on Charcot triad of pain, fever, and jaundice. Blood cultures are positive in 40% of cases, most commonly revealing Escherichia coli and Klebsiella species.[2] Endoscopic or radiographic biliary drainage is an essential component of care in addition to antimicrobial therapy. Even with improved treatment, the mortality rate for acute cholangitis is approximately 5%.[3] Biliary tract diseases can mimic cardiac symptoms, including chest pain and bradycardia. In certain cases, they can manifest with electrocardiographic changes similar to those typical of acute coronary syndromes, such as ST-segment elevation, right bundle-branch block, and T-wave inversion.[4]

Our case involved unexplained bradycardia accompanying acute cholangitis. Bradycardia in acute cholangitis is thought to be due to the vagally mediated cardiobiliary reflex. The vagus nerve and spinal T4 to T6 neurons innervate the heart and biliary tract, respectively. Morrison and Swalm[5] showed that intermediary neurons that connect the 2 groups of spinal neurons can mediate such a cardiobiliary reflex. According to O'Reilly and Krauthamer,[1] patients with cholecystitis may have a cardiobiliary reflex, also known as the Cope sign, which can cause sinus bradycardia.

A review of the literature shows very few cases, and all reported cases are associated with the cardiobiliary reflex occurring with gallbladder inflammation rather than biliary tract inflammation. Iftikhar et al[6] found 9 previous cases in their review of the literature and 1 new case, which they presented in their publication: all were associated with inflammation of the gallbladder and not the biliary tract.

Our case stands out because it associates the cardiobiliary reflex with the biliary tract. The cardiobiliary reflex is often a diagnosis of exclusion. Electrolyte abnormalities (eg, hyperkalemia), endocrine abnormalities (eg, hypothyroidism and hyperthyroidism), ischemic heart disease, and use of rate-limiting drugs are important considerations in the differential diagnosis. In the absence of an underlying cause of bradycardia, combined with the presence of acute biliary disease, an association between the 2 clinical entities should be considered. What makes our case notable is CHB that we believe was related to the cardiobiliary reflex in a patient who presented with acute cholangitis and had previously undergone cholecystectomy. To our knowledge, we did not identify a similar case in the literature.

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Declarations:

Ethical Approval

Ethical approval was not required for conducting this research.

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Conflict of Interest

The authors report no conflict of interest.

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