Severe Bradycardia Following Electrical Cardioversion for Atrial Tachyarrhythmias in Patients with Acute Myocardial Infarction*

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Bradycardia following electrical cardioversion is an uncommon complication. The present report describes three patients who developed life-threatening bradycardia following electrical cardioversion for atrial tachyarrhythmias in the setting of an acute myocardial infarction. All three patients had multivessel coronary artery disease with a totally occluded right coronary artery and a possibility of ischemic sinus node dysfunction. When electrical cardioversion is undertaken for new onset of atrial tachyarrhythmia in the setting of an acute myocardial infarction, measures for immediate, temporary pacing should be easily available. (Chest 1990; 97:241-42)

Symptomatic bradycardia is an uncommon complication following electrical cardioversion for an atrial tachyarrhythmia. In patients with acute myocardial infarction, atrial tachyarrhythmia associated with hemodynamic compromise or worsening of ischemia requires electrical cardioversion. We describe three patients who developed severe bradycardia following electrical cardioversion for an atrial tachyarrhythmia in the setting of an acute myocardial infarction.

CASE REPORTS

CASE 1

A 74-year-old man was admitted to the hospital with unstable rest angina. Forty-eight hours after admission he developed severe prolonged chest pain of 3 hours' duration. His electrocardiogram revealed sinus rhythm and changes of anterior and inferior myocardial infarctions of indeterminate age and a possible acute lateral wall infarction. Creatinine kinase (CK) peak was 2,002 IU/L, with a peak CK MB of 15.2 percent. He exhibited recurrent rest angina and subsequently developed hypotension requiring insertion of an intra-aortic balloon pump. His chest pain resolved following insertion of the balloon pump, but he required vasopressors to maintain an adequate blood pressure, and later he developed atrial flutter with a ventricular response of 150/min and was hypotensive in spite of pressor infusions. In view of his hemodynamic instability, urgent electrical cardioversion was performed. An initial countershock of 50 joules was unsuccessful, and a second 50-Joule countershock was repeated. This resulted in severe sinus bradycardia (less than 30 beats/min) and worsening of hypotension. Cardiopulmonary resuscitative efforts (including IV atropine and IV epinephrine) failed and the patient died.

CASE 2

A 61-year-old man was admitted to a hospital with new onset of rest angina and a normal ECG. Subsequently, the patient developed severe retrosternal chest pain lasting several hours. His ECG at this time showed 1 mm ST segment elevation with T wave inversions in leads 2, 3, and aVF and 2 mm ST segment depression in leads V1 through V4, associated with new onset of atrial fibrillation with rapid ventricular response. Chest pain was relieved with IV nitroglycerin, and the rapid ventricular response was controlled with two IV boluses of 0.25 mg digoxin. Subsequently, he developed episodes of slow ventricular response with pauses up to 8.4 s. At this time, digoxin therapy was discontinued, a temporary pacemaker electrode was inserted, and demand ventricular pacing was initiated. Over the next two days, his serum CK peaked at 3,130 u/L, with a CK MB of 9 percent. He continued to display atrial fibrillation with paroxysms of rapid ventricular response and systemic hypotension. In view of the precarious hemodynamic state, electrical cardioversion for atrial fibrillation was undertaken. Following electrical countershock, he developed complete asystole, and demand ventricular pacing was initiated via the temporary pacemaker electrode. Over the next 12 h, the patient remained in a paced rhythm with no spontaneous atrial activity. Subsequently, he demonstrated a junctional escape rhythm with a rate of about 30 beats/min. Thirty-six h later, he once again developed atrial fibrillation, at which time ventricular rate was controlled with IV digoxin. He underwent coronary artery bypass surgery for significant left main coronary artery stenosis and total right coronary artery occlusion. Over a one-year follow-up period he is in sinus rhythm, with no clinical manifestations of sinus node dysfunction.

CASE 3

A 64-year-old woman with previous myocardial infarctions and subsequent coronary artery bypass graft surgery was admitted with unstable rest angina. She had no previous history of a sick sinus node, and her ECG on admission showed sinus rhythm. The patient experienced a non-Q wave myocardial infarction, with CK peak of 520 IU/L and a positive CK MB band. One day later, she developed atrial flutter with variable atrioventricular block. A total of 1.25 mg of digoxin was given IV, and quinidine gluconate (324 mg orally three times a day) therapy was also initiated. After eight doses of quinidine were given, atrial flutter persisted, and therefore, it was decided to perform electrical cardioversion. At this stage, there was no clinical or laboratory evidence of digitalis toxicity or renal failure, and the last dose of digoxin was given more than 14 h prior to cardioversion. Following electrical countershock of 20 joules, asystole developed, which persisted despite 2 mg of IV atropine. Thoracotomic pacing was utilized while a temporary transvenous pacemaker electrode was inserted. Atrial activity did not recur for two days. During this period, the pacemaker was turned off, there was a slow junctional escape rhythm. Coronary angiography was performed, which revealed a severe triple-vessel disease, and the patient later died during coronary artery bypass graft surgery.

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Discussion

Acute myocardial infarction with multivessel coronary artery disease was the common feature in our three patients. During the course of their myocardial infarctions all three developed atrial tachyarrhythmias that required electrical cardioversion. This was followed by severe, prolonged bradycardia that required ventricular pacing. There was no clinical evidence of intrinsic sinus node dysfunction prior to their hospitalization, and one of the patients (case 2) has not demonstrated any evidence of sinus node dysfunction over a one-year follow-up period.

Bradycarrhythmias following electrical cardioversion for atrial tachyarrhythmias are uncommon. Waldecker et al reported their results of 112 episodes of direct current countershocks for tachyarrhythmias in 75 patients. In their series significant bradycardia following countershock for supraventricular tachycardia occurred only in one patient (sinus bradycardia—50 beats/min), who in fact was clinically suspected to have a sick sinus syndrome. Lemberg et al reported 101 episodes of direct current electrical countershock in patients with atrial fibrillation and did not encounter a single instance of significant bradycardia after electric countershock. Bradycarrhythmias following ventricular defibrillation have been described, but these are related to cholinergic stimulation, are transient, and respond to IV atropine. None of these electrical cardioversions was performed in the setting of an acute myocardial infarction. The present report documents the occurrence of serious bradycardia following electrical cardioversion for atrial tachyarrhythmia in the setting of an acute infarction. While the pathogenesis of the bradycardia in our patients is unclear, it is not likely to be related to cholinergic stimulation alone, because IV atropine was ineffective, and in patients 2 and 3 the atrial activity did not recur for a long time. Although there was no clinical or laboratory evidence of drug toxicity, it is possible that the bradycardia may have been potentiated by digoxin and/or quinidine. The most likely explanation is ischemic dysfunction of the sinus node during the course of the acute myocardial infarction.

This report describes a potentially life-threatening complication that should be recognized when performing electrical cardioversion for new-onset atrial tachyarrhythmia in the setting of an acute myocardial infarction. In such a situation, measures for immediate, temporary pacing should be readily available.

References


Bilateral Paramediastinal Post-traumatic Lung Cysts*

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Unilateral post-traumatic and paramediastinal lung cysts are uncommon. Conservative therapeutic measures are usually sufficient. This report describes a patient whose bilateral post-traumatic paramediastinal lung cysts, a previously undescribed entity, were presumed to be bilateral hemidiaphragmatic hernias and, consequently, led to unnecessary surgery.

(CHEST 1990; 97:242-44)

Unilateral post-traumatic and paramediastinal lung cysts are uncommon and usually do not require treatment. Nevertheless, recognition of a traumatic lung cyst is important, since misdiagnosis may lead to unnecessary surgery.1 This report describes a patient whose bilateral post-traumatic paramediastinal lung cysts were mistaken for hemidiaphragmatic hernias. This unusual complication of blunt chest trauma has not, to our knowledge, been previously described.

Case Report

The patient was a 23-year-old white man with a history of intravenous drug abuse, bisexual behavior, HIV antibody-positive status, a perirectal abscess, hepatitis B, and recurrent lower-extremity staphylococcal infections, who presented to University Medical Center after a motorcycle accident. An emergency-room, portable chest roentgenogram was initially interpreted as revealing no gross abnormality (Fig 1). The patient was treated for chest lacerations and discharged the next day. Four days later, he was readmitted with cough, hemoptysis, pleuritic chest pain, and low-grade fever. The patient denied dyspnea or production of purulent sputum. Physical examination revealed normal vital signs, healing chin lacerations, and mild abrasions over the anterior chest. Coarse crackles were heard scattered throughout both lung fields, and there was no cyanosis. There was no abnormal tenderness, and the bowel sounds were normal. No arterial blood gas levels were obtained. The white blood cell count was 6,800/mℓ with no left-ward shift, and the hemoglobin level was 12.2 g/dl. The chest roentgenogram revealed prominent bilateral air-fluid levels at the medial aspects of both hemithoraces, initially thought to be consistent with a ruptured diaphragm and entry of bowel into each pleural cavity (Fig 2). An exploratory laparotomy revealed both hemidiaphragms to be intact, with no protrusion of bowel into the chest. A CT scan of the chest after surgery demonstrated prominent bilateral air-fluid interfaces located in large, elliptical, thick-walled paravertebral cystic spaces (Fig 3). Upon further question, the patient denied pulmonary or systemic complaints prior to his motorcycle accident. Review of the chest roentgenogram from the admission four days earlier revealed linear band-like densities extending caudally from both hila, consistent with bilateral traumatic pneumoceles or abnormal collections of air entrapped by the inferior pulmonary ligaments (Fig 1). A follow-up chest roentgenogram two weeks later showed near total resolution of both lung cysts.

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Bilateral Paramediastinal Post-traumatic Lung Cysts (Ulstad, Bjelland, Quan)