

Sudden atrial fibrillation associated with acute alcohol ingestion and cor triatriatum

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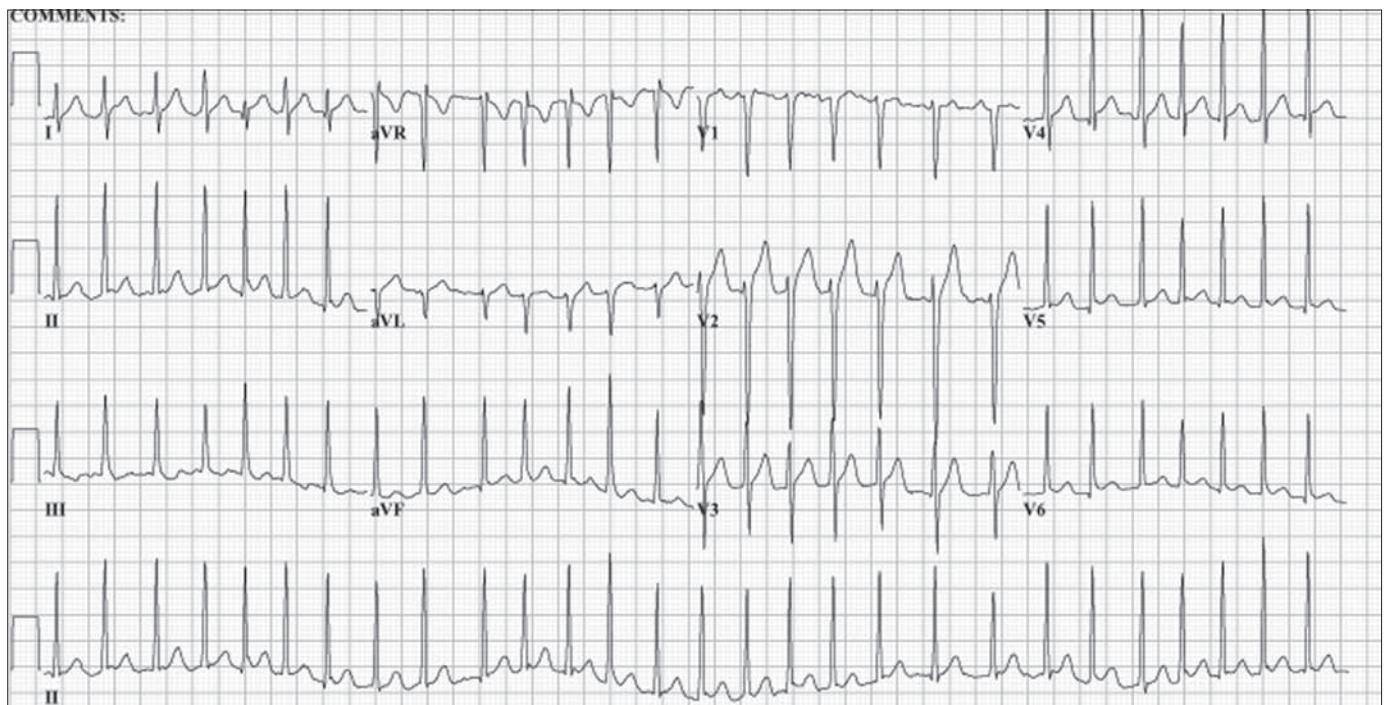


Figure 1. Electrocardiogram demonstrating atrial fibrillation with rapid ventricular response (heart rate, ~150 beats per minute) in a 21-year-old patient with recent binge drinking.

We present a case of alcohol-induced atrial fibrillation in a patient with a rare congenital malformation that is known to be associated with atrial arrhythmias.

CASE REPORT

A 21-year-old African American man with no significant past medical history presented to the emergency department with a chief complaint of increased heart rate and dyspnea. The patient reported that when he awoke on the morning of presentation, “his heart was racing.” Over the next 3 hours his symptoms did not improve, and he started to become dyspneic. He had no chest pain, syncope, or dizziness. The patient had never had symptoms like this previously.

The patient had consumed excessive amounts of alcohol the evening prior but denied any illicit drug use recently or in the past. When evaluated in the emergency department, he was afebrile, with a blood pressure of 122/86 mm Hg, oxygen

saturation of 100% on a nasal cannula of 2 L/min, and a respiration rate of 16 breaths per minute. The patient’s peripheral pulses were irregular in rhythm and rate. His lungs were clear to auscultation, and he had an irregularly irregular heart rhythm without precordial rubs or murmurs. His electrocardiogram (*Figure 1*) showed a heart rate of 169 beats per minute and atrial fibrillation with rapid ventricular response. Chest radiograph showed no acute cardiopulmonary process.

His complete blood count, complete metabolic panel, and urine toxicology screen were all within normal limits. The patient received 20 mg of diltiazem intravenously, which achieved a mild heart rate reduction; he was continued on oral diltiazem and hydration with normal saline. His heart rate began to nor-

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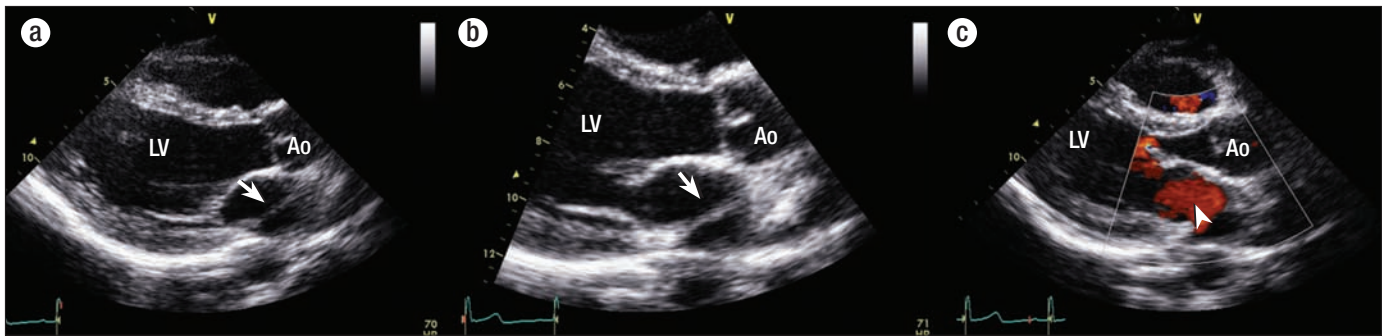


Figure 2. Echocardiographic images of the patient. (a, b) A dense band of tissue (arrow) is seen in the left atrium. (c) Flow across the left atrium is redirected across fenestrations through the band of tissue before it can cross the mitral valve (arrowhead).

malize, and he spontaneously returned to normal sinus rhythm. Follow-up electrocardiogram 10 hours later demonstrated a heart rate of 77 beats per minute and normal sinus rhythm. Transthoracic echocardiogram revealed a left ventricular ejection fraction of 45% and an “incidental” finding of cor triatriatum of the left atrium (Figure 2). Right ventricular systolic pressure was estimated at 24 mm Hg (normal), and no other significant abnormalities were noted.

DISCUSSION

“Holiday heart syndrome” was first described by Ettinger in 1978. He found that during the holiday season, December 24 to January 1, and every Saturday to Tuesday, there was a higher incidence of atrial arrhythmias associated with excessive alcohol consumption (1). The most common supraventricular arrhythmia noted was atrial fibrillation.

There has been much controversy over the exact mechanism by which alcohol induces atrial fibrillation. Studies using epidemiologic data and studies of the electrophysiologic effects of alcohol on humans, atrial cells, and animal models have resulted in conflicting results. Engel and Luck postulated that alcohol-induced atrial arrhythmias were related to intramyocardial catecholamine release in response to the toxic effects of acetaldehyde (2). Other studies have suggested that an increase in sympathetic reaction could be related to the production of atrial fibrillation based on the increased density of beta-adrenergic receptors in lymphocytes (3, 4).

In a recent review, Balbão and associates proposed multiple mechanisms for the acute and long-term consumption of alcohol resulting in atrial fibrillation (5). They thought that alcohol consumption acutely affected catecholamine release, metabolic acidosis, electrolyte disturbances, and increased oxidative distress. In the long term, this resulted in myocardial fibrosis/dilatation, structural heart disease, metabolic disturbances, and increased sympathetic tone. The combination of these effects contributed to the increase in atrial arrhythmias.

Cor triatriatum is a rare congenital malformation where the atrium is divided into two chambers. This congenital anomaly

was first described by Church in 1868 (6). The malformation can occur in the right or left atrium, described in Latin as *cor triatriatum dexter* or *sinister*, respectively. In our patient, the malformation occurred on the left side of the heart. In *cor triatriatum sinister*, the common pulmonary venous chamber is divided from the true atrium by a fibromuscular band or membrane (7, 8).

To our knowledge, this is the only reported case of alcohol-induced atrial fibrillation in a patient with the finding of *cor triatriatum*. It is unknown whether there is an association between the two disorders. Given that both alcohol consumption and *cor triatriatum* are associated with atrial fibrillation, it is unclear if the presence of the congenital abnormality put the patient at a higher risk of arrhythmias. He has been counseled to reduce his alcohol intake. At 6-month follow up, the patient has had no further instances of palpitations, and he will continue to be monitored for further arrhythmias or symptoms of obstruction.

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