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Case Report Open Access

A Case of 2:1 Atrio -Ventricular Block in Digoxin Toxicity

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Introduction

Digoxin is mostly used in patients with systolic heart failure for enhancing cardiac contractility and in patients with supraventricular tachyarrhythmias for ventricular rate control [1]. With the advent of new therapies for heart failure, overall use of digoxin has declined but the number of patients requiring hospitalization with digoxin toxicity has remained stable [2]. Here we report a case of 2:1 AV block caused by digoxin and review other cardiac manifestations of digoxin toxicity.

Case Report

An 87 year old male with history of congestive heart failure on digoxin was sent to the emergency room (ER) after he was found to be bradycardic by a visiting nurse. The patient did not have any complaints of nausea, vomiting, chest pain, palpitations or dizziness. The patient was not taking a beta blocker, calcium channel blocker or any antiarrhythmic medication.

On exam, patient had a heart rate of 28, respiratory rate of 14 and BP of 120/84. The lungs were clear to auscultation. Cardiac exam revealed bradycardia with normal heart sounds. The lab work was significant for serum potassium of 5.5 meq/l, serum creatinine of 3.1 mg/dl (baseline 0.9 mg/dl) and a serum digoxin level of 3.80 ng/ml (0.8 - 2.0 ng/ml). The EKG showed a 2:1 AV block with heart rate of 28 and a left bundle branch block (Figure 1).

The patient was treated with digoxin-specific antibody (Fab) fragments in the ER and admitted to Cardiac Care Unit (CCU) for observation. The patient remained asymptomatic and hemodynamically stable during the entire course of his admission. The 2:1 block resolved after the treatment and serum potassium also normalized (Figure 2).

Discussion

Digoxin has a narrow therapeutic index and life threatening arrhythmias can occur if plasma levels rise above 2 ng/ml. Predisposing factors for digoxin toxicity include renal insufficiency, interactions with other drugs that affect digoxin metabolism and electrolyte abnormalities (particularly hypokalemia) [3].

Digoxin can potentially cause almost any type of arrhythmia. There can be tachyarrhythmias associated with delayed afterdepolarizations or bradyarrhythmias due to increase in the vagal tone [4].

The classic "digitalis effect", a characteristic down-sloping ST depression in the lateral leads (reversed tick sign) with QT interval shortening, is often seen with chronic digoxin use but it doesn't correlate well with the toxicity.

Premature ventricular contractions (PVC's) are often the first sign of digoxin toxicity. There can be isolated PVCs, ventricular bigeminy or bidirectional ventricular tachycardia, a distinctive tachyarrhythmia associated with digoxin intoxication. AV junctional rhythms, including the accelerated junctional rhythm may also represent digoxin toxicity. If not recognized and treated early, these arrhythmias can progress to ventricular tachycardia or ventricular fibrillation.In bradyarrhythmias, sinus bradycardia may be one of the earliest signs of digoxin toxicity. AV blocks including the first degree block with widening of PR interval, second degree block (Mobitz type I being more common) and third degree block can also occur with digoxin toxicity.

Non-paroxysmal atrial tachycardia with AV block (usually 2:1) is a result of both increased ectopy and enhanced vagal activity and is also a characteristic arrhythmia of digoxin toxicity.

Rapidly conducted atrial arrhythmias like atrial flutter and atrial fibrillation are generally not induced by digoxin. Similarly, Mobitz type II AV block is also a rare phenomenon in digoxin toxicity.

The left bundle branch block in our patient was due to the underlying conduction defect which persisted even after the treatment but the 2:1 AV block was most likely the result of digoxin toxicity, although the possibility of hyperkalemia affecting the conduction system cannot be ruled out. We conclude that even though the use of digoxin has declined significantly, we continue to see patients with digoxin toxicity. In our opinion, digoxin should only be considered when the heart failure drug therapy (beta-blockers and ACE inhibitors/ARBs) has been maximized and also patients on digoxin should be monitored carefully.

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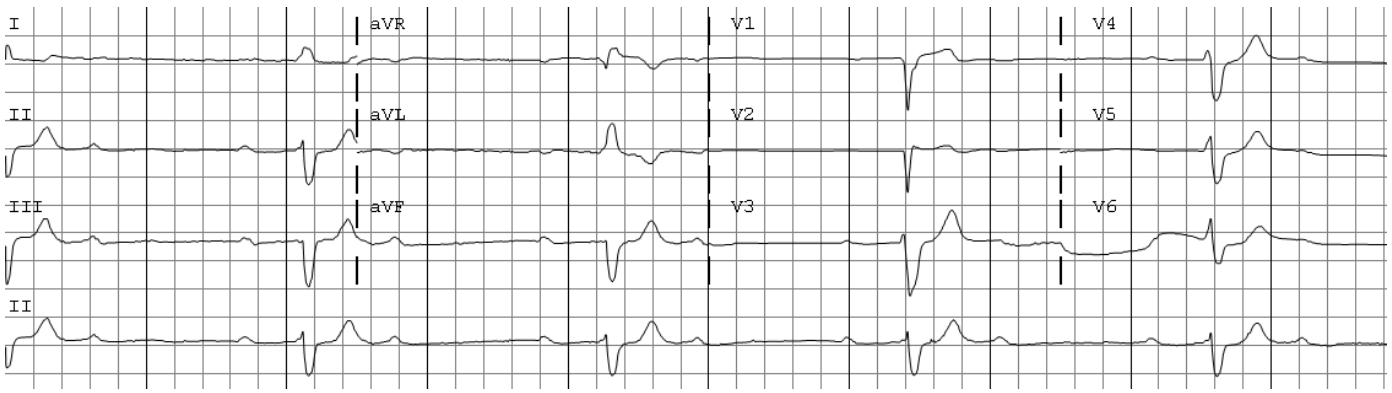

Figure 1:

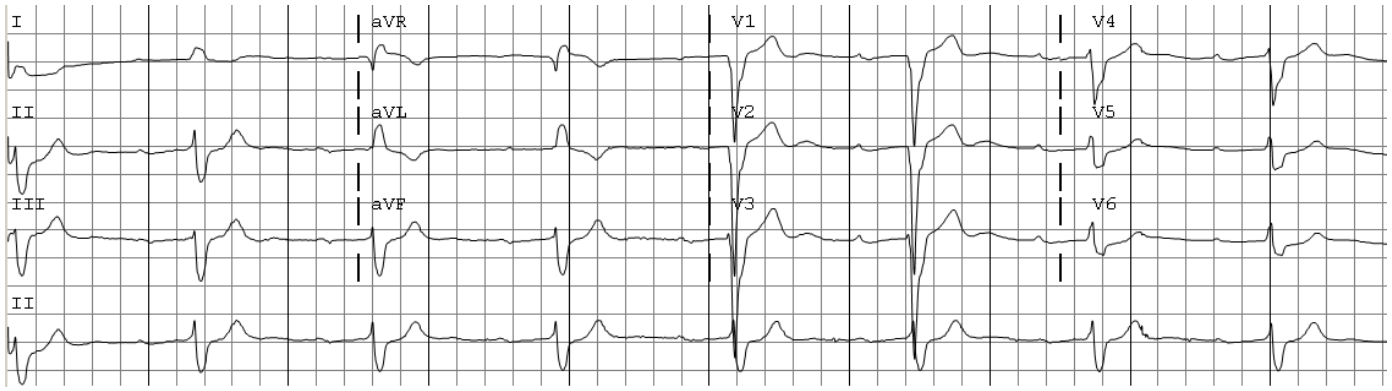

Figure 2: