Case Report

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A Case of Recurrent VT: ED Challenges in Evaluation and Management

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Abstract

Digoxin is a cardiac glycoside used widely in India for the treatment of heart failure and atrial fibrillation. Despite the apprehension of its use abroad, it continues to be a drug used extensively in our country when symptoms of heart failure remain despite use of other drugs. Digoxin toxicity can occur after an acute overdose or as a result of long-term therapy. It is also known to occur when serum digoxin levels are well within the therapeutic range owing to a variety of factors discussed below. Digoxin toxicity can present as almost any cardiac arrhythmia except rapidly conducted atrial dysrhythmias. Premature ventricular contractions and rarely, bidirectional ventricular tachycardia have be seen in this toxicity. Here we have reported a case of a 58 year old female, known case of RHD – post MVR with severe LV dysfunction presenting to the Emergency Department with progressively worsening palpitations and multiple epsiodes of vomiting over 1 day. On initial ED evaluation; patient was hypotensive and initial ECG was suggestive of first degree heart block. During further evaluation while in the ED; patient developed a wide complex regular tachycardia (Monomorphic VT) which was aborted only after DC cardioversion.

Keywords: Digoxin; Hypokalemia; Recurrent VT; Rheumatic heart disease (RHD).

Introduction

Digoxin toxicity serves as a diagnostic challenge in Emergency. These cases are more prone to misdiagnosis as a large number of patients have non cardiac presenting complaints such as GI distress (nausea, vomiting, abdominal pain, anorexia) or neurological symptoms (headache, dizziness, confusion, delirium, coma). Atrioventricular blocks and supraventricular tachycardia can also mimic digoxin toxicity.

Hence, early diagnosis warrants a keen eye for correlation of symptoms with the medication history. An elderly patient taking digoxin presenting with mental status change should be evaluated for toxicity. The severity of an acute toxicity correlates closely with the level of hyperkalemia, rather than the serum digoxin levels. Declining renal function and drug interactions, both common in elderly patients, lead to chronic toxicity even if digoxin is taken at therapeutic doses. Renal impairment also leads to delayed clearance of the drug from the body despite use of digoxin-specific antibody fragments (Digoxin Fab); which ideally causes clinical improvement within 1 hour. The lack of clinical awareness and atypical presentations can lead to delayed diagnosis; which can at times be fatal. Asymptomatic patients usually only require monitoring for symptoms of toxicity whereas symptomatic patients need to be evaluated depending on the degree of instability.

Case Presentation

A 58 year old female, known case of RHD – post MVR with severe LV dysfunction~ LVEF 20% and type II DM, presented to the Emergency Department with progressively worsening palpitations and multiple epsiodes of vomiting over 1 day. Patient was on cardiac medication as per brought by, drug

history was not available at emergency arrival of the patient. On initial ED evaluation; patients systolic blood pressure was 90 mmHg, diastolic BP was not recordable. Pt was conscious but restless, initial ECG was suggestive of first degree heart block (Fig 1). Pt was put on dobutamine and noradrenaline support. During further evaluation while in the ED; patient developed a wide complex regular tachycardia (Fig 2: Monomorphic VT); two boluses of IV amiodarone and one 5 mg dose of lidocaine was given but rhythm was aborted only after DC cardioversion. Again patient had two episodes of monomorphic VT; each aborted with DC cardioversion. Later laboratory evaluation shows low serum potassium and raised serum creatinine and urea levels, probably owing to dehydration due to concomitant use of prescribed oral diuretics and multiple vomitings. On admission to the cardiac care unit, serum digoxin levels were found to be 3.4 ng/mL (normal 0.8-2). Digoxin and diuretics were discontinued immediately. Patient was given Potassium supplementation and DigiFab (according to the serum digoxin levels). Pt responded well to the treatment; renal parameters normalised over the next few days and patient was discharged in satisfactory condition after a week.

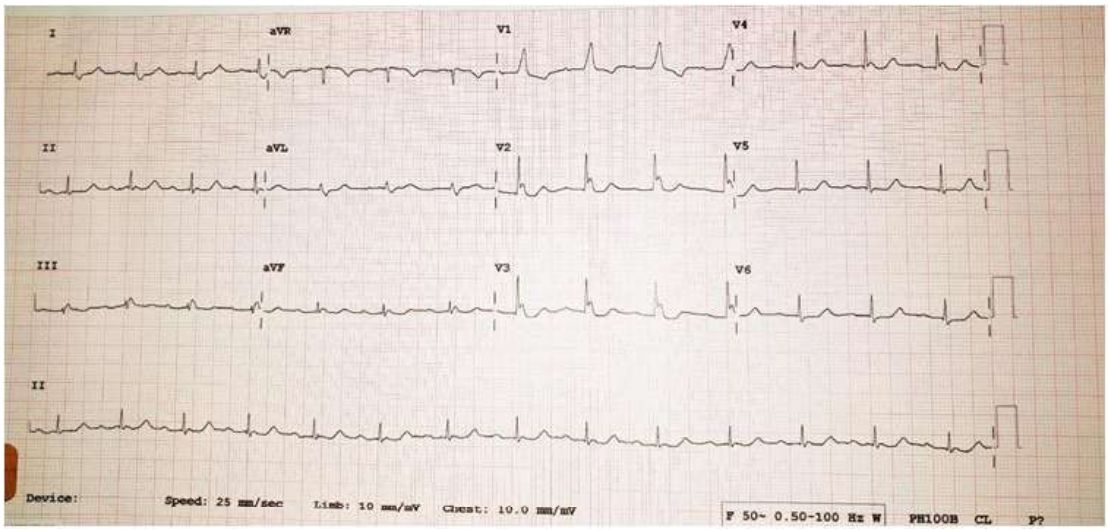

Fig. 1: 1st degree heart block

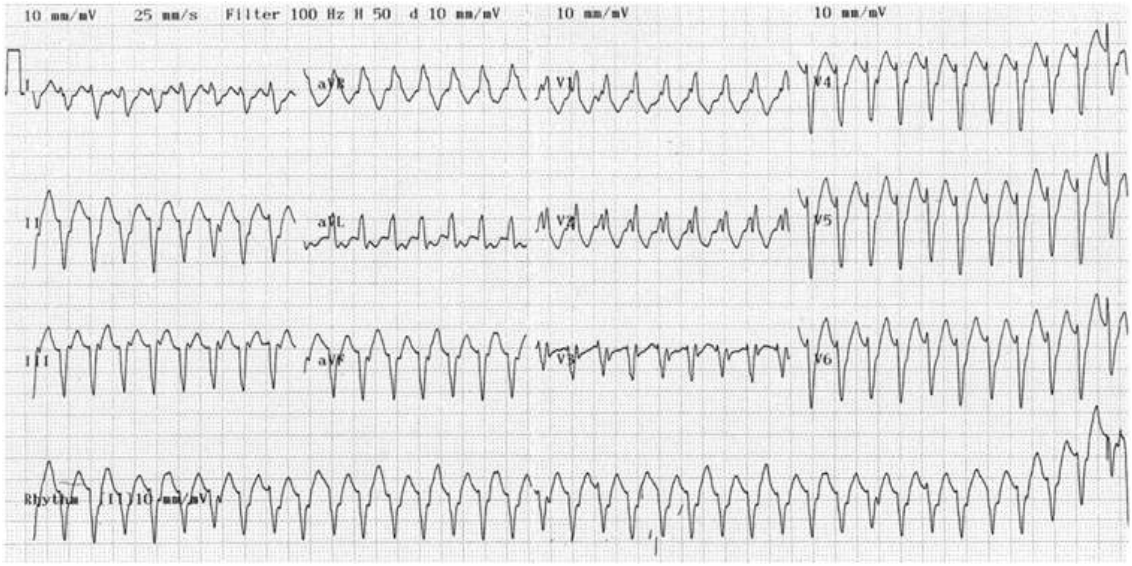

Fig. 2: VT (Ventricular Tachycardia)

Conclusion

Digoxin toxicity in this case was most likely a result of hypokalemia, which was a result of dehydration. Digoxin is an inhibitor of the sodium-potassium ATPase pump which leads to higher intracellular calcium levels and thus increased cardiac inotropy, raising the risk for tachyarrhythmias. Inhibition of this pump causes hyperkalaemia which is commonly seen in digoxin toxicity, but was not the case in this patient. In states of low potassium, digoxin toxicity is worsened because digoxin and potassium normally bind to the same site of the ATPase pump. During hypokalemia, digoxin binds to the ATPase pump easily, leading to the inhibitory effects.

Correction of serum potassium levels and use of adequate dose of digoxin Fab led to a favorable outcome of the patient. We report on this patient as it is an uncommon presentation of digoxin toxicity in a patient taking therapeutic doses of the same owing to impaired renal function. The case also demonstrates the prompt relief of symptoms post treatment as well as timely discharge from the hospital to satisfactory outpatient follow up.

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Permission- All pre-published information has been taken as a reference.

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