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DIGOXIN TOXICITY WITH NORMAL DIGOXIN AND SERUM POTASSIUM LEVELS: BEWARE OF MAGNESIUM, THE HIDDEN MALEFACTOR

Mamatha Punjee Raja Rao, MBBS,* Prashanth Panduranga, MRCP,† Kadhim Sulaiman, FRCPI,† and Mahmood Al-Jufaili, FRCPC*

*Department of Emergency Medicine and †Department of Cardiology, Royal Hospital, Muscat, Oman
Reprint Address: Mamatha Punjee Raja Rao, MBBS, Department of Emergency Medicine, Royal Hospital, Post Box 1331, Muscat-111, Sultanate of Oman

□ **Abstract—Background: In recent years, digoxin use has been on the decline, with decreased incidence of digoxin toxicity. Hence, digoxin toxicity, when it occurs, remains an elusive diagnosis to emergency physicians. Objective: To present a case of digoxin toxicity with normal levels of digoxin and serum potassium, but with severe hypomagnesemia. Case Report: A 66-year-old woman presented with junctional tachycardia and ectopic atrial tachycardia. She was known to have congestive cardiac failure on diuretic therapy. Her serum digoxin level was within the normal range (2.4 nmol/L [normal = 1.9–2.6]) along with a normal serum potassium level (3.9 mmol/L [normal = 3.5–5]). However, there was severe hypomagnesemia (0.39 mmol/L [normal = 0.65–1.25]) precipitating digoxin-induced dysrhythmia, which responded well to intravenous magnesium therapy. Conclusion: This case reiterates that digoxin toxicity can occur in patients with normal digoxin and potassium levels, and in such patients, magnesium needs to be checked and treated to prevent potentially life-threatening dysrhythmias.** © **2013 Elsevier Inc.**

□ **Keywords—digoxin toxicity; hypomagnesemia; hypokalemia; dysrhythmia**

INTRODUCTION

Digoxin toxicity can be acute, due to overdose, or chronic, when taken for a prolonged period of time. In the Digitalis Investigation Group trial, the overall incidence of digoxin toxicity was 2% over a 3-year period (1). Recently, the incidence of digoxin toxicity has been decreasing due to less use of digoxin in heart failure patients, and it remains an elusive diagnosis to emergency physicians (2). We present a case of digoxin toxicity presenting with junctional tachycardia and ectopic atrial tachycardia in an elderly patient with congestive cardiac failure.

CASE REPORT

A 66-year-old woman was referred from a health center to the Emergency Department (ED) with a 1-day history of abdominal discomfort, nausea, vomiting, and intermittent palpitations. Her past history was significant for diabetes, hypertension, severe left ventricular systolic dysfunction, and congestive cardiac failure. She was being treated for the last 6 months with anti-failure medications, and her current medications included furosemide 40 mg twice daily, spironolactone 25 mg once daily, digoxin 0.125 mg once daily, carvedilol 6.25 mg twice daily, and lisinopril 10 mg once daily, along with metformin and calcium supplement.

At presentation, the patient was conscious and oriented with a blood pressure of 145/70 mm Hg, pulse rate of 110 beats/min with no gallop, and a clear chest. Her initial electrocardiogram (ECG) done in the referral

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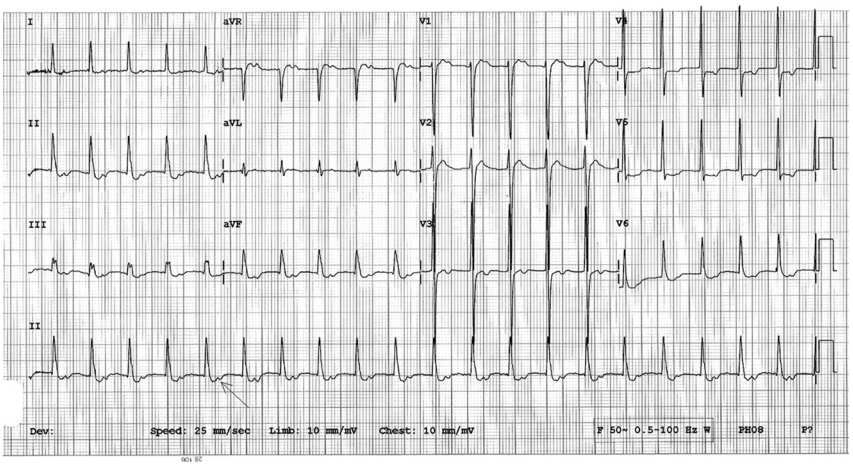

Figure 1. Twelve-lead electrocardiogram demonstrating a narrow QRS tachycardia with a heart rate of 130 beats/min with inverted P waves falling on T waves (arrow), and upright P in aVR suggestive of a junctional tachycardia.

health center demonstrated narrow QRS tachycardia with a heart rate of 130 beats/min with inverted P waves falling on T waves and upright P in aVR (Figure 1, arrowheads), suggestive of junctional tachycardia. An ECG done in our ED demonstrated a narrow QRS tachycardia, peaked P waves with prolonged PR interval at 110 beats/min heart rate, and typical ECG signs of the "digoxin effect" in the form of a scooped appearance of the asymmetric down-sloping ST depression ("reversed tick" sign) (Figure 2). This ECG was suspicious for an ectopic atrial tachycardia with 1:1 conduction. In view of the patient taking digoxin, she was suspected to have digoxin toxicity, and her blood was sent for routine blood tests along with digoxin levels. All of the patient's blood investigations were reported as normal, including free digoxin level (2.4 nmol/L [normal = 1.9–2.6] using Abbott AxSYM analyzer; Abbott Laboratories, Abbott Park, IL). Her troponin T was negative, serum potassium was 3.9 mmol/L (normal = 3.5–5), creatinine was 91 μmol/L (normal = 45–90), calculated glomerular filtration rate (GFR) was 54 mL/min/1.73 m^2, and brain natriuretic peptide was 2017 pg/mL (normal = 20–285). These results initially ruled out digoxin toxicity and suggested that the dysrhythmia may be due to underlying cardiomyopathy. However, in view of the ECG changes being highly suspicious for digoxin toxicity, her magnesium and calcium levels were requested. The calcium level was normal, but the serum magnesium level was low at 0.39 mmol/L (normal = 0.65–1.25). She was diagnosed to have digoxin toxicity precipitated by hypomagnesemia.

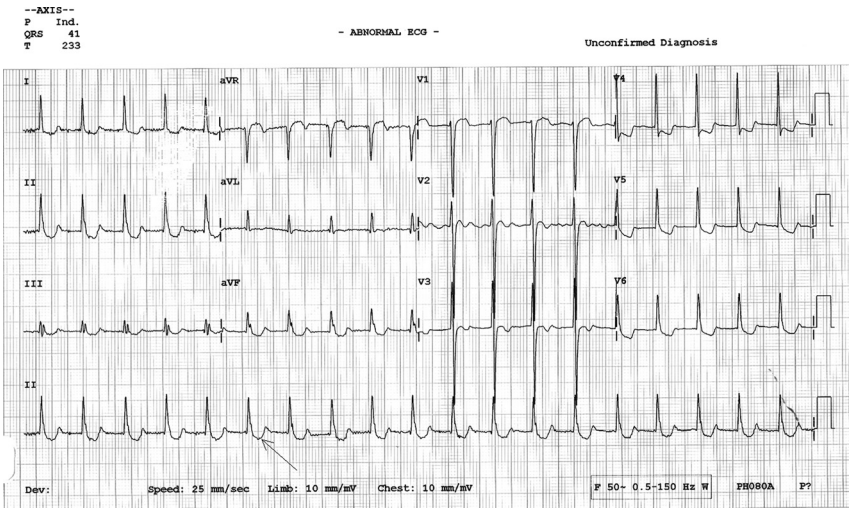

Figure 2. Twelve-lead electrocardiogram (ECG) illustrating a narrow QRS tachycardia, peaked P waves with prolonged PR interval at 110 beats/min heart rate, and typical ECG signs of the "digoxin effect" in the form of a scooped appearance of the asymmetric down-sloping ST depression ("reversed tick" sign; arrow). This ECG is suggestive of an ectopic atrial tachycardia with 1:1 conduction.