Case Report

Falling into complexity: A case of digitalis-induced fall, trauma, symptomatic bradycardia, and syncope

Rajathadri H. Ravikumar, Baby Pegu, Himanti Bansal, Kapil Dev Soni

Department of Anaesthesiology, Pain Medicine and Critical Care, All India Institute of Medical Sciences, New Delhi, Delhi, India

Abstract

Digoxin, a cardiac glycoside, functions by inhibiting the sodium potassium ATPase pump. It's crucial to note that digoxin has a very narrow therapeutic range. Its serum level vary due to changes in body weight, age, renal function, hepatic impairment and concomitant drug therapy. Chronic toxicity can lead to different types of arrrythmia,which span from heart blocks to ventricular tachycardia.This report present a case of an elderly male, where Digoxin toxicity resulted in syncope and mild traumatic brain injury. Initially upon patient's presentation ECG indicated myocardial infarction, subsequently bradycardia and complete heart block. The patient had a known history of chronic kidney disease and was prescribed 0.25mg of digoxin regularly without dose adjustment, which might have resulted in reduced digoxin elimination, leading to toxicity. Thus this case demonstrates a classic presentation of digoxin toxicity. Multiple risk factor such as old age, impaired renal function with continued digoxin treatment without dose adjustment was likely the cause of toxicity.

Keywords: Complete heart block, digoxin toxicity, trauma

Introduction

It is quite common to observe elderly individuals experiencing falls on even surfaces that result in trauma. These falls can stem from causes like a sudden foot slip or underlying factors such as syncope or seizures, leading to a loss of consciousness and subsequent fall. Among the potential triggers for syncope and falls, symptomatic bradycardia or heart blocks stand out as notable contributing factors.[1]

Digoxin, a medication often prescribed for patients with atrial fibrillation and heart failure, comes with a narrow therapeutic range. This means that the margin between its effective and toxic doses is quite small. One of the factors that can elevate drug levels in the body is impaired kidney function.[2] Elevated levels of the drug can trigger various types of arrhythmias, some of which can be life-threatening.

Here, we present a case exemplifying the consequences of digoxin toxicity, which led to a syncopal episode and subsequently resulted in mild traumatic brain injury and facial trauma.

Case

An elderly male presented to the trauma emergency department with a reported history of falling on level ground, resulting in a loss of consciousness (LOC). The patient was initially taken to a different hospital and subsequently referred to a higher center. Upon arrival, the patient's airway was clear, and cervical spine stabilization was ensured with a cervical collar. Breathing was unaffected, and the heart rate was measured at 80 beats per minute. The patient exhibited a good level of consciousness, registering a Glasgow Coma Scale (GCS) score of 15 out of 15.

Address for correspondence: Dr. Baby Pegu, Department of Anaesthesiology, Pain Medicine and Critical Care, All India Institute of Medical Sciences, New Delhi - 110 029, Delhi, India.
E-mail: Babypegu@gmail.com

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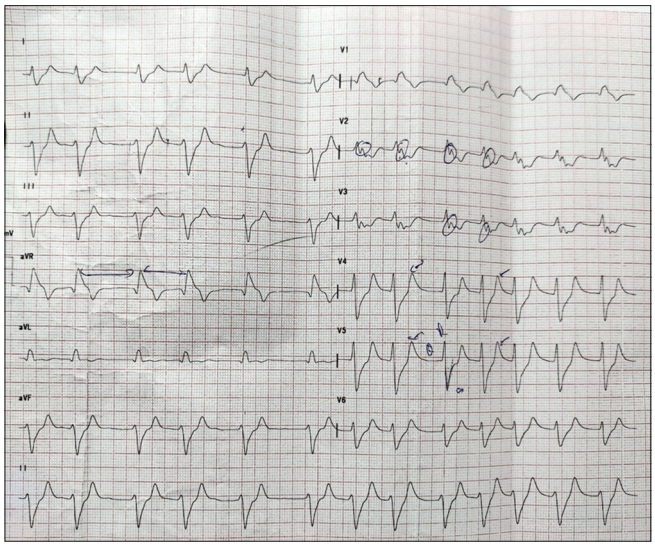

Figure 1: ECG on arrival showing hyperkalemic changes

An electrocardiogram (ECG) conducted in the emergency room revealed broad QRS complexes, a heart rate of 78 beats per minute, absent p waves, and an irregular rhythm see [Figure 1]. Subsequent arterial blood gas (ABG) analysis indicated a potassium level of 6.4. Prompt measures were taken to correct the elevated potassium levels, involving salbutamol nebulization and an insulin dextrose drip. Over time, the patient developed acidotic breathing and respiratory distress, for which he was intubated in the emergency department. History was taken during a secondary survey, which revealed that the patient is a known case of type 2 diabetes mellitus, hypertension, and chronic kidney disease (CKD) with a native urine output of 1.5 liters per day. He was not on any medication for the same. Chest X-ray was within normal limits.

Subsequently, the patient was transferred for a CT brain scan, which identified a small hemorrhagic contusion in the left frontal lobe, bilateral Le Fort III fractures, bilateral maxillary hemosinus along with fractures involving all walls, a fracture of the right mandibular body, a fracture of the left coronoid process, and a fracture of the angle of the mandible.

Following this, the patient was brought to the intensive care unit (ICU). Upon arrival in the ICU, the patient exhibited a heart rate ranging from 50 to 60 beats per minute, with the monitor displaying ST-T changes on the electrocardiogram (ECG). A subsequent 12-lead ECG [see Figure 2] revealed ST depression with T wave inversion in leads 2, 3, AVF, and chest leads V3-V6. Due to these ECG changes suggestive of a possible myocardial infarction, a qualitative troponin I test was conducted, which returned elevated results.

Unfortunately, the patient developed a complete heart block [see Figure 3] accompanied by a reduction in blood pressure. In response, an infusion of adrenaline was initiated. To further investigate potential cardiac issues, right-sided ECG leads and posterior leads ECG were obtained to rule out right ventricular myocardial infarction and posterior wall infarction, respectively [see Figure 4]. Echocardiography was found to be within normal limits.

After conducting a more comprehensive review of the patient's drug history, it came to light that the patient had been regularly ingesting a daily dose of 0.25 mg of digoxin for 6 months. However, dose adjustment was not conducted in accordance with any change in renal function. In hindsight, considering the patient's CKD, which resulted in reduced digoxin elimination, a diagnosis of digoxin toxicity was established. This toxicity likely contributed to the episode of syncope, ultimately resulting in the fall, symptomatic bradycardia, and hyperkalemia experienced by the patient. Laboratory analysis confirmed elevated digoxin levels, registering at 2.04 ng/ml. Also, a detailed history was taken to rule out intake of any other medication that might affect digoxin metabolism or renal clearance.

The patient's treatment involved the administration of adrenaline infusion for a duration of 18 hours, during which time his hemodynamic parameters showed improvement. ECG 24 hrs after the presentation showed down-sloping ST depression seen in patients taking digoxin. Subsequently, the patient underwent successful extubation and was subsequently transferred out of the ICU. After 48 hours of admission, the patient exhibited a normal electrocardiogram (ECG), and the digoxin-related ECG changes had been reversed [Figure 5].

Discussion

We present a case involving an elderly male who was regularly taking digoxin. His fall and subsequent syncope were attributed to digoxin toxicity, resulting in a mild traumatic brain injury.

Digoxin, a cardiac glycoside, functions by inhibiting the sodium-potassium ATPase pump, thereby enhancing heart contractility through increased intracellular calcium levels. It is crucial to note that digoxin's narrow therapeutic range has been well established. In 2022, the American Heart Association (AHA), American College of Cardiology (ACC), and Heart Failure Society of America (HFSA) recommended an upper therapeutic limit for digoxin in heart failure at 1.0 ng/ml. Elevated risks of mortality are associated with levels exceeding ≥1.2 ng/ml.[3] It is crucial to assess the electrolyte and renal status of each patient before initiating treatment, particularly in the elderly with multiple comorbidities. Impaired renal function can lead to higher-than-anticipated serum drug levels. Renal insufficiency also reduces the extravascular volume of distribution, necessitating a reduction in both the loading dose and maintenance dose for patients with underlying kidney disease.[4]

In our case, the patient had a known history of CKD and was prescribed 0.25 mg of digoxin regularly without adjusting the dose. Impaired renal function can lead to toxic levels of the drug in the body, which in turn can cause arrhythmias, syncope, or even sudden cardiac death due to ventricular tachycardia. In our patient's case, the toxicity reached a life-threatening stage,

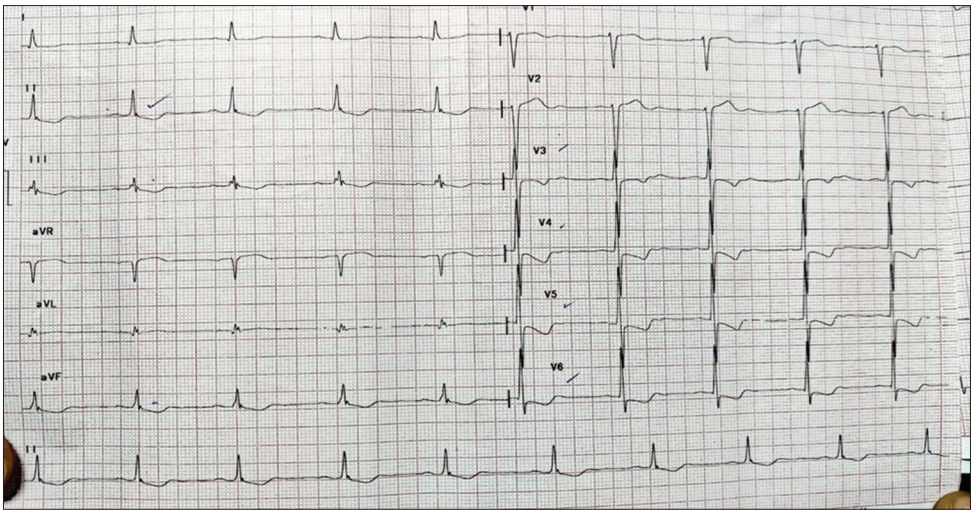

Figure 2: ECG showing ST depression and t wave inversion in leads 2,3, AVF, and V3-V6

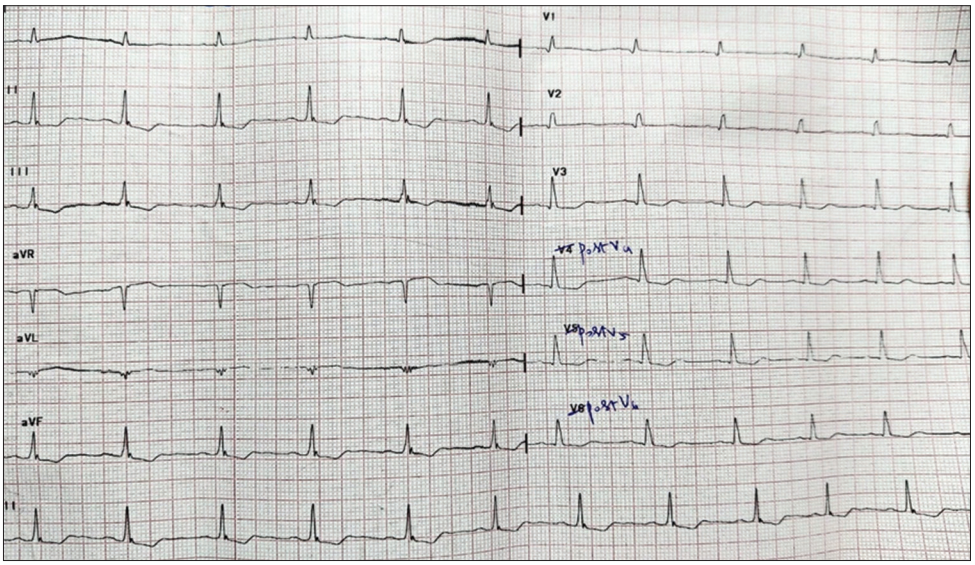

Figure 3: ECG showing a complete heart block

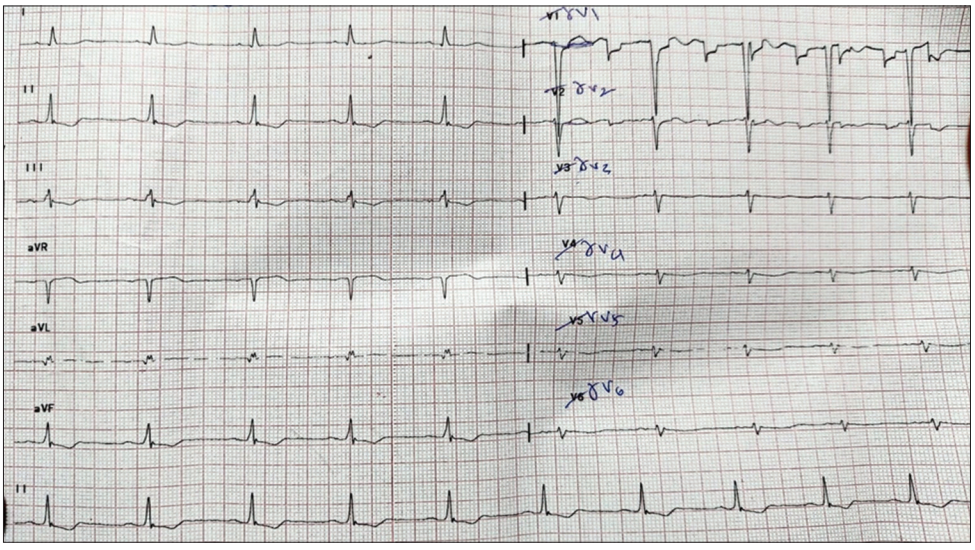

Figure 4: ECG performed with right-sided and posterior leads to rule out right ventricular and posterior wall myocardial infarction, respectively

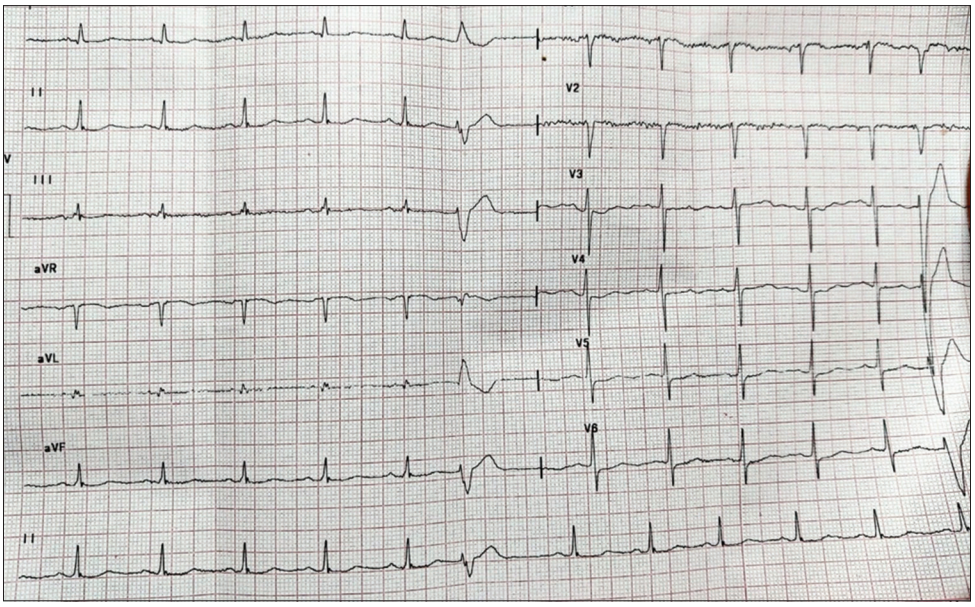

Figure 5: ECG showing normalization of digoxin-related changes 48 hrs post-admission

leading to a complete heart block and sudden syncope attack. Unfortunately, dialysis is not a feasible option for digoxin removal as it is not effectively eliminated this way. The antidote for digoxin toxicity is Digibind, a monoclonal antibody used in life-threatening cases. While the underlying cause was being identified, the acute crisis was managed with the administration of vasopressors until the critical phase was resolved.[5]

Conclusion

In conclusion, our case underscores the critical importance of thoroughly investigating the cause of falls in the elderly trauma patient population. It serves as a poignant reminder that seemingly isolated incidents of falls may be indicative of underlying medical conditions, necessitating a comprehensive diagnostic approach. In our case, the correlation between digoxin toxicity and the patient's fall emphasizes the need for meticulous monitoring and judicious use of medications with a narrow therapeutic index.

Digoxin, with its narrow therapeutic window and intricate interactions, demands careful consideration before initiation. Our case highlights the potential consequences of initiating digoxin without clear indications, underscoring the imperative for healthcare providers to adhere to established guidelines and exercise caution in prescribing medications with such characteristics.

This report not only contributes to the understanding of digoxin toxicity but also serves as a call to action for healthcare practitioners to approach falls in the elderly population with heightened vigilance. Identifying and addressing the root causes of falls can significantly impact patient outcomes and pave the way for more targeted and effective interventions in this vulnerable patient group.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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