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Case Report

A case report on digoxin toxicity

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ABSTRACT

Digoxin is an inotropic drug that is commonly prescribed in patient with heart related diseases. The effective dose of digoxin is 0.8-2.0 ng/ml beyond which digoxin causes toxic effects like visual dysfunction, irregular heartbeat, and cardiovascular collapse. Digoxin toxicity is caused due to increased automaticity and inotropy due to intracellular calcium and decreased dromotropy due to poisoning of sodium potassium transporter and AV nodal blockade. Digoxin toxicity should be assessed by continuous hemodynamic and cardiac monitoring including 12-lead electrocardiogram. There should be timely and immediate evaluation of electrolyte levels like potassium, calcium, serum creatinine and digoxin levels and prompt intensive care unit admission, if necessary. In this case study we studied a 43 old female patient prescribed with digoxin 0.25 mg, who had a medical history of congestive cardiac failure presented with symptoms of digoxin toxicity. The patient was assessed with electrocardiography (ECG) and managed by keeping digoxin on hold and effective patient counselling.

Keywords: Digoxin, Toxicity, Cardiac failure

INTRODUCTION

Digoxin is used in the treatment of various cardiac diseases such as congestive heart failure and atrial fibrillation. It is a cardiac glycoside derived from *Digitalis lanata* with a narrow therapeutic index.[1] The dose of digoxin in patients taking digoxin should be maintained at 0.8-2.0 ng/ml. Beyond the dose of 2.0 ng/ml, digoxin can cause toxic effects such as headache, gastrointestinal complaints, visual dysfunction, irregular heartbeat, and cardiovascular collapse.[2,3]

The pathophysiology of digoxin toxicity is due to increased intracellular calcium which leads to increased inotropy and automaticity and poisoning of Na-K transporter and AV nodal blockade from increased vagal tone which leads to decreased dromotropy. Digoxin is an inotropic drug used in patients with congestive heart failure where it improves systolic dysfunction and in patients with atrial tachydysrhythmias where it acts as atrioventricular nodal blocking agent.[4]

General principles of management include assessing the severity of toxicity and its aetiology like overdose, drug interaction, impaired drug metabolism due to diminished renal clearance or accidental ingestion, factors affecting the treatment like age, past and present medical history, chronicity of drug toxicity, comorbidities of heart or renal diseases and changes in electrocardioghraphy (ECG). There should be continuous hemodynamic and cardiac monitoring including 12-lead electrocardiogram. There should be timely and immediate evaluation of electrolyte levels like potassium, calcium, serum creatinine and digoxin levels and prompt intensive care unit admission, if necessary.[5]

CASE REPORT

A 43-year-old female patient came to the general medicine department of a tertiary care hospital with complaints of shortness of breath and palpitations. The patient is on irregular medication of tablet (tab.) lasix 40 mg, tab. digoxin 0.25 mg and tab. aldactone 50 mg. The past medical history includes congestive cardiac failure and

secondary mitral regurgitation. On examination the patient had orthopnoea and paroxysmal nocturnal dyspnoea (PND). The patient's vital data included afebrile, blood pressure of 180/100 mm Hg, pulse rate of 96 b/min, acute exacerbation, and paradoxical septal motion at mitral area. The patient was suspected to have sustained ventricular tachycardia, bigeminy secondary to digoxin toxicity.

Laboratory investigations

The patient was advised to get ECG, 2D Echo and other blood tests. The lipid profile was slightly elevated as shown; cholesterol (207.4 mg/dl), high density lipoprotein (HDL) (34 mg/dl) and low-density lipoprotein (LDL) (132 mg/dl) levels. The other tests like liver function test, renal function test, urine analysis, complete blood picture (CBP) and prothrombin time were found to be normal.

Echo Doppler study showed dilated left atrium/left ventricle/right atrium/right ventricle with ejection fraction of 24%. The ECG was abnormal with prolonged QT, left ventricular hypertrophy and lateral ST-T abnormality.

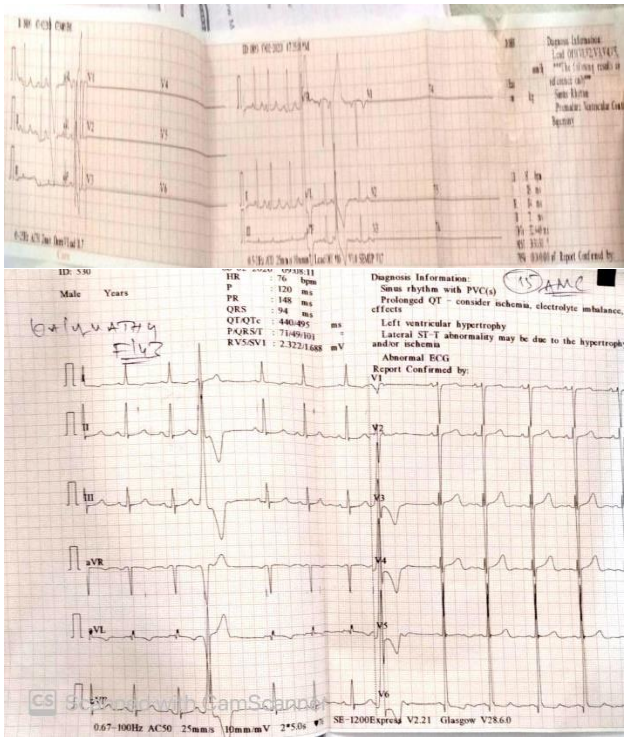

Figure 1: Electrocardiogram report.

From the above results obtained the patient was diagnosed with "ventricular bigeminy, secondary to digoxin toxicity."

The patient was managed with amiodarone, metoprolol, pantoprazole, aspirin, atorvastatin, furosemide, spironolactone and telmisartan during the stay at hospital. The symptoms subsided and the patient was feeling better. The patient was made aware about the symptoms and drug interactions of digoxin toxicity. The patient was advised to maintain adequate hydration.

DISCUSSION

The inotropic effects of digoxin on the heart are due to inhibition of sodium-potassium adenosine triphosphate (Na-K ATPase) pump. This results in transmembrane sodium gradient leading to increased intracellular sodium concentration and extracellular potassium concentration. This further leads to decrease in activity of Na/Ca exchanger that cause an increase in intracellular calcium levels. Other effects of digoxin include increase in cardiac output by improving left ventricular ejection fraction in patients with systolic dysfunction. Digoxin also acts as neurohormonal modulator by effecting Na-K ATPase in extra-cardiac tissues.[4,6,7]

Several drugs when prescribed along with digoxin can decrease or increase the concentration of digoxin. The drugs which increase the digoxin concentration are ACE inhibitors, angiotensin receptor blockers, calcium channel blockers, antibiotics (erythromycin, tetracyclines), antiarrhythmics, β-blockers (carvedilol), statins (rosuvastatin), diuretics, antifungals, proton pump inhibitors, anticholinergic drugs, some psychiatric drugs, ivabradine, cyclosporine, diclofenac, ibuprofen, indomethacin, and metformin. The dose of digoxin should be reduced or the frequency of dosing should be changed if the above-mentioned drugs are prescribed along with digoxin.[2,8]

The symptoms of digoxin toxicity are heterogenous and non-specific, hence digoxin toxicity is often insufficiently recognized by prescribers. The death rate of digoxin is also high when compared to other narrow therapeutic drugs. Therefore, it becomes mandatory to evaluate the effectiveness of digoxin level monitoring.[9]

The guidelines used to monitor digoxin levels in patients include assessment of condition of the patient by evaluating the subtherapeutic response which include atrial fibrillation/flutter, no improvement or worsening of heart failure or suspected noncompliance, drug interactions, and malabsorption. The patient is assessed for symptoms of digoxin toxicity like arrhythmias especially in the patients with renal dysfunction, diuretic therapy, and electrolyte abnormalities. The patients should be assessed 10 days after starting digoxin or upon admission if not checked within last 9 months or after a 10-month interval in outpatients on a stable dose of digoxin.[9]

CONCLUSION

Patients with congestive heart failure is often prescribed with digoxin; hence they should be frequently monitored for side effects and toxicity. Clinical pharmacist should play a crucial role in patient counselling and therapeutic drug monitoring. Effective therapeutic drug monitoring

can lead to a better treatment outcomes and improved quality-of-life of the patients.

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