A CASE REPORT ON DIGOXIN INTOXICATION IN A CLINICAL PHARMACIST’S PERSPECTIVE

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ABSTRACT

Context: Digoxin is a commonly used inotropic drug that has a narrow therapeutic range and is monitored poorly for its plasma concentration. It is commonly used in the management of cardiovascular disorders for its inotropic effects. Increased risk of chronic digoxin toxicity among the patients receiving digoxin pharmacotherapy is related to its narrow therapeutic range. Apart from its inotropic properties, it can also produce chronotropic and dromotropic effect. Its serum levels vary due to changes in body weight, age, renal function, hepatic impairment, and concomitant drug administrations. Patients receiving digoxin should be monitored periodically for potential drug interactions, adverse effects, toxicity, and other drug-related problems. Symptoms of digoxin-induced cardiotoxicity are difficult to be identified and may become fatal too. Therapeutic drug monitoring will play a significant role in reducing such drug therapy problems and will ensure safety and efficacy of the given drug.

Case Report: We report a case of chronic digoxin toxicity in a 22-year-old male with congestive cardiac failure - Class IV and atrial fibrillation with ventricular bigeminy.

Results: Patient’s renal parameters were elevated and among electrolytes, sodium level was decreased and potassium level was increased. A reduction in the volume of distribution of digoxin, due to impairment of renal function or congestive cardiac failure, might be one of the reasons for digoxin toxicity. Drug therapy problems (DTPs) such as untreated indication, inappropriate drug therapy, potential major drug-drug interactions, and drugs prescribed when contraindicated were observed.

Conclusion: Monitoring digoxin drug therapy can bring down the risk of digoxin intoxication.

Keywords: Digoxin toxicity, Digoxin plasma concentration, Therapeutic drug monitoring, Therapeutic range, Cardiovascular disease, Case report.

INTRODUCTION

Digoxin, a cardiac glycoside, is commonly used in the management of cardiovascular disorders such as congestive cardiac failure and cardiac arrhythmias for its inotropic effects. Increased risk of chronic digoxin toxicity among the patients receiving digoxin pharmacotherapy is related to its narrow therapeutic range (0.8–2 ng/ml) [1]. Apart from its inotropic properties, it can also produce chronotropic and dromotropic effect [2]. Symptoms of digoxin-induced cardiotoxicity are difficult to be identified and may become fatal too [3]. In this article, we discuss a case of digoxin intoxication in a clinical pharmacist perspective.

CASE REPORT

A 22-year-old man, Mr. X, was admitted to the coronary care unit through emergency.

Presenting complaint

Mr. X had been suffering from abdominal pain, reduced urine output, loss of appetite (symptoms of digoxin toxicity), loss of weight, and constipation for the past 1 week. He was also suffering from excessive cough and Class II–IV dyspnea. After admission, on the 6th day, he developed visual disturbance and delirium.

Medical history

Mr. X’s medical history included acyanotic congenital heart disease, congestive cardiac failure-Class IV, hyperacidity, and asthma. He was diagnosed with congenital corrected transposition of the great arteries at 17 years of age.

Medication history

Mr. X was on the following medications:
- Digoxin 0.25 mg OD
- Furosemide 20 mg BD
- Seroflo inhaler 2 puffs TDS

He was on irregular follow-up in multiple hospitals with anti-heart failure medications.

Physical examination

On examination, Mr. X was found to have AF with a pulse rate of 72 beats per minute (irregular). His jugular venous pressure (JVP) elevated up to the angle of mandible (An elevated JVP is typically a sign of congestive heart failure) and ‘A’ wave was absent (the absence of ‘A’ wave is seen in atrial fibrillation [AF]). He also had pedal edema up to knee and facial puffiness – ++.

Systems examination

Cardiovascular system (CVS): Upon CVS examination, Heart sound S1 was normal whereas S2 was irregular with a palpable impulse hyperdynamic over right midaxillary region. Parasternal heave was positive, grade IV/VI presystolic murmur over apical area and grade II/VI high pitched early diastolic murmur over pulmonary area was observed. A positive bilateral air entry and fine crepts were perceived on examining the respiratory system. Upon palpation, per abdomen was soft, fluid was present (+++) and huge hepatomegaly was noticed.

Other investigation results

Other investigation results were as follows:
have reported that digoxin is a drug with narrow therapeutic range and its serum levels vary due to changes in body weight, age, renal function, hepatic impairment, and concomitant drug administrations. An impaired volume of distribution of digoxin, due to reduced renal function (digoxin is excreted mainly by the kidneys) or congestive cardiac failure, might be one of the reasons for digoxin toxicity [6,7]. In Mr. X’s case, his renal function was deteriorating and he developed ARF and congestive cardiac failure.

Chadha et al. have reported that digoxin is a drug with narrow therapeutic index and can cause life-threatening arrhythmias when the plasma levels rise >2 ng/ml. Renal insufficiency interactions with other drugs that affect digoxin metabolism and electrolyte abnormalities (particularly hypokalemia) are the risk factors for digoxin toxicity [8,9]. Similarly, Mr. X’s renal parameters were elevated and among electrolytes, sodium level was decreased and potassium level was increased to 6.12 mmol/L. Hyperkalemia is a major concern in digoxin toxicity [10,11]. Potential drug-drug interactions might have also summed up the risk of hyperkalemia in him. Sani has reported that concomitant drugs prescribed (such as amiodarone, calcium antagonists, quinidine, diuretics, and indomethacin) may interfere with the plasma protein binding of digoxin [4]. Mr. X was also receiving diuretics that might have increased the risk of digoxin toxicity.

Bremner et al. have stated that it is significant to monitor plasma electrolyte levels and pH frequently in patients with digoxin toxicity. Digoxin intoxication may lead to hyperkalemia by generalized cellular loss of potassium with subsequent cardiac arrest. Induction of hyperkalemia has been proposed as a therapeutic measure in digoxin overdose, as elevated plasma potassium levels antagonize glycoside uptake at the sodium-potassium pump site [4,12,13]. In context to that, Mr. X’s potassium level was also elevated and hyperkalemia was left untreated monitoring potassium levels frequently.

Potential drug interactions:

<table>
<thead>
<tr>
<th>Category</th>
<th>Number of interactions</th>
<th>Drugs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Major</td>
<td>6</td>
<td>Calcium+Digoxin, Digoxin+Dopamine, Digoxin+Spironolactone, Fentanyl+Morphine, Fentanyl+Tramadol, Morphine+Tramadol</td>
</tr>
</tbody>
</table>
Mr. X was discharged on request despite being in an unstable condition (inotropic support). He was given furosemide, losartan, and pantoprazole.

CONCLUSION

Patients receiving digoxin, a drug with narrow therapeutic index should be monitored periodically for potential drug interactions, adverse effects, toxicity, and other drug-related problems. Therapeutic drug monitoring will play a significant role in reducing such DTP and will ensure safety and efficacy of the given drug.

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