Failed suicide attempt: A case report: Digoxin and insulin toxicity, antagonist effect

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ABSTRACT

Digoxin is used to treat heart failure and arrhythmias. Even though its used is decreased, the toxicity of digoxin still remains a problem for physicians. We present a case of a middle-aged male pharmacist, diabetic, who intentionally ingested a toxic dose of digoxin and insulin with his random blood glucose (RBS) of 40mg/dl. He was hemodynamically stable and his ECG showed no arrhythmias. His initial investigations showed normal potassium level and digoxin level of >7.51ng/ml. He was closely observed in the emergency Department (ED). Patient was admitted to the hospital for close observation. Even though the patient’s digoxin level was toxic; his potassium levels were within the normal limits, and his vitals and serial ECGs were all unremarkable. These findings shine the light regarding the insulin and digoxin, in that the insulin maybe a life saving intervention in case of digoxin toxicity.

Keywords: Digoxin, Insulin, Suicide, ECG, overdose, and Potassium.

Introduction

Digoxin is a cardiac glycoside, which was discovered in 1785 to treat heart failure and arrhythmias [1]. With the overall use of digoxin has decreased, the toxicity of digoxin has still remained stable and the use of the digitalis antibody fragments has increased [2]. Digoxin work on the inhibition of sodium-potassium ATPase pump, which leads to increase the intracellular sodium. We present a case of suicidal attempt by ingestion of large amount of digoxin and insulin simultaneously and was effectively managed in the emergency department. Case report: An adult male, pharmacist, who is known to be diabetic, presented to the emergency department by the ambulance after ingestion of 25mg of digoxin and taking 300 IU of aspartate insulin subcutaneously in attempt of suicide. His initial vitals were Blood Pressure: 135/70 mmHg, Pulse: 85 bpm, Oxygen saturation 100% in room air, Respiratory rate: 17, except of his RBS, which was 40mg/dl. He received D50W 50cc IV bolus. In the ED, he was connected to a monitor. He was maintained on D5NS 150cc/hr. His vitals continued to be within the normal limits. His physical examination was unremarkable. His EKG showed in Figure 1. His following blood works shows RBS of 67mg/dl, Liver enzymes within the normal range, renal function test are within normal, as well as the Potassium level of 4.8 mEq/L, digoxin level was more than >7.51ng/ml. As hospital’s laboratory cannot detect levels higher, Acetaminophen level & ASA level were normal. The patient was closely observed in the ED for 8 hours, with his serial ECGs show no arrhythmias, his potassium level 4.9 mEq/L, RBS 73mg/dl, and his digoxin level 6.71ng/ml. He was admitted to cardiac care unite for close observation for two days and was then admitted to psychiatric department for evaluation. Discussion: Digoxin work on the inhibition of sodium-potassium ATPase pump, which leads to increase
the intracellular sodium [3]. This increase in sodium leads to the reduction of the sodium-calcium exchanger. The resulting increase in calcium is the responsible of the positive inotropic activity of digoxin [3]. Digoxin also works on the vagal tone, by increasing its effect centrally and peripherally [3]. Life threatening digoxin-induced arrhythmias occurs when digoxin levels raises above 2.0ng/mL [4,5]. However, in the presence of comorbidities such as hypokalemia, hypomagnesaemia, hypercalcaemia, the toxic effect of digoxin can occur in much lower levels or even normal levels [6,7]. Insulin is a 51-amino acid peptide hormone that is synthesized and secreted by pancreatic beta cells. Also, the insulin has found to shift the potassium inside the cell. In DKA and HHS (moderate to severe) the insulin is used as a treatment if the serum potassium is ≥3.3 mEq/L, since the insulin will worsen the hypokalemia to avoid possible arrhythmias, cardiac arrest, and respiratory muscle weakness [8,9,10]. Plasma potassium is a major determinant of the resting potential of all cells. Hyperkalemia and hypokalemia are silent yet fatal disturbances, because of their arrhythmogenic potentials. Hyperkalemia is a very common finding in acute digoxin toxicity, and its considered as a reflection of toxicity and a risk of death [11]. In our case the potassium is normal, this raises the question of the effect of insulin on the levels of potassium and the outcome of patients in the sitting of acute digoxin toxicity. Oubaassine R found that glucose-insulin infusion in acute toxicity of digoxin is cardio-protective [12]. He also found that insulin interact with sodium-potassium ATPase pump [13], which support our case in which that insulin can have a protective effect in digoxin toxicity and can improve the outcome. Ashish Garg et al reported a case of severe digoxin toxicity and propranolol and simultaneous insulin administration, the patient shows normal Potassium levels and non-arrhythmic ECG [14]. Trang Q. Nguyen et al found that insulin-stimulated intracellular uptake of glucose and potassium are independent of each other [15].

Conclusion

Insulin administration in case of acute Digoxin toxicity maybe a life saving intervention, affecting the level of Potassium, which in turns prevent life threatening arrhythmias.

References


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