Suicidal Failure Following A Massive Digoxin Overdose:

A Case Report

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ABSTRACT

A 22-year-old Palestinian female was under digoxin therapy after a mitral valve replacement for one and half year duration. She swallowed 75 tablets (0.25mg) of digoxin, in suicidal attempt. Prior to admission to the hospital, she vomited 4-5 times at home. The prominent sign was ejection systolic murmur in aortic area. The patient showed no signs of toxicity e.g., palpitation, altered vision, abdominal pain, diarrhea and the vital signs were normal. Serum digoxin level was 7.6 ng/ml. No anti-digoxin antibody fragments were administered. After 5 days of admission to intensive cardiac care unit she was discharged and referred to psychiatric counseling. Although the digoxin level was very high, the patient was asymptomatic and without complains. This case reports indicates that some patients can tolerate high digoxin overdoses without deleterious effects.

(Key words: Digoxin, Therapeutic Effect, Toxic Dose, Therapeutic Index, Lethal Dose, Poisoning)

INTRODUCTION

Digoxin is a cardiac glycoside and is used in the treatment of congestive heart failure, ventricular and paroxysmal atrial tachycardia. The ancient Egyptians used cardiac glycosides as a poison but the Romans used them as a cardiovascular tonic. Digoxin inhibits the Na-K-ATPase pump in the myocardial membrane. Its inotropic action is to increase intracellular calcium (Smith, 1988), which also forms the basis for arrhythmias related to digitalis intoxication. It is believed that increased intracellular concentrations of calcium allow for greater activation of contractile proteins (e.g., actin, myosin). Digoxin is rapidly absorbed from the Gastrointestinal tract following an oral dose. It is distributed throughout the body tissues, with the highest concentrations found in the heart, kidneys, intestine, liver, stomach, and skeletal muscle (Stan R., 1996). Small amounts can be found in the brain. The presence of congestive heart failure slows the rate at which steady-state distribution is achieved. Only 20—30% of the drug is protein-bound. Onset of therapeutic effect generally occurs within 30 minutes to 2 hours after oral administration and within 5–30 minutes following Intravenous administration. Thirty to fifty percent of a dose is excreted unchanged in the urine. A small amount of digoxin is metabolized in the liver to inactive metabolites. In approximately 10% of patients, however, significant amount of orally ingested digoxin is metabolized in the gut by the intestinal bacteria. The elimination half-life of digoxin is 16 to 20 hours but patients with renal impairment and end-stage renal disease have elimination half-life values that are prolonged up to 10-fold in magnitude, while volume of distribution is unaffected (Goodman and Gilman,1996).

DISCUSSION

The patient described in this case study is an assistant pharmacist by occupation, so she had free and easy access to the drug. She ingested 75 tablets (0.25mg), in a suicidal attempt after a social conflict. According to this massive dose, the expected serum concentration should be > 35 ng/ml, but serum digoxin level when measured by TDX/FLX automated machine was only 7.6 ng/ml. The patient vomited at least four times, in addition to the gastric wash done in emergency room by which most of the ingested drug was eliminated. This emphasizes the importance of emesis induction and gastric lavage to mechanically remove unabsorbed drugs from the stomach in certain overdoses. Clinical toxicity occurs with digoxin levels in excess of 3 to 5 ng/ml (Harison's Principles of Internal Medicine, 1998). The mortality rate was as high as 50% among those with serum digoxin level above 6 ng/ml (Chamberlin Susan, 1997).

The patient serum level of digoxin was 7.6 ng/ml, however, the patient did not develop any symptoms. This may show that she developed some tolerance to the drug. The haematology profile was as follows: White Blood Cells = 11.3 THSD/CU MM, Red Blood Cells = 5.53 MILL/CU MM, Haemoglobin = 16.1 GRAMS/DL, HCT = 46.8%, Platelet = 175 THSD/CU MM, Prothrombin time: normal, ABG: normal. The biochemical results were as follows: Urea = 2.77 mmol/L, Creatinine = 63.8 umol/L, Sodium = 145 mmol/L, Potassium = 4.3 mmol/L), Aspertate transaminase = 12 U/L, Alanine transaminase = 37 U/L, Alkaline phosphatase = 78 U/L, Total Bilirubin = 4.54 umol/L, Cholesterol = 401 mmol/L, Triglyceride = 0.7 mmol/L, Mg = 0.8, Phosphorus = 1.1 mmol/L, Total protein = 67 GRAMS/DL, Albumin = 47 GRAMS/DL, Creatinine phosphokinase = 95 U/L, Lactate dehydrogenase = 298 U/L. All of these results were within the normal limits.

Knowing the plasma digoxin concentration alone is not sufficient for optimal treatment. Several factors change the tissue response to digoxin and must be considered, e.g., electrolyte disturbances, thyroid disease and age (AronsonJ K, and Hardman M., 1993). Patients with a serum potassium level above 5 mmol/L usually need digoxin immune Fab, too. When digoxin levels are elevated, the body can no longer pump potassium into the cells, and serum potassium rises. When it exceeds 5 mmol/L, cardiac arrest may occur (Chamberlin Susan, 1997). It should be mentioned here that the potassium level in our patient was not elevated (4.3 mmol/L) therefore an acute poisoning was ruled out. However the patient was observed in the intensive cardiac care unit. Five days later she was discharged and referred to psychiatric counseling.

CONCLUSIONS

Although the digoxin level was very high, the patient described in this case study was asymptomatic and without complaints. This observations reported in this case study indicates that some patients can tolerate even higher digoxin overdoses without deleterious effects generally reported in the literature. Keeping in view of our observations, it is highly recommended that psychiatric patients should not be exposed to or have easy access to drug storage cabinets or distribution centers. Special care must be given to those with a

history of previous suicidal attempts, because they are at high risk of repeated attempts (Lebdeh, W.A.R.A., 2000).

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