

Digitalis toxicity report

[Health & Medicine](#), [Drugs](#)



The accounts of digitalis toxicity due to overdose in 1985 specify 1, 015 cases including 584 patients that are below 6 years old and 56 patients aging 6-17 years old. The greater part of these documented toxicity cases (83%) come about without the purpose of overdosing (Kwon, 2006). The prevalence of digitalis toxicity had a rising trend for some time until it was acknowledged in the early 1990's that reduction in toxicity cases was observed. Among the studies that concluded the decreased cases of digitalis toxicity was the research conducted by Haynes et al.

In there study, it was noted that the cases of digitalis toxicity in United States and United Kingdom manifested a decreasing trend in the past two decades. Hospitalizations in relation to digitalis toxicity were notably reduced in United States whereas in United Kingdom the cases of ambulatory digitalis toxicity also lessened. The decreased incidence of digitalis toxicity in the U. S. is correlated to the diminished administration of this drug. The dilemma due to digitalis toxicity has significantly reduced in the two above mentioned countries (Haynes, et al, 2008).

Though incidence of digitalis toxicity is turning to the decreasing side it is no reason to disregard the threats of toxicity that consumers of this substance are exposed to. Digitalis is drug extracted from the leaves of the plant called Foxglove (*Digitalis purpurea*). The utilization of foxglove as a medicinal plant already exists for centuries. But the popularity of this plant was not that intense until an English botanist and physician named William Withering in the 1700's pioneered the establishing of digitalis as a cardiac drug.

This doctor conducted a detailed study of digitalis. Withering was also responsible for the determination of the most effective preparation of the drug as well as the correct dosages for various heart ailments. This English physician was also responsible for the setting up the standards of when to abort the therapy using digitalis because of its toxic effects (NetIndustries, 2008). The mode of action of digitoxin involves the inhibition of the Na-K ATPase in myocytes to increase heart muscles contractility.

The drug attach to the binding sites situated in the extra cytoplasm of the sodium- and potassium-activated adenosine triphosphate (Na-K ATPase) pump preventing the active transfer of Na and K across the cell membranes. The resulting high concentrations of sodium and calcium as well as the low amounts of potassium in the intracellular part of the muscle cell promotes the fourth stage myocardial action potential creating a decreased conduction velocity and amplification of ectopic activity.

The end result boost in the contractility of heart muscles due to the action of digitalis is beneficial to various heart ailments (Kwon, 2006). This is utilized as a drug therapy for heart problems. This substance is specifically indicated in cases of persistent systolic heart failure symptoms despite the administration of diuretics, angiotensin converting enzyme (ACE), and beta blocker; and, cases of congestive heart failure with atrial fibrillation.

The objective of the therapy using digitoxin ranges from 0.5 to 1.0 ng/mL. The administration of digitoxin is contraindicated in patients that are receiving primary therapy for acute decompensated heart failure stabilization and during cases of sinus or atrioventricular (AV) except for cases of prior pacemaker treatment applied (Kwon, 2006). Medicine

administered within the range of its therapeutic dose rarely produce toxicity. The established daily therapeutic dose of digitoxin varies from 0.0005 mg/kg (for young infants) to 0.75 mg/kg (for mature individuals).

This drug in tablet preparations has the estimated absorption of 70-80% and a bioavailability of 95%. In oral administrations (per os / PO), the action onset of digitoxin transpires after 30-120 minutes whereas in intravenous route action onset to occur requires only 5-30 minutes. The threshold of the effect of this drug after oral and intravenous routes is 2-6 hrs and 5-30 minutes respectively. An estimate of 60-80% of the digitoxin intake is excreted by the kidney without structure and properties change (Kwon, 2006). The lethal dose of this drug varies with the age of the patients.

Doses above 10 mg per individual even in healthy adults will cause death but doses lower than 5 mg infrequently produces problems such as toxicity. In children, the intake of doses above 0.3 mg/kg or 4 mg per individual often causes fatality (Kwon, 2006). The population which is highly at risk with the development of digitoxin toxicity are the infants and the old people. The threats of digitalis toxicity include intake of medicines like digitoxin and digoxin; and, digitalis interaction with other drugs like verapamil, amiodarone, and quinidine.

Having below normal levels of potassium in the body such as the patients medicated with potassium losing diuretics is also at risk of the toxic effects of digitalis. People with kidney damage and having little amounts of magnesium are also prone to digitalis toxicity. Caution should be observed in administering digitalis as well as other medicines to patients with kidney damage because the capacity of the body to excrete any drug taken is also

diminished along with the kidney problem. Thus, the drug has the tendency to accumulate in the kidney and increasing the possibility of toxicity (“Digitalis Toxicity”).

Occurrence of digitalis toxicity can be due to two mechanisms: the above therapeutic amounts of digitalis in the patient’s body, and the lowering of the patient’s digitalis tolerance. The toxicity can be caused by either or both of the mechanisms. The toxicity of this drug can happen with one exposure to the drug as well as the gradual toxicity. Some patients suffer the effects of digitalis toxicity despite the normal blood levels of this drug because of the existence of other digitalis toxicity risk factors (“Digitalis Toxicity”).

Other disease and metabolic conditions that serve as risk factors of the toxic effects of this drug are: hypoxemia, hypothyroidism, and alkalosis (Kwon, 2006). The mortality rates due to digitalis toxicity vary with the details of the population. The direct consequence of cardiac toxicity in digitalis toxicity result to 3-21% mortality rate. Male individuals are more prone to this drug’s toxicity compared to the females. The young and old people have increased risks to digitalis toxicity than the other age brackets.

Ingestion of digitalis medicines of their grandparents is the primary cause of toxicity among children (Kwon, 2006). The symptoms of toxicity due to digitalis include strange changes in vision like color perception problems, blurring of vision, having visual blind spots, and having visual bright light spots; nausea; vomiting; pulse irregularities; appetite loss; palpitations; confusion; general swelling; lower urine volume; lowered consciousness; and, breathing difficulty during lying down (“Digitalis Toxicity”).

The treatment regimen for digitalis toxicities comprise of specific, symptomatic, and supportive therapy phases. The supportive therapy phase for this toxicity case consists of electrolyte imbalance correction, dehydration treatment using IV fluids, and oxygen support equipped with ventilation. It is frequently prescribed by medical practitioners to supplement potassium in cases wherein the patient has potassium levels lower than 4 mmol/L.

The recommendation of diuresis induction is not approved due to the tendency to aggravate the electrolyte imbalances and the renal excretion of the drug is not enhanced by this process (Kwon, 2006). The specific therapy phase involves the administration of digoxin-specific Fab antibody fragments that are noted to be of significant success in treating severe acute digitalis toxicity. This drug is sort of the antidote for digitalis toxicities as well as other complications in relation to digitalis.

Immediate administration of digoxin-specific Fab antibody is recommended upon deducing digitalis toxicity. The prompt treatment digoxin immune fab will decrease the morbidity and mortality rates of digitalis toxicities. To contradict arrhythmias that might occur in digitalis toxicity treatment with phynetoin is advised (Kwon, 2006). The recommended method for gastrointestinal cleansing is the utilization of multiple-dose activated charcoal (1gram/kilogram weight of patient/day). Administration of ipecac syrup to induce emesis is contraindicated due to the activation of the vagal tones.

Other possible methodologies of eliminating the toxic amounts of digitalis in a patient's body are gastric lavage, whole-bowel irrigations, and steroid

binding resins like colestipol and cholestyramine. These three aforementioned therapeutic regimens though have constraints like the vagal effects and the lack of substantial data to support their efficacy in these toxicity cases (Kwon, 2006). Even if the incidence of digitalis toxicity cases have plunged the vigilance regarding this condition should not stop.

The drug prescriptions of digitalis for heart problems should be ensured by the medical practitioners to be under the therapeutic dosages. The availability of this drug to children should also be eliminated to prevent the accidental ingestion of this drug. Since digitalis is an important cardiac drug various researches has been conducted involving this medicinal substance. The medical industry should not stop there though; further studies can still be done to improve the value of digitalis as a therapeutic agent without compromising the patient's safety.