Digoxin Therapeutic Drug Monitoring

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Opinion

The kidneys are the main organs that remove digoxin from the body. Because renal malfunction and low potassium levels can cause symptoms of digoxin poisoning, your healthcare practitioner may wish to monitor kidney function and blood potassium levels if you have kidney difficulties. Digoxin is a cardiotonic glycoside from the digitalis family that is primarily used to treat heart failure and atrial fibrillation Hydrogen Fluoride/Atrial Fibrillation (HF/AF). Because of its limited therapeutic scope, its significant toxicity, as well as the large inter-individual variation, are all factors to consider.

Variability in pharmacokinetics and the possibility of medication interactions a consistent dose occurs with chronic cardiac therapy; individualization of dosages necessitates digoxinemia administered. The goal of this research is to demonstrate the interest in Digoxin therapeutic medication monitoring in patients with heart failure while aiming for a low digoxinemia that corresponds to a maximum. It has a therapeutic effect and a low danger of overdosing.

This is a 5-month prospective trial in patients with (HF/AF), conducted in the context of Therapeutic Drug Monitoring (TDM). All of the patients were given digoxin in addition to other cardiology medications. An information sheet was used to collect data on the patient, his clinical and biological state, dose, the date of treatment initiation, the date of the most recent dosage modification, the time of sample, and the medicines involved. The Enzyme-Multiplied Immunoassay Technique (EMIT) was used to assess digoxinemia, with a measurement range of 0.5 ng/ml to 1.2 ng/ml. The study included 38 digoxin-treated individuals ranging in age from 20 months to 90 years. The findings show that, on the one hand, a maximum therapeutic effect was obtained for weak digoxinemias between 0.5 ng/ml and 0.8 ng/ ml, further associating a minimal risk of overdose, and, on the other hand, a concentration higher than 1.2 ng/ml resulted in the appearance of severe signs of toxicity, further associating significant cardiac rhythm disturbances.

Therapeutic levels of digoxin are 0.8 ng/mL-2.0 ng/mL. The toxic level is >2.4 ng/mL. Digoxin is a drug that is used to treat heart failure and irregular heartbeats (arrhythmias). It aids in the proper functioning of the heart as well as the regulation of your heart rate. Digoxin is a class of cardiac glycosides used to treat systolic heart failure and atrial fibrillation. The sodium-potassium ATPase is inhibited by this drug. This enzyme transports sodium out of the cells while bringing potassium in. This enzyme is inhibited by digoxin. The intracellular sodium level rises as a result of this. The sodium-calcium inhibitor comes after the inhibition. Ca accumulates intracellularly as a result of the exchange. This mechanism's specific mechanism is unknown. The nature of the exchange is yet unknown. The intracellular calcium then binds to Tropin C, which triggers a cascade of events. The heart muscle's contractility improves.Many factors influence the quantity of digoxin in the blood, including medicines, diet, electrolytes, and renal function. The toxicity of digoxin is extremely prevalent. Ventricular tachycardia, ventricular ectopic beats, 2nd or 3rd degree heart block, and SA node arrest are the most prevalent side effects of digoxin. Anorexia, diarrhea, weariness, confusion, and altered colour vision (excess yellow/green) are also typical non-cardiac adverse effects

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