HA-230 Hemoperfusion Cartridge in the Treatment of Digoxin Toxicity in a 67-year-old male with End-Stage Renal disease: A Case Report

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BACKGROUND: Digoxin is a non-dialyzable drug that is part of a drug class called cardiac glycosides. These medications work by inhibiting the Na-K-ATPase within the cardiac myocytes thereby causing shifts in the intracellular sodium gradient within the muscle. This increase in sodium gradient increases intracellular calcium ions allowing for increased contractility of the myocytes [5]. Additionally, digoxin affects vagal tone, these actions of digoxin can easily cause arrhythmias in patients especially if digoxin levels are at toxic levels [10]. Having a narrow therapeutic range (0.5-1.0 ng/ml)[1], digoxin can easily result in toxic or sub-therapeutic levels, hence doses must be adjusted to cater to an individual's renal function[8]. In patients with End Stage Renal Disease, digoxin use is associated with increased risk for toxicity and subsequently mortality [7]. Hemoperfusion refers to the circulation of anticoagulated blood into an extracorporeal circuit utilizing adsorbent cartridges to remove specific toxins. [9] The HA-230 in particular has a resin pore size of 200 D–10 kD and can remove drugs and toxins of molecular weights of 500 D – 10 KD [2]. Clearance of digitalis is therefore possible through hemoperfusion using this cartridge.

CASE PRESENTATION: A 67-year-old male, diabetic, hypertensive with heart failure on digoxin, and ESRD on renal replacement therapy and hemodialysis 3x/week came in due to 1 episode of vomiting few hours prior to admission. He reported feeling nauseated during the hemodialysis, which led to an episode of vomiting after the session. This prompted laboratories and ECG to be done revealing normal blood chemistry however ECG revealed sinus bradycardia with poor R wave progression on leads V1-V3 and a mobitz type II heart block. Patient was immediately brought to our institution for observation and evaluation.

At the ER, physical exam revealed rhonchi on both lower lung fields and Grade II bipedal pitting edema. Patient was admitted at the ICU for close monitoring and observation. ECG done at the ER (Fig. 1) noted findings of atrial fibrillation in controlled ventricular response with occasional ventricular ectopic complexes, suspecting digoxin toxicity hence, a digoxin serum assay was done.

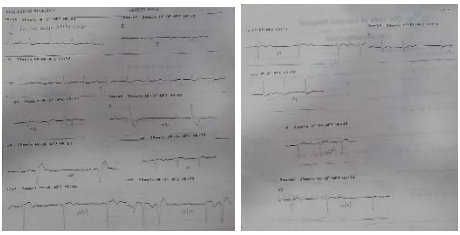

Fig 1. (ECG– Atrial fibrillation in controlled ventricular response, Occasional ventricular ectopic complexes, Non-specific ST wave changes)

On the 2nd hospital day, digitalis assay revealed a value of 2.7 ng/ml (0.4-2.4), confirming the diagnosis of digoxin toxicity. Patient underwent hemoperfusion with HA-230 cartridge for 3 hours. Patient's status was noted to be steadily improving as documented by the repeat ECG (Fig. 2). His repeat digitalis assay after 1 session of hemoperfusion was recorded at 0.5 ng/mL. Patient was subsequently discharged on the 4th hospital day and outpatient hemodialysis was continued.

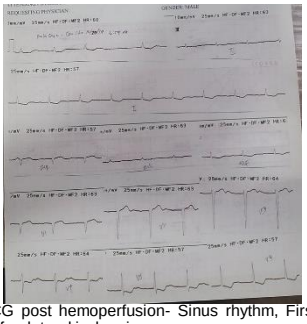

Fig 2. (ECG post hemoperfusion- Sinus rhythm, First Degree AV block t/c inferolateral ischemia

DISCUSSION: In the case above, Digoxin Toxicity was confirmed by the elevated serum digoxin assay of 2.7 ng/ml and evidenced by ECG changes and patient's symptoms (nausea, vomiting, bradycardia).

Treatment for patients with digoxin toxicity involves the use of Digitalis immune Fab, this a digoxin specific antibody and a first line drug that specifically binds to digoxin in the body preventing it from binding to its binding site, and allowing the kidney to expel it [1]. However in this patient's case we utilized hemodialysis as well as hemoperfusion using the HA-230 cartridge. The HA-230 is a hemoperfusion cartridge specific for its use on poisoning. It utilizes the adsorptive capability of neutromacroporous resin to remove toxins in the blood. [2,4]

After undergoing hemoperfusion, patient's condition notably improved documented by the ECG, digitalis assay as well as the patient's symptoms.

Shi et. al, (2012), studied the effects of hemoperfusion using the HA-230 in 85 patients with paraquat poisoning. In their study, they noted that the decline in paraquat concentration was greatest in the 1st hour of treatment than the succeeding hours and that higher level of the initial toxin equated to a better response to the treatment. With this they also recommended that frequent therapies might be more effective compared to a single prolonged session of hemoperfusion. This recommendation was echoed by a study done by Hui Dong Et al. (2017). In their study they utilized the use of standard therapy + hemodialysis + hemoperfusion in the treatment of organophosphate poisoning. Their findings showed that frequent therapy showed better cure rates, less atropine use, shorter time of recovery from coma compared to those that did one prolong hemoperfusion session.

CONCLUSIONS: This report highlights that use of HA-230 hemoperfusion cartridge in the treatment of a 67-year-old ESRD patient with digoxin toxicity. Patient underwent 1 session of hemoperfusion with HA-230 cartridge and has shown notable progress as evidenced by improvement of symptoms and normalization of ECG.

RECOMMENDATIONS: Further studies of the use of the HA-230 hemoperfusion cartridge is recommended since the utilization of this cartridge is neither widespread nor well documented in the Philippines.

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