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A Case Analysis of Drug Implications in Hypertension's Na-K-ATPase Inhibition

Mohamed Lindberg
Asst. Prof.
Barante Technological University, Singapore (BTU)

Abstract
In the case presented, Davis experiences high-value international normalized ratio (INR), positive bruising on legs and arms, nose bleeding tendencies, atrial fibrillation, and high blood pressure. Additional evidence suggests drug interactions that include digoxin with pseudoephedrine, warfarin and cimetidine. Therefore, the first action would be to control high blood pressure and proceed to reduce high-level INR. Similarly, there is a need to replace cimetidine with alternative medication. Indeed, the replacement aids in relieving a patient's heartburn. Another mechanism would be to aide Davis with the complaints regarding the cough and a runny noise.

Keywords: Drug Implications, Hypertension etc.

1 Introduction

According to Cormican and Beaman (2015), digoxin is usually prescribed for heart failure and atrial fibrillation. Notably, the procedure of monitoring serum levels is important because digoxin could become toxic to the patient's body. Specifically, levels exceeding 2ng/ml are considered toxic (Davis, 2014). For the case of Davis, a laboratory result suggesting elevated digoxin at 3.8 ng/ml is, indeed, suggestive of toxicity. Specifically, digoxin is metabolized in the patient's liver and leads to an increase in the efficacy and strength of heart contractions. As observed by Rodenburg, Visser and Hoorn et al. (2014), this increase is attributed to the inhibition of sodium-potassium ATPase that fosters an increase in calcium, translating into an increase in heart contractions; eventually excreted in the patient's urine.

2 Methodology

Cimetidine: As documented by Edmunds and Mayhew (2013), this drug is also metabolized in the liver of the patient. Specifically, the drug is an antagonist responsible for the inhibition of histamine H2's action. Warfarin: This drug leads to the inhibition of vitamin K's enzyme activity, with the latter observed to be a crucial cofactor during the synthesis of the body's blood clotting factors X, IX, VII, and II; besides two vitamin K-dependent plasma proteins S and C (Shahin & Johnson, 2016). Particularly, warfarin fosters an interference with the mediated γ-carboxylation process. Psedoephedrine: This drug acts on the patient's alpha-adrenergic receptors. According to the Truven Health Analytic (2016), the action occurs in the respiratory tract's mucosa in which vasoconstriction is produced. Indeed, the drug aids in shrinking the nasal

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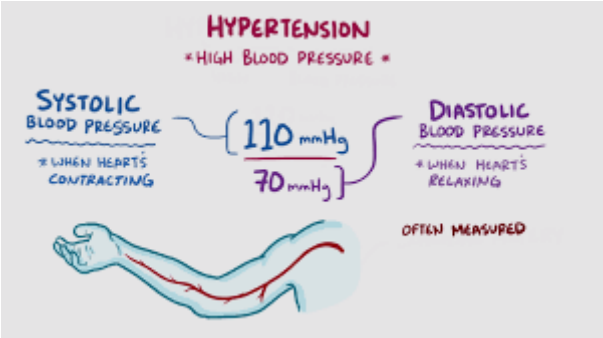
mucous membranes that are swollen; besides yielding reductions of nasal congestion, edema, and tissue hyperemia. Similarly, Weber, Schiffrin and White et al. (2014) observed that pseudoephedrine increases the patency of nasal airways. In turn, sinus secretions are drained, opening the obstructed Eustachian ostia.

3 Results and Discussion

As mentioned earlier, Davis is taking over-the-counter medication. Specifically, pseudoephedrine has been administered for purposes of relieving the cold symptoms depicted by chest/head congestion, cough, and a runny nose. Given that pseudoephedrine's mechanism of action lying in the reduction of nasal congestion occurs through the vasoconstriction of the patient's blood vessels, Agarwal and Amsterdam (2015) documented that the eventuality may be an increase in blood pressure. Other adverse effects that have been associated with the drug include atrial fibrillation and hypertension. It is also established that digoxin has been prescribed for Davis, a drug that Cormican and Beaman (2015) observed to exacerbate the blood pressure elevation effect posed by pseudoephedrine. Warfarin-Cimetidine interaction: According to Davis (2014), the drug interaction leads to a moderate severity when the medications are taken concurrently. For example, cimetidine makes the body to process blood-thinner at a slower pace, yielding an increase in nosebleeds and unusual bruising. On the other hand, Edmunds and Mayhew (2013) asserted that cimetidine leads to the inhibition of hepatic metabolism of warfarin over a period of one to two weeks. In turn, warfarin levels are increased by 20-30 percent. Upon discontinuing cimetidine, a period estimated to be one week is required before returning to pretreatment states. Thus, it is essential to monitor prothrombin on a frequent basis until manifestations are achieved regarding any responses on one's anticoagulant effect, ensuring stability at the acceptable levels (Truven Health Analytics, 2016).



Due to the blood-thinner effect, chances of nose bleeding remain high. Additional adversities of the blood-thinner effect include unusual bruising and gum bleeding. Shahin and Johnson (2016) documented that hemorrhage has also been reported in some cases to accrue from the blood-thinner effect. Notably, the removal of pseudoephedrine was not the sole cause responsible for the hypertension. Instead, it was one of the factors accounting for the elevation. Upon the medication's discontinuation, it is noted that Davis remains hypertensive, attracting the provision of supplemental medication not only to decrease the levels but also alleviate secondary risk factors for myocardial infarctions or stroke. Indeed, a full review of the illnesses presented and Davis's history reveals that a thiazide remains the best prescription or option. Whereas other medications (such as thiazide diuretics) may act in a similar manner in terms of relieving the extra fluid perceived to circulate in Davis's system (leading to a decrease in the pressure

required to move the volume), they are unlikely to work well when it comes to the removal of large volumes of fluid and treat edema (Truven Health Analytic, 2016). Therefore, a thiazide prescription is highly ideal because it stretches beyond the treatment of hypertension to aid in the reduction of peripheral vascular resistance; with hypertension decreased further (Agarwal & Amsterdam, 2015).

The medication will start with a low dose of 12.5mg of Hydrochlorothiazide per day. However, Cormican and Beaman (2015) cautioned that the medication poses the risk of digitalis toxicity, prompting the monitoring of Davis for a possible decrease in hypertension; especially regarding the degree to which the medication management is reduced the perceived baseline of 195/80. As concurred by Cormican and Beaman (2015), thiazides inhibit sodium reabsorption and work at the patient's distal convoluted tubules, increasing the excreted volume. Hence, laboratory work would be monitored during the follow-up visits to determine the potential metabolic effects on Davis's anatomy, including hyperglycemia, hypercalemia (due to increased reabsorption), and hypomagnesemia, as well as hyponatremia, and hypokalemia-hyperchloremic metabolic alkalosis – as concurred by Davis (2014).

4 Conclusion

The most likely outcome as depicted by the symptoms is that Davis is experiencing digoxin toxicity. According to the literature, symptoms associated with digoxin toxicity include severe cardiac arrhythmias, visual changes, weakness, fatigue, decreased appetite, vomiting, and nausea. Imperative to highlight is that digoxin leads to the inhibition of the Na-K-ATPase membrane pump. The eventuality is a patient's experience of increased intracellular sodium concentration. As affirmed by the results, the high intracellular sodium induces an increase in the concentration of intracellular calcium. The latter arises from the sodium-calcium exchanger. Hence, this exchanger remains highly active in smooth vascular and myocardium muscles. In turn, the rising intracellular calcium leads to elevations in the force of the heart's contraction, as well as the contracture of a patient's smooth vascular muscles. Overall, Davis's Na-K-ATPase inhibition is seen to yield a depolarizing effect.

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