



A CASE REPORT OF DIGOXIN INDUCED VENTRICULAR TACHYCARDIA

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ABSTRACT

Digoxin is extensively used in the treatment of congestive heart failure (CHF). It improves blood circulation to peripheral tissues by increasing contractility of myocardial cells by binding to Na⁺-K⁺ ATPase pump. Arrhythmias are the major Adverse Drug Reaction (ADR) of Digoxin which may lead to hospitalization, morbidity and even mortality. A 60 years male patient was admitted with breathlessness, pedal edema and was diagnosed as CHF. He was prescribed with Digoxin 5 day therapy along with other medications, on 5th day we observed Electro Cardio Gram (ECG) changes indicating ventricular tachycardia as a suspected ADR of Digoxin and was confirmed by causality assessment, immediately suspected drug was stopped, patient was monitored for progression and on 9th day ECG showed no tachycardia changes. We strongly recommends to monitor the patients receiving digoxin (especially those aged > 60years of age) for safe and effective outcome of therapy.

Key Words: Digoxin, Ventricular tachycardia, ADR, CHF, Causality assessment, ECG

INTRODUCTION

Digoxin is widely used in patients with heart failure to increase the contractility of myocardium¹. In the second half of 20th century, more effective cardiac drugs were developed and as a result the usage of digoxin has reduced, but not completely abandoned. Hence the incidence of toxicity was reduced but the number of hospitalization cases due to digoxin toxicity are yet stable². The toxicity range from understandable symptoms like nausea, vomiting, palpitations to various non-specific symptoms. It is often difficult to diagnose patients with digoxin toxicity because it is difficult to distinguish whether the signs, symptoms and ECG changes are due to digoxin toxicity or underlying disease condition^{3,4}. Digoxin improves contractility by inhibiting sodium-potassium ATPase pump in myocytes, but continuous exposure of cell to the drug can cause abnormal effects of ATPase blockade including arrhythmias most commonly ventricular premature beats, A-V block, ventricular tachycardia, atrial tachycardia etc.

Thus the exchange of intracellular sodium and calcium increases in cardiac cell. Intracellular Calcium concentration increases and increases the contractility of heart. It also has sympatholytic action in patients with heart failure. Nearly 80 to 90 % of toxicity cases found to involve some type of abnormal heart rhythm⁵. In this case report, we are presenting a case in which the manifestations of digoxin adverse effects were observed.

CASE

A 63 years old male patient was admitted in general medicine ward with complaints of severe breathlessness, pedal edema and generalized body weakness since 3 days and fever associated with emesis since 5 days and for which he took OTC medication (paracetamol). The patient was a known hypertensive since 5 years, known diabetic and was on irregular medication. He was a known smoker and alcoholic since 15 years. On general examination, patient was conscious and coherent. He was afebrile, PR was 60 beats per minute, BP was 100/70 mm of Hg, Respiratory rate 20 cycles per minute. On systemic examination CVS shows S₁S₂ and S₃+ve, CNS shows no abnormalities, pupils were reactive to light. X-ray revealed bilateral pleural effusion and cardiomegaly, Ultrasound abdomen showed ascites with congestion of liver and ECG shows sinus bradycardia. Based on the subjective and objective evaluation the patient was diagnosed with CHF. Initially the patient was admitted in emergency department and

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was administered with oral cardiac glycoside Digoxin 0.25 mg once a day for 5 days/week, oral antiplatelet drugs Ecospirin 150 mg + Clopitab 75 mg once a day, oral anti lipidemic drug Atorvastatin 20 mg HS and parenteral antibiotics (Augmentin 1 gm iv bd), anti ulcerative (Pantop 40 mg bd). Patient was advised to continue his past medications for HTN and T2DM i.e. oral ACE inhibitor Enalapril 5 mg od, Tab Glibenclamide 2 mg p/o od. ECG was performed periodically i.e. day 1, 3 and 5. The patient was showing response to the given therapy without any other complaints but on the fifth day of treatment, he had complained of palpitations, vomiting, head ache, weakness and slight visual disturbances. His pulse rate increased to 150 bpm. Then the patient was referred to ECG examination which showed bizarre and wide QRS complex, no P waves and is not followed by R wave, it is represented in **Fig. 1**. Based on above findings we suspected it as possible ADR (Ventricular Tachycardia). On analysis, compared to all other drugs prescribed, Digoxin's pharmacology and literature supports the occurrence of arrhythmias. In order to confirm the relationship between the effect and drug, we have also done dechallenge test i.e. the drug was withdrawn from the regimen. We followed the patient continuously and the 9th day i.e. after 4 days of dechallenge (because half-life of digoxin is around 36 to 48 hours) we have repeated the ECG again to check the improvement which showed no QRS prolongation **Fig. 2**. On the same day the following findings were observed: PR 75 bpm, BP 130/70 mm of Hg.

ADR ANALYSIS

Causality assessment:

To evaluate the relationship between the drug and reaction, we have performed causality assessment by using scales like WHO causality assessment scale, Naranjo's scale and Karch and Lasagna scale (**Table- 1**). We further analysed to find the severity, preventability and predictability of ADR and were represented in **Table- 2**.

DISCUSSION

The normal function of heart is to pump sufficient amount of blood to various tissues in our body. CHF is a condition in which there is inability or inefficient contraction of heart. This leads to decreased blood supply to various tissues. The main aim for the usage of digoxin in CHF is to increase the contractility of heart for maintaining sufficient amount of blood supply to various tissues. Digoxin acts by binding to Na^+-K^+ ATPase pump and increase the sodium concentration inside the cardiac cell followed by calcium ions. This calcium increases the myocardial contractility. Digoxin is a narrow therapeutic drug. Its normal therapeutic range is 1 to 2 ng/ml⁶. If slight increase in plasma concentration, causes adverse effects. Digoxin can possibly cause any type of abnormal heart rhythm⁷. Digoxin is concentrated in the heart, kidneys, liver and skeletal muscles. It is mainly excreted through urine in unchanged form. The risk factors which further aggravates the digoxin effects are Age (due to renal and hepatic function), Route (IV), Hypokalaemia (binding of digoxin to Na^+-K^+ ATPase increases), Hypercalcaemia, Hypomagnesaemia, Hypothyroidism, Hyperthyroidism, Hypoxia, Renal failure and Myocarditis. In aged persons decreased renal and hepatic function will be observed. This might increase the concentration of digoxin in plasma which leads to adverse effects. This may be happened in our case. Hence dosage adjustment and therapeutic drug monitoring (TDM) is essential for patients with digoxin therapy.

CONCLUSION

Even though digoxin remains to be a common drug used by patients presenting to the CHF, the diagnosis of toxicity is difficult to establish because of nonspecific signs and symptoms. It is essential to identify the ADR and establish a causal relationship between the drug and adverse effects among the patients receiving digoxin therapy to prevent morbidity, hospital admissions and even mortality. Co administration of potassium supplements along with Digoxin will reduces the incidence of ADRs.

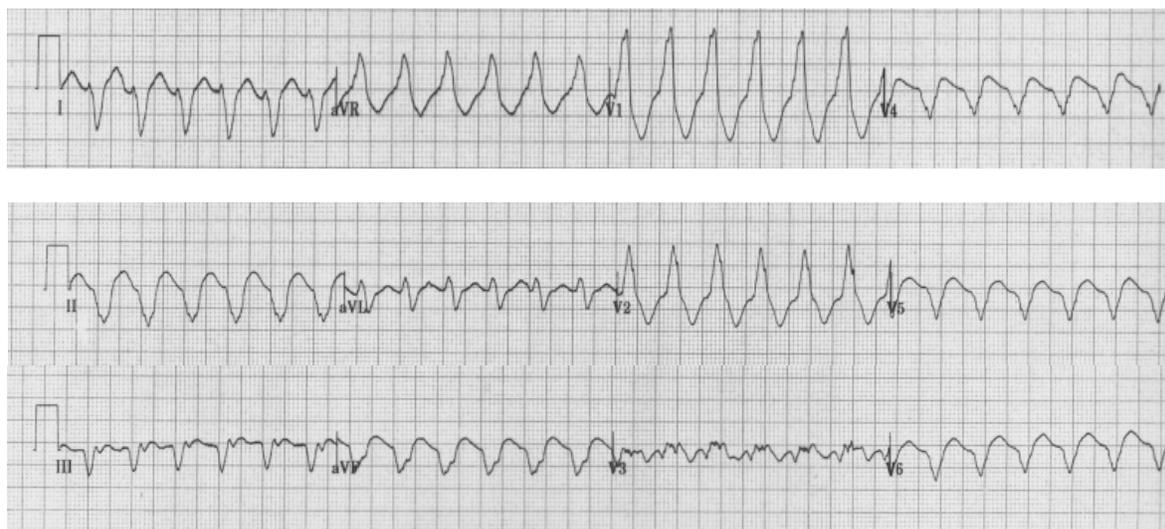


Figure – 1: ECG Report of Day 5



Figure -2: ECG Report of Day 9

ADR SCALE	WHO-UMC	NARANJO'S	KARCH&LASAGNA
ASSESSMENT	Probable	Probable	Probable

Table-1: Causality Assessment of Suspected ADR

SEVERITY ASSESSMENT	Moderate Level 4 (a)
PREVENTABILITY	Probably Preventable
PREDICTABILITY	Predictable (TYPE - A)

Table-2: Analysis of Suspected ADR

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