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BMH Medical Journal 2016;3(4):100-103 **Case Report**

Digoxin Dilemma

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Abstract

Digoxin is a very commonly used drug in patients with systolic heart failure for enhancing cardiac contractility and in patients with supraventricular tachyarrhythmias for ventricular rate control. Due to its varied presentation and non specific symptoms, clinical suspicion should be high in order to diagnose a case of digoxin toxicity.

Chronic toxicity is more common among the elderly and renal failure patients. The definitive treatment for severe digoxin toxicity is Fab fragments of digoxin antibody and there are specific indications for the same. However, due to its high cost and limited availability alternative measures may need to be used to manage severe intoxications.

We present the case of an elderly patient with multiple co morbiditiess who presented to us in acute pulmonary edema and complete heart block. Serial ECGs reverted showing digoxin effect after symptomatic treatment and withdrawal of digoxin. In this case report, we discuss the various effects of digoxin and also the symptoms and signs of digoxin toxicity along with indications to use digoxin antibodies.

Key Words: digoxin toxicity; Fab fragments, digoxin antibody

Background

Digitalis toxicity has considerably decreased over the past few decades. The Australian Institute of Health and Welfare reported cardiac glycoside toxicity in 280, 233 and 139 patients in 1993-94, 2003-04 and 2011-12 respectively. This clearly documents a declining trend over the past few decades. Chronic digoxin toxicity is commoner than acute intoxication [1]. Even though the prevalence of digitoxicity is declining, it is to be considered in all patients on digoxin. Diagnosis is often challenging because we need a high index of suspicion and symptoms of toxicity are often vague and varied.

In a study conducted in 10 urban and rural Department of Veterans Affairs Medical Centers in the Rocky Mountain region during 1989 to 1990, of the 183 patients on digoxin, 50 (27.3%) had one or more risk factors for digoxin toxicity [2]. Serum digoxin levels were elevated in 13.6% of patients in whom a level was obtained. Hypokalemia was noted in 14.3%, elevated creatinine levels in 17.9%, and possible drug interactions in 5.5% of patients. Yet, digoxin toxicity occurred in only 2 cases.

Although suspicion of digoxin toxicity might be present, the lack of availability of prompt drug levels especially at late hours makes the diagnosis of digoxin toxicity in the emergency department (ED) challenging. Chronic digoxin toxicity varies in severity and is associated with a mortality at one week of 15-30%. Thus diagnosis is crucial.

Case Presentation

67 year old female, a known case of rheumatic heart disease with severe mitral stenosis and pulmonary hypertension was brought to ED with complaints of breathing difficulty since 5 days. On examination she had mild respiratory distress, heart rate was 40/min, blood pressure 110/50 mm Hg and oxygen saturation 97% on room air. Bilateral crepitations and a mid diastolic murmur in the mitral area were audible.

The initial ECG (**Figure 1**) showed ventricular rate of about 45/min and atrial rate of around 160/min. There was right axis deviation and mild inferolateral ST depression. Her serum potassium and renal parameters within normal limits. This ECG is suggestive of atrial tachycardia with varying AV block, one of the characteristic arrhythmias of digoxin toxicity.

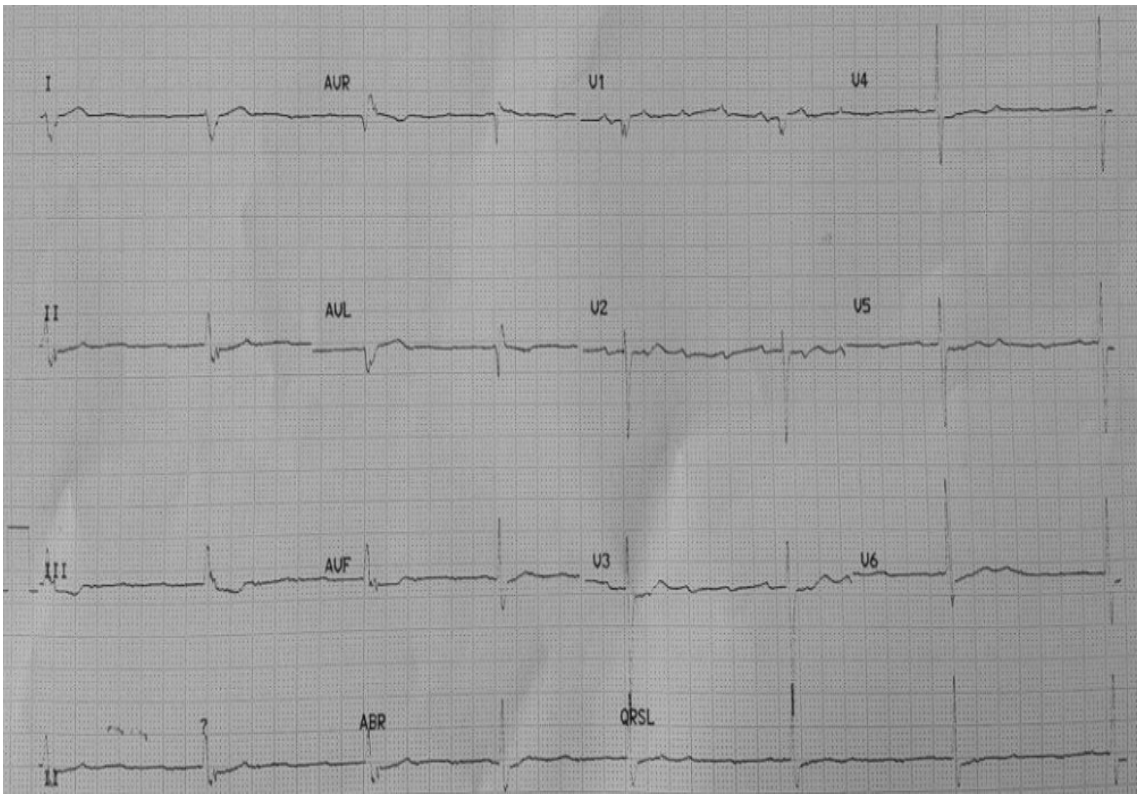

Figure 1: Initial ECG showing atrial tachycardia at a rate of 160/min and a ventricular rate of 45/min

Digoxin was with held and she was managed in the intensive care unit with continuous rhythm monitoring and intravenous diuretics for her early pulmonary edema. ECG on the next day (**Figure 2**) showed sinus rhythm at around 75/min, with first degree AV block and lateral ST segment depression mimicking the *mirror image correction mark*, indicating digoxin effect.

Discussion

Digitalis is most frequently prescribed for its positive inotropic effect in patients with congestive heart failure and for slowing the ventricular rate in patients with atrial fibrillation or flutter. The most frequently used cardiac glycoside is digoxin. Digoxin has both direct and indirect effects. The direct effect of digoxin is the inhibition of Na/K ATPase on the cell surface which leads to an increase in

the intracellular sodium and extracellular potassium. This in turn leads to increase in the intracellular calcium which mediates an increase in inotropy. The indirect effect of digoxin is an increase in the vagal tone. Digoxin excretion is predominantly through the kidneys. Hence the chance of digoxin toxicity is higher in renal failure.

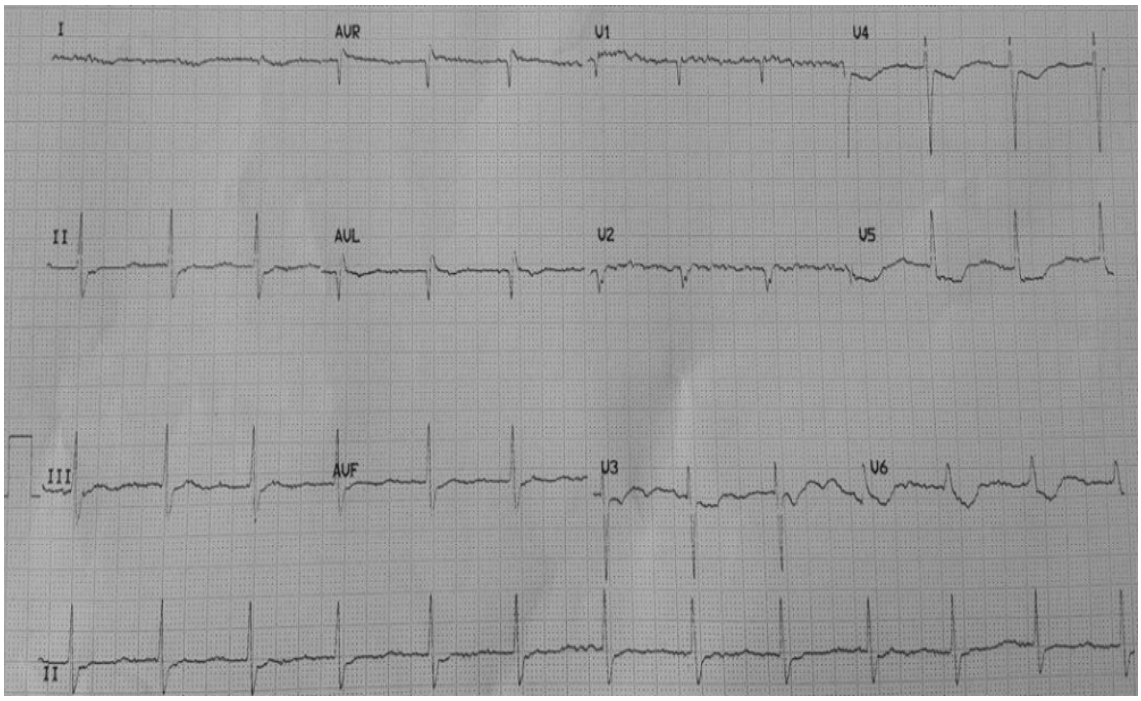

Figure 2: ECG taken on the day after admission showing *mirror image correction mark* type of ST segment depression in lateral leads

Digoxin effect on the ECG refers to the presence of downsloping ST depression with inverted T waves (*mirror image correction mark pattern*). This pattern is seen in leads with dominant R waves. If it is seen in leads without a dominant R wave, it may indicate digoxin toxicity rather than digoxin effect. Digoxin shortens the QT interval. Digoxin prolongs the PR interval due to its indirect effect on the AV node by increased vagal tone. One of the commonest ECG manifestation of digitoxicity is the presence of ventricular ectopic beats, usually occurring in a bigeminal pattern. Characteristic arrhythmia of digoxin toxicity is bidirectional ventricular tachycardia. Another classical arrrhythmia of digoxin toxicity is a combination of atrial tachycardia due to increased automaticity and a slow ventricular response due to decreased AV conduction, known as atrial tachycardia with block. This is well illustrated in our case (**Figure 1**).

Common symptoms of digoxin toxicity are gastrointestinal - anorexia, nausea and vomiting. Characteristic visual disturbance in digitoxicity is yellow vision or xanthopsia. They can also present with nervous system manifestations like confusion, dizziness and delirium.

Management of digoxin toxicity starts with stopping digoxin and observing for potentially life threatening arrhythmias. Renal function and electrolyte assessment is very important in the management. Hypokalemia can worsen arrhythmias of digoxicity and needs correction. But caution need to be exercised to prevent over correction as hyperkalemia worsens AV conduction abnormalities due to digoxin. Fab fragments of digoxin antibodies (Digibind) can be considered as standard treatment in the presence of life threatening arrhythmias, though availability is limited. Digoxin level assessment may be artefactually high for weeks after administration of antibody. Severe hyperkalemia due to digoxin toxicity, high digoxin levels in blood and history of ingestion of high amounts of digoxin are considered indications for treatment with antibody, though clinical trial based data are lacking.

As the use of digoxin, especially at higher doses have declined over the past few decades, occurrence of digitxocity is also rare. Hence one should be quite vigilant to identify an occasional patient presenting with digitoxicity to the ED.

References

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