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Digoxin level as a limited drug toxicity indicator in the presence of electrolyte imbalance.

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Abstract

An 86 year old lady with background of small cell lung cancer was found to have a trial fibrillation (AF) with a fast heart rate while being in the hospital. She was loaded with digoxin for rate control. Though her heart rate settled yet she developed early signs of digoxin toxicity just with the loading dose. Only abnormal result among her bloods was low potassium(K) with normal digoxin levels. She recovered completely on correction of hypokalemia.

Introduction

Digoxin is commonly used in clinical practice for rate control in atrial fibrillation(AF)[1]. The electrolyte and renal status of each patient including weight should be ascertained prior to initiating treatment and not only during treatment. Hypokalemia or hypomagnesemia, for example, may promote the development of digoxin induced arrhythmias, while impaired renal function may result in higher than anticipated serum drug levels[2] .Digoxin related cardiac arrhythmias and extra-cardiac symptoms can occur when the serum digoxin concentration is in the therapeutic or even sub therapeutic range. Clinicians should be aware that presence of digoxin toxicity or excess is often a **clinical diagnosis** irrespective of circulating levels.

Case Presentation

We present the case of an 86 year old lady who was admitted in a geriatric ward for newly diagnosed small cell cancer of the lung(SCC) in its advanced stage. She had a background history of congestive cardiac failure & asthma and was currently awaiting her discharged destination to be sorted. One morning she was found to be tachycardic by the nursing staff when she complained of shortness of breath(SOB) and palpitations.ECG showed atrial fibrillation (AF)with a heart rate between 140-150 *(figure2).* No obvious precipitating cause was found from history examination and subsequent investigations which excluded pneumonia ,pulmonary embolism and ischemic event as possible differentials. Keeping in view her background medical history of asthma, congestive cardiac failure and low systolic blood pressure 95 mmhg ,rate control with digoxin was considered to be an appropriate strategy at that point of time by on-call team .Having an average built & almost normal renal functions she was loaded with intravenous digoxin 500mcg .However within an hour she became nauseous and started vomiting .Her routine observations were stable and her systemic examinations was unremarkable. A repeat ECG showed resolution of tachycardia but QRS complexes and ST segments showed the appearance of an ischemic event with prolonged PR interval *(figure 3).* She never complained of any chest pain and subsequent cardiac biomarkers excluded an acute coronary syndrome. Her routine Bloods came back to be normal along with therapeutic digoxin levels except for hypokalemia (potassium =3.2mmol/l).She recovered spontaneously when her potassium was corrected (4.9mmol/l)and abnormal ECG changes also disappeared. *(figure 4,5)*

Investigations

Pottasium (K)3.2mmol/L & after correction 4.9 mmol/L,Calcium (Ca)2.51mmol/L,Urea 5.2 mmol/L ,Creatinin (Cr)98mmol/L. Magnisium(Mg) 0.76 mmol/L ,Troponin T(0.06 ug/L and 0.05ug/L at 12 hours) ,Arterial Blood gas ABG on air showing a pH of 7.46 ,paO2 11kpa,ECHO showed ejection fraction of 38% with moderate left ventricular systolic dysfunction .Right heart systolic functions was mildly impaired similar to previous ECHO with no evidence of raised right sided pressure & no regional wall motion abnormality.

Treatment

Potassium was replaced with both intravenous(40mmol in normal saline(0.9%) 1L over 12 hours as she was clinically dry at that point and was vomiting) with strict fluid balance & oral supplements (sando-k " 0.6g potassium chloride., 0.4g potassium bicarbonate" 2 tab three times a day) once vomiting settled. She was also worked up for acute coronary syndrome(ACS).Though at high risk for

pulmonary embolism (PE) clinical suspicion of having a PE was low due to history ,normal oxygen saturations (96% on room air) with subsequent normal ABG(arterial blood gas) & also the fact that patient was on prophylactic low molecular weight heparin(LMWH).Subsequent echocardiogram further supported the above impression.

Outcome and Followup

Patient improved symptomatically and biochemically following above management within a period of approximately 5-10 hours. She stayed in hospital for a couple of weeks but unfortunately died later due to her progressive terminal stage cancer.

Discussion

Atrial fibrillation(AF) is the most common arrhythmia encountered in elderly population[3] . Most patients who present with AF will require slowing of the ventricular rate to improve symptoms. Beta Blockers (B-Blockers) are considered first line treatment for rate control unless contraindicated. However In patients with heart failure due to systolic dysfunction and B-blockers intolerance digoxin is the preferred drug[4]. For maximal early benefits, digoxin requires loading doses, which can be administered intravenously or orally. Patients receiving digoxin for rate control in atrial fibrillation will usually require more rapid loading and higher maintenance doses than those treated with digoxin for heart failure .The total loading dose of digoxin varies from patient to patient but is usually between 0.75 to 1.5 mg with intravenous administration and 1 to 1.5 mg with oral administration in divided doses. Patients who are underweight ,have electrolyte imbalance or renal impairment are more sensitive to the effects of digoxin. For such patients an initial loading dose in the lower range (eg 0.75 mg or less) should be considered[5]. Life-threatening digoxin-induced arrhythmias and other toxic manifestations occur at substantially increasing frequency as the plasma digoxin concentration rises above reference range. However, clinicians should be aware that signs of toxicity may occur even at therapeutic levels in the presence of one or more of the predisposing factors noted above. Similar results were published in in a study in Belgium which emphasised the inadequacy of serum digoxin levels being an independent marker for digoxin toxicity specially in presence of hypokalemia[6].Another study published in'' American journal of cardiology'' showed resolution of digoxin induced arrhythmias after correction serum potassium levels though digoxin levels remained the same[7] .Though mentioned in literature quite frequently ,digoxin toxicity in presence of normal digoxin levels is still underreported leading to increase dependence on digoxin levels as a sole indicator of toxicity[8].

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Illustrations

Illustration 1

Figure 1: Baseline ECG

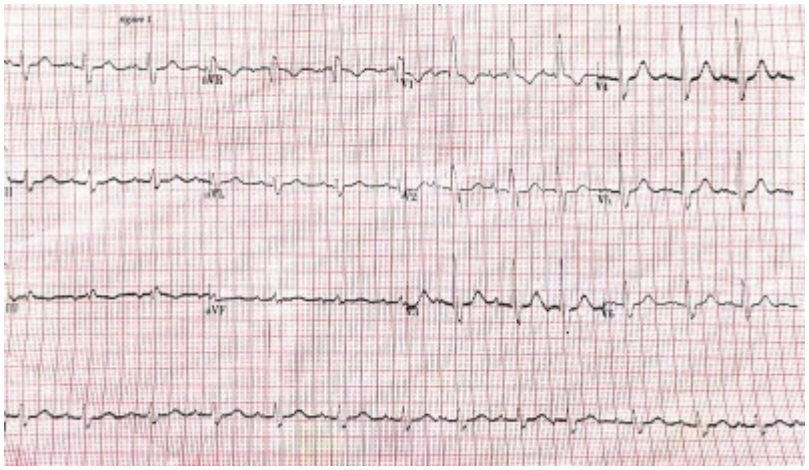

Illustration 2

Figure 2: Development of fast atrial fibrillation(AF)

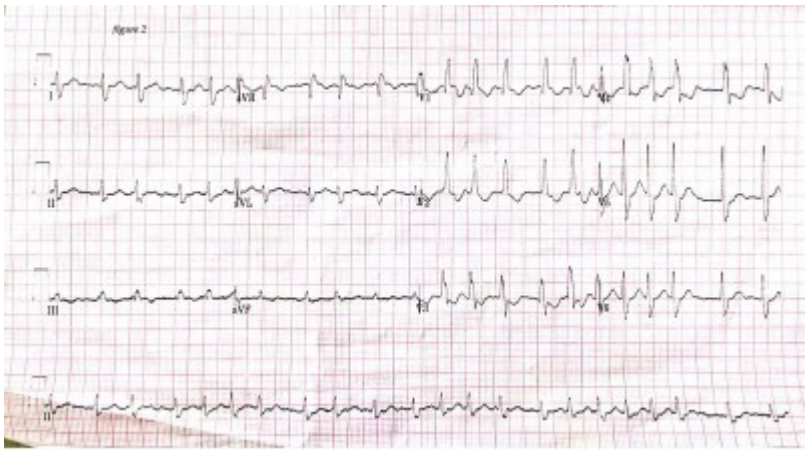

Illustration 3

Figure 3: Initial effects of digoxin within couple of hours

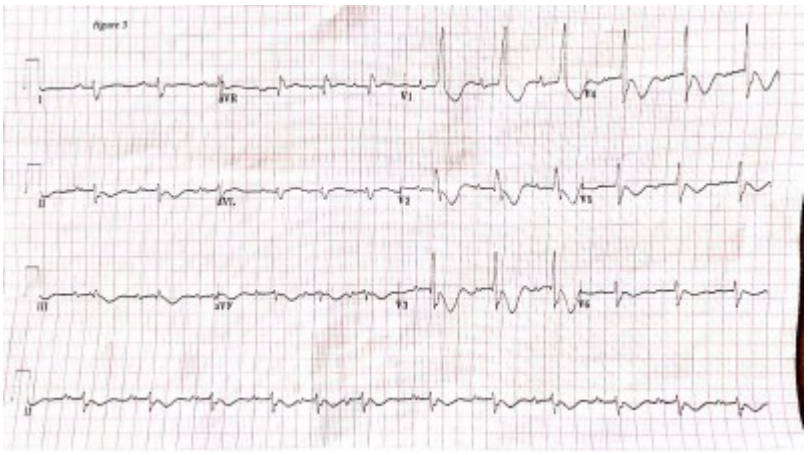

Illustration 4

Figure 4: After correction of electrolytes

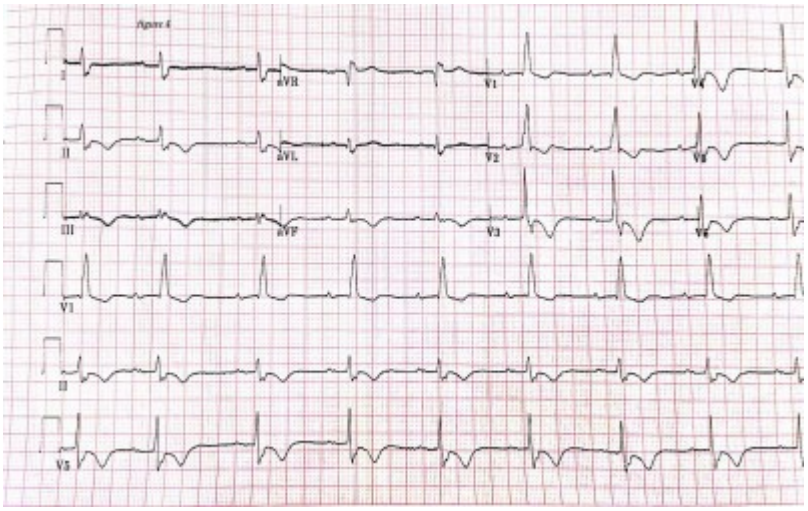

Illustration 5

Figure 5: After correction of electrolytes