

ECG Quiz

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A cardiac consultation about a 76-year-old lady with chest pain and congestive heart failure was received. The cardiologist was asked to comment on an "abnormal ECG" (Figure 1) which was done 10 days after admission.

What was the rhythm diagnosis ?

1. a) Sinus rhythm
- b) Atrial fibrillation
- c) Accelerated junctional rhythm
- d) Others

The busy cardiologist reviewed the history and all ECG's. The patient immigrated from China two

months ago. She had history of heart disease and chronic lung disease. She has been given frusemide and some unknown medicine. The patient presented with exertional shortness of breath and atypical chest discomfort. Clinical examination revealed a 39 kg lady with tachycardia and congestive heart failure. The potassium level and renal function was within normal on admission. The following was the ECG on admission. (Figure 2)

What was the ECG diagnosis ?

2. a) Atrial fibrillation
- b) Atrial flutter with variable block
- c) Atrial tachycardia with variable block

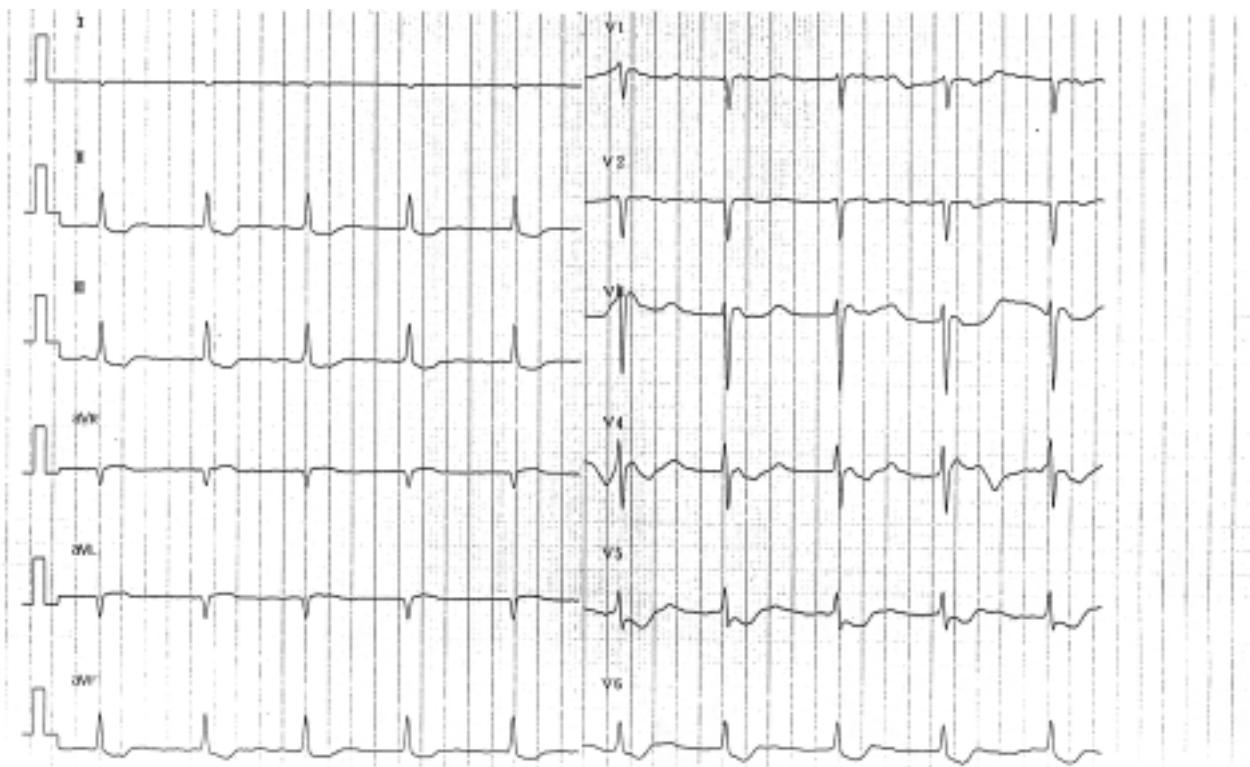


Figure 1. "Abnormal ECG" 10 days after admission.

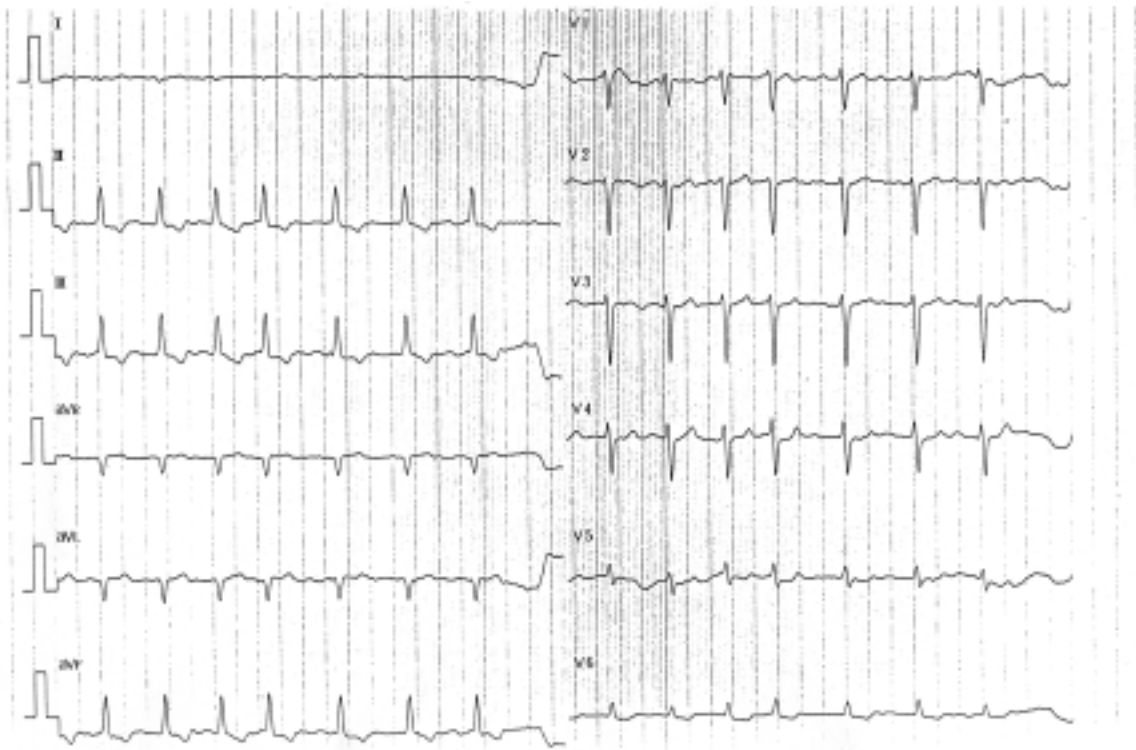


Figure 2. ECG on admission.

The patient was given aspirin, nitrate and frusemide. Digoxin loading of 0.25 mg Q8H for four doses followed by 0.125 mg per day was given orally.

The patient developed nausea, vomiting, confusion and easy forgetfulness 10 days afterwards. Clinically she was dehydrated. Serum electrolytes and blood glucose were normal. Renal function test was impaired with urea 13.1 mmol/L and creatinine 201 μ mol/L. CT brain was normal. ECG was done at that time and was the one mentioned in the cardiac consultation. (Figure 1)

3. What was the likely unifying cause for the symptoms and the "abnormal ECG" 10 days after admission?

The digoxin level was 5.6 nmol/L. Digoxin and frusemide were stopped. The patient was cautiously rehydrated. The symptoms gradually subsided over 2 weeks' time. The renal function test was then normalised and the digoxin level dropped to 2.0 nmol/L. The cardiac rhythm reverted back to that of admission.

Answers

1. d) Accelerated junctional rhythm with underlying atrial fibrillation
2. a) Atrial fibrillation
3. Digoxin toxicity

Discussion

Digoxin toxicity is not an uncommon clinical problem partly because of its widespread use. Timely recognition and appropriate management requires knowledge on the risk factors predisposing to and clinical manifestations of digoxin toxicity.

Factors predisposing to digoxin toxicity include hypokalaemia, hypomagnesaemia, hypercalcaemia, renal impairment, advanced age, pulmonary disease and thyroid disease. In this thin elderly lady with history of chronic lung disease, a lower maintenance dose of digoxin should probably be given. The subsequent renal function

impairment which contributed to digoxin toxicity was likely to be due to overdiuresis with frusemide.

This patient demonstrated the classical constellation of gastrointestinal and neurologic symptoms of digoxin toxicity. Dementia like symptoms should alert one to look out for chronic digoxin overdose. Noncardiac manifestations of digoxin toxicity include anorexia, nausea, vomiting, headache, malaise, neurologic pain, disorientation, alteration in color perception, scotoma and halo vision.

Digoxin toxicity produces two major electrophysiologic effects. The first one is a direct and/or vagally mediated slowing of conduction and block in the sinus node and atrioventricular node. This may cause sinus rate slowing, sinus pauses and atrioventricular conduction disturbances. The second one is an enhanced abnormal automaticity and triggered activity in atrial muscle, atrioventricular junction, His-Purkinje system and ventricular muscle. Specific arrhythmias of digoxin toxicity include (1) paroxysmal

atrial tachycardia with variable block, (2) atrial fibrillation with complete heart block, (3) second or third-degree AV block, (4) supraventricular tachycardia with alternating bundle branch block, (5) complete heart block with accelerated junctional rhythm or accelerated idioventricular rhythm, (6) fascicular ventricular tachycardia. In this patient, the ECG shown in Figure 1 demonstrated a very specific arrhythmia due to digoxin toxicity. Regularisation of RR interval with underlying atrial fibrillation shown in leads II, III, V1 and V2 often represents complete heart block with accelerated junctional rhythm (with ventricular rhythm >60 bpm).

Apart from arrhythmias, digoxin therapy may also cause alterations in ST segment and T wave. The T wave amplitude is lowered and ST segment is depressed and shortened with occasional appearance of a prominent u wave. The characteristic ST segment sagging is also found in this patient. However, it is always difficult to differentiate it from ST depression of other causes like myocardial ischaemia.