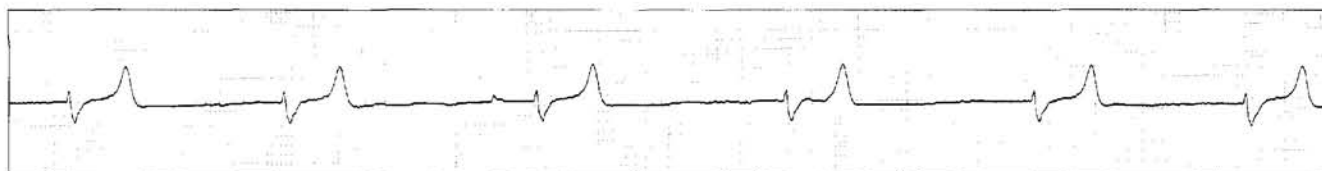


A Lady With Syncope

Clinical History:

This 75-year-old lady presented with syncope and the following ECG was obtained (**Figure 1**).

Figure 1



Question 1: What was the underlying cause for the ECG abnormalities?

- A. Hypokalaemia
- B. Digoxin overdose
- C. Acute myocardial infarction
- D. Hyperkalaemia

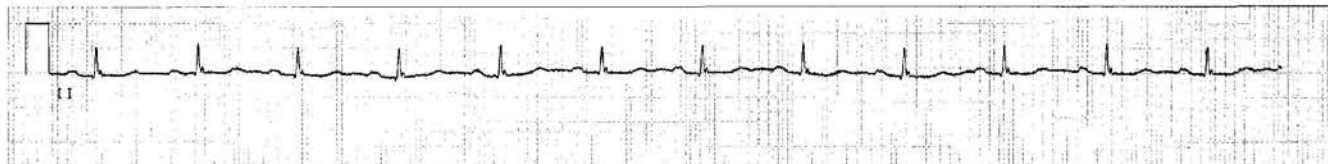
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(Continued on page 342)

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ECG ROUND

Figure 2



Answer : D. Hyperkalaemia

The ECG in **Figure 1** shows a ventricular rate of about 30/min without obvious P waves. Thus the patient had a junctional escape rhythm. The QRS duration was prolonged to 160 ms (normally 120 ms). The T waves were tall and peaked. These ECG abnormalities are typical of hyperkalaemia. In fact, the earliest sign of potassium intoxication is the appearance of tall and peaked T waves. Later the PR interval becomes prolonged and the QRS duration lengthens. Finally the P waves disappear and the QRS widens further and ventricular fibrillation may occur.¹ P waves are not inscribed on the ECG because atrial depolarization is unable to occur in the presence of severe hyperkalaemia.²

In this patient, the serum potassium level at that time was 7.2 mmol/l (normal range in our laboratory: 3.2 – 5.2 mmol/l). She was also found to have renal impairment.

Common causes of hyperkalaemia encountered by family physicians include renal impairment, potassium-sparing diuretics, and angiotensin-converting enzyme inhibitors, especially if patients are on potassium supplement as well.

Question 2: Figure 2 was the ECG of the same patient one day later. What treatment did the patient receive?

- A. Calcium gluconate
- B. Glucose-insulin infusion
- C. Cation-exchange resin
- D. All of the above

Answer: D. All of the above

The approach to treat hyperkalaemia depends on the degree of hyperkalaemia as determined by the plasma potassium concentration and changes on the ECG. Severe hyperkalaemia requires emergency treatment directed at minimizing membrane depolarization, shifting potassium into cells and promoting potassium loss.³ This patient had high serum potassium level (7.2 mmol/l) and associated ECG abnormalities. She was given intravenous calcium gluconate to decrease the cell membrane excitability. Insulin infusion (with glucose) was also administered to cause shifting of potassium into cells, thus lowering the plasma potassium level. Oral cation-exchange resin was also given to this lady to enhance removal of potassium. Because of the severe bradycardia, temporary cardiac pacing was needed for the first few hours while pending the effect of potassium lowering therapy.

The ECG in **Figure 2** shows sinus rhythm (rate around 75/min), with normal QRS complexes. The previous tall peaked T waves has now disappeared. This ECG was obtained one day after admission when the serum potassium level was 5.3 mmol/l. Her condition remained stable and was subsequently discharged home. ■

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