Tachycardia following low-tension electrocution

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A 33-year-old woman sustained an electric shock to the dorsum of her right hand from a domestic power supply. The only symptom she reported was a transient numbness in her right arm. There was no loss of consciousness. On arrival at the accident and emergency department she was well perfused and alert. Examination of her skin revealed no superficial burns. She had a regular tachycardia at a rate of 150 beats/min, and her blood pressure was 120/70 mmHg. The remainder of the examination was normal. Her admission electrocardiogram (ECG) is shown in figure 1. The patient was given 3 mg and then 6 mg of adenosine intravenously without effect: the outcome of 12 mg of adenosine is shown in figure 2.

Further investigations included serial creatinine kinase (myocardial bound fraction) and troponin I levels to exclude myocardial damage. These were within normal limits. The lung fields were clear on chest X-ray. An echocardiogram showed normal ventricular and atrial dimensions and function and no valvular abnormality.

Figure 1 Admission ECG

Figure 2 Rhythm strip during intravenous administration of 12 mg of adenosine

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Questions

- 1 Describe the features shown in the ECG (figure 1).
- 2 What does the rhythm strip in figure 2 show, and what information does this give us about the mechanism of the tachycardia?

Answers

QUESTION 1

The ECG shows a narrow complex tachycardia with retrograde P wave activity. The P wave axis indicates that is a junctional tachycardia.

QUESTION 2

The rhythm strip shows normal P wave activity followed by a few sinus beats before the junctional rhythm returns. As the tachycardia was temporarily abolished by adenosine, it must involve the AV node.

Outcome

The following day, as the patient remained in the junctional tachycardia, she was given 25 mg of atenolol which led to a restoration of sinus rhythm and she was discharged home.

Discussion

Electrocution can be subdivided into low-tension shocks and high-tension shocks. It is well recognised that electrical injury may cause immediate cardiac arrhythmias. High-tension shocks (>1000 Volts) from lightning strikes and overhead power cables cause severe skin burns and may cause cardiac arrest usually due to asystole.¹ High-voltage injuries lead to a high incidence of cardiac abnormalities, up to 30% of those with cardiac arrhythmias on presentation have long-term reduction in cardiac function measured by echocardiography.² Changes in cardiac conduction including complete heart block, and prolongation of the QT interval suggest that electric shocks can significantly modify intracardiac conduction.³

Low-tension shocks which occur from domestic appliances (<300 Volts at 50Hz) can cause ventricular fibrillation,¹ but the incidence of cardiac arrhythmias following low-tension electrocution is not known. One retrospective study demonstrated that those patients with a normal ECG on presentation did not subsequently develop a serious arrhythmia.⁴ However, there is an appreciable risk of late ventricular fibrillation in injuries involving the passage of current through the thorax. In these cases, myocardial biopsies have shown patchy myocyte necrosis and fibrosis.⁵

The likelihood of tissue injury and cardiac arrhythmias following electrocution is depend-

Summary points

- electrocution can be divided into high-tension (>1000 V) or low-tension injury (<350 V)
- there is a high incidence of cardiac damage with high-tension injuries
- low-tension injuries may cause ventricular fibrillation
- patients with high-tension injuries or low-tension injuries with an abnormal ECG or passage of current through the thorax should be admitted to hospital for cardiac monitoring

ent on the size of the current flow. For example, an alternating current of approximately 70 mA is required to induce ventricular fibrillation. Current flow is predominantly influenced by the voltage of the shock and resistance of the tissues involved. These principles are expressed by Ohms law:

$$\text{Current} = \frac{\text{Voltage}}{\text{Resistance}}$$

Bone has the highest resistance of the internal structures of the body, followed by fat. As electrical wounds enter through the skin, the resistance of the skin at the time of the injury contributes significantly to the total body resistance. Therefore, an electrical shock on wet skin has potential to cause more serious damage to tissues.^{6,7}

Patients should be admitted to hospital and monitored in all cases of high-tension electrocution because of the high incidence of myocardial damage. In low-tension injuries, admission and monitoring of cardiac rhythm is necessary if the admission ECG is abnormal or if there is any suggestion that the passage of electrical current was through the thorax. This may be inferred from any superficial burns at the entry and exit site or from the clinical history. In our patient, the initial ECG precipitated admission for further monitoring.

Final diagnosis

Junctional tachycardia following low-tension electrocution.

Keywords: electrocution; tachycardia

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Collapse and hypothermia in an elderly woman in early summer

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A 74-year-old woman was admitted during a warm spell, having been found at home collapsed. She was conscious but confused (Abbreviated Mental Test score (AMTS) 4/10). Her sister related her medical history which included an underactive thyroid diagnosed 8 years before, an operation to close an atrial septal defect as a young woman, a small stroke and several admissions over the last 3–4 years with difficulty coping and confusion. In fact, she had been told by one doctor that her sister had dementia. The patient was taking digoxin 62.5 µg, co-amilofruse 2 tablets, and thyroxine 50 µg, all once daily. On examination she was pale and drowsy and her rectal temperature was 33.6°C. Body hair was absent. The pulse rate was 60 beats/min, irregularly irregular, and her blood pressure was 120/60 mmHg. Although the heart was clinically enlarged, auscultation was normal. There were fine crackles audible at the lung bases. Apart from the cognitive impairment, central nervous system examination was normal.

Initial investigations showed haemoglobin 10.4 g/dl (mean cell volume 88 fl), white cell count $3.9 \times 10^9/l$, platelets $88 \times 10^9/l$, urea, electrolytes and thyroid function tests were normal. Plasma glucose was 1.4 mmol/l. An electrocardiogram revealed slow atrial fibrillation with a prolonged QT interval and right axis deviation. A chest X-ray confirmed cardiomegaly. The patient was given 50 ml of intravenous 50% dextrose with some improvement in her confusion and she was slowly re-warmed. She was then able to eat and drink. The following day her serum sodium was 123 mmol/l and blood glucose 2.0 mmol/l. Her old notes at this time revealed that during the admissions with confusion and not coping over recent years, her sodium had often, and her glucose had occasionally, been low. The notes also confirmed that she had had primary hypothyroidism diagnosed 8 years before. Computed tomography (CT) was performed and, following injection of contrast medium, a coronal image through the pituitary fossa obtained (figure).

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Figure Coronal CT scan showing view through pituitary fossa

Questions

- 1 What further investigations would confirm the diagnosis and what is the significance of the CT findings?
- 2 Why did she present with hypothermia?
- 3 What is the most probable mechanism causing the hypoglycaemia?
- 4 What is the cause of the hyponatraemia?