An unconscious patient with an electrocardiogram mimicking an acute myocardial infarct

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A 72-year-old woman was admitted to the emergency room for sudden loss of consciousness preceded by headache without tonic–clonic movements, urine loss, or tongue bite. There was no history of trauma. Her medical history was unrevealing: she had presented with bronchopneumonia in 1953, and she had been operated for a strangled hernia in 1968 and for an ovarian cyst in 1983. She had no cardiovascular risk factors and took no medications.

On admission, blood pressure was 110/85 mmHg, pulse was equal and regular at 80 beats/min. She had no fever and there was no sign of shock. Heart sounds were normal and no vascular bruit was detected. Lungs were clear. She was unconscious and showed extension (decerebrate) posturing to painful stimuli. The rest of the neurologic examination was normal. Chest X-ray was unremarkable. Haemogram, renal function, electrolytes, cardiac enzymes, liver function tests and arterial blood gases were in the normal range. Glucose was 7.85 mmol/l (1.42 g/l). Ophthalmologic examination revealed normal papillae. Her admission electrocardiogram (ECG) showed a regular sinus rhythm, a normal atrioventricular conduction of 160 ms, a Q wave and a ST segment elevation from V_2 to V_6 (figure 1).

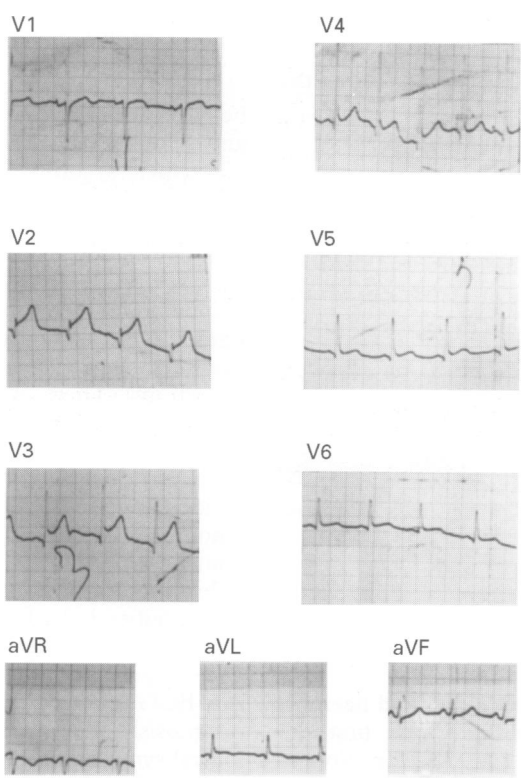

Figure 1 Admission ECG showing an acute myocardial infarction pattern

Questions

1 What investigations should be done?
2 What is the most likely diagnosis?
3 What treatment should be proposed?

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Answers

QUESTION 1
Computed tomography (CT) scan of the brain demonstrated a left subarachnoid haemorrhage.

QUESTION 2
ECG abnormalities associated with subarachnoid haemorrhage.

QUESTION 3
No cardiac treatment is warranted if there is no life-threatening arrhythmia.

Outcome

The patient was admitted to the intensive care unit and recovered consciousness in 24 hours with a normal neurological examination despite retrograde amnesia. No rise in cardiac enzymes was noted during the first 48 h (measured every 4 h). The ECG performed the day after admission showed a T-wave inversion in the anterior leads (figure 2). Carotid angiography revealed five aneurysms (right and left middle cerebral arteries, right anterior and left posterior communicating arteries and a right pericallosal artery). Myocardial perfusion scintigraphy at rest was normal. A few days later, the patient was successfully operated on and the anterior communicating artery aneurysm was clipped. An ECG performed 2 months later was normal.

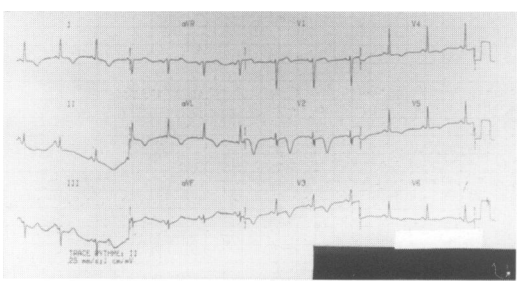

Figure 2 ECG performed a day after admission showing a T- wave inversion in the anterior leads

Discussion

An association between subarachnoid haemorrhage and ECG anormalities was first described by Byer *et al* in 1947.[1] QT prolongation, and ST and T modifications are the most prevalent abnormalities. Supraventricular and ventricular arrhythmias, although less frequent, are not exceptional. Some of the ECG abnormalities noted in such conditions may closely mimic those observed in myocardial infarction. Their frequency, in association with subarachnoid haemorrhage, varies from 0.8% to 10%.[2] Many attribute this ECG pattern to an autonomic imbalance that would result from ischaemia or irritation of the hypothalamic area by blood present in subarachnoid space, leading to an excessive sympathetic stimulation and norepinephrine release in the myocardium.

Assessment of cardiac function in these patients by echocardiogram or ventriculography has shown transient abnormalities of segmental contractility,[2] although some authors deny any relationship between the ECG alterations and the myocardial dysfunction observed. Others state a possible correlation between localisation of ST segment elevation and alterations of segmental contractility.[2] Nevertheless, coronarographic studies have not been able to demonstrate perfusion abnormalities in these patients, either from lesions or spasms of the coronary arteries.[2] On the contrary, perfusion abnormalities have been demonstrated using thallium myocardial scintigraphy in patients having subarachnoid haemorrhage associated with repolarisation alterations. There was no close correlation between those perfusion abnormalities and the ECG pattern or neurological status.[3] On the other hand, it has been postulated that the incidence of ECG alterations is linked to the amount of the haemorrhage quantified by CT scan.[4]

In some patients, autopsy reveals haemorrhagic subendocardial lesions, myocytolysis or myofibrillar degeneration.[2] Neither the presence of perfusion and contractility alterations, nor ECG changes have an influence on vital prognosis.[4] In those patients whose neurological symptomatology may be discrete, ECG abnormalities may lead to a disastrous therapeutic decision such as anticoagulant therapy or fibrinolysis. Serial ECGs and creatine kinase myocardial enzyme (CK-MB) measurement should be performed in patient presenting with subarachnoid haemorrhage.[5] Screening echocardiography is proposed in patients with obvious haemodynamic instability, pulmonary oedema, acute ischaemic ECG changes (Q waves or ST segment elevation or depression), or the combination of symmetric T-wave inversion with QT prolongation.[5] Borderline eleva-

Learning points

- subarachnoid haemorrhage can be associated with ECG abnormalities, frequently QT prolongation, ST and T modifications (sometimes mimicking an acute myocardial infarct), or supraventricular and ventricular arrythmias
- serial ECGs and measurements of CK-MB are recommended in all patients; echocardiography is recommended in patients with obvious haemodynamic instability, pulmonary oedema, acute ischaemic ECG changes (Q waves or ST segment elevation or depression), or the combination of symmetric T-wave inversion with QT prolongation.
- ECG monitoring during the first days following acute stroke is recommended for detection of life-threatening arrhythmias
- ECG abnormalities have no prognostic value by themselves and should not lead to anticoagulation or fibrinolysis

tions of CK-MB that are typically less than expected given the extent of left ventricular dysfunction (echocardiography) will aid differential diagnosis.[5] The frequency with which arrhythmias are encountered in these patients mandates continuous ECG monitoring during the first days following the acute event.[5]

1 Byer E, Ashman R, Toth L. Electrocardiograms with large, upright T waves and long QT intervals. *Am J Heart* 1947;**33**:796–806.
2 Kono T, Morita H, Kuroiwa T, Onaka H, Takatsuka H, Fujiwara A. Left ventricular wall motion abnormalities in patients with subarachnoid hemorrhage: neurogenic stunned myocardium. *J Am Coll Cardiol* 1994;**24**:636–40.
3 Samra SK, Kroll DA. Subarachnoid hemorrhage and intraoperative electrocardiographic changes simulating myocardial ischemia—anaesthesiologist's dilemma. *Anesth Analg* 1985;**64**:86–9.
4 Manninen PH, Ayra B, Gelb AW, Pelz D. Association between electrocardiographic abnormalities and intracranial blood in patients following acute subarachnoid hemorrhage. *J Neurosurg Anesthesiol* 1995;7:12–6.
5 Mayer SA, Swarup R. Neurogenic cardiac injury after subarachnoid hemorrhage. *Curr Opin Anaesthesiol* 1996;**9**:356–61.

Final diagnosis

Subarachnoid haemorrhage causing ECG abnormalities.

Keywords: subarachnoid haemorrhage; electrocardiography; coronary disease; arrhythmia

An abdominal mass following a road traffic accident

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An 18-year-old woman presented to her local Emergency department three weeks after a road accident. As an unrestrained backseat passenger she had been ejected from a car rolling over at 60 mph. Initially she was well and she was discharged from the local hospital after a period of observation. During the following weeks she had developed abdominal and back pain associated with anorexia. On examination she was found to have a tachycardia (108 beats/min) and a tender mass in the epigastrium. Following blood tests showing hyperamylasaemia (1271 IU/ml), and anaemia (9.8 g/dl), she had an abdominal computed tomography (CT) scan (figure).

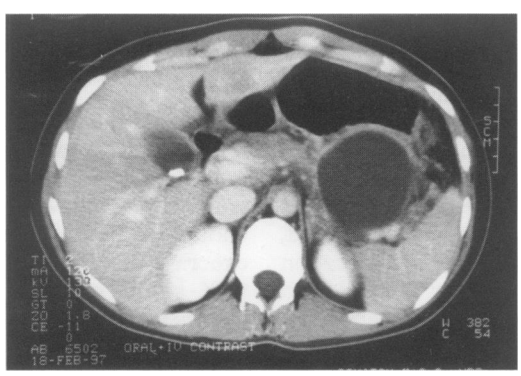

Figure Abdominal CT scan

Questions

1 Comment on the history and CT scan.
2 What is the injury and the mechanism?
3 What further investigations would you do?
4 How would you manage this patient?
5 What other complications might this patient develop?

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