Other full case

Electrocardiographic abnormalities in a patient with subarachnoid haemorrhage

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Summary

A 69-year-old woman presented after collapsing. She denied chest pain, breathlessness or headache. She was afebrile and vital signs were unremarkable. She was confused but the remaining physical examination was unremarkable. Routine blood tests were unremarkable. Cardiac enzymes were raised with a troponin I of 0.54. ECG showed Q waves in leads V1-V3 and widespread T wave inversion in leads II, III, aVF and V1-V6. Acute coronary syndrome (ACS) was suspected and antiplatelet treatment started. The following day her confusion worsened. Further review of the ECG found extensive changes unexplained by occlusion of a single artery suggesting extra-cardiac pathology. An urgent CT head was arranged and revealed subarachnoid haemorrhage. ACS treatment was stopped and she was transferred to neurosurgery where her right posterior communicating artery aneurysm was coiled. Fortunately her recovery was uneventful and she was discharged home with no neurological impairment.

BACKGROUND

Subarachnoid haemorrhage (SAH) is a serious neurological disorder, which may be complicated by the occurrence of electrocardiographic abnormalities. ECG changes associated with SAH can mimic changes associated with acute coronary syndromes (ACS). These may be misinterpreted resulting in inappropriate treatment. Institution of blood thinning medication in a patient with SAH may be fatal; therefore, it is essential that physicians are aware of the cardiac effects of this condition.

This case also highlights that sudden collapses, contrary to popular belief, are rarely due to ACS. Therefore, it is important not to treat collapses with no history of chest pain or sweating as ACS unless there is hard evidence beyond an abnormal ECG.

CASE PRESENTATION

A 69-year-old woman presented with a history of collapsing to the acute medical receiving unit. She denied any associated chest pain, breathlessness or headache. She had a medical history of chronic obstructive pulmonary disease for which she was on symbicort and bricanyl inhalers. On examination she was apyrexial and haemodynamically stable. She was confused but her Glasgow Coma Scale was 14 with no focal neurology.

The remainder of her physical examination was unremarkable.

INVESTIGATIONS

Her full blood count, electrolytes, liver function tests and inflammatory markers were all normal. Cardiac enzymes were raised with a troponin I of 0.54. Her 12-lead ECG showed sinus rhythm with normal axis. There was Q waves in leads V1-V3 and marked T wave inversion in leads II, III, aVF and V1-V6 (figure 1). Urine dipstick did not show evidence of infection.

DIFFERENTIAL DIAGNOSIS

Given the history of collapse, abnormal ECG and raised cardiac enzymes, an ACS was suspected.

TREATMENT

The patient was treated with antiplatelet agents and low molecular weight heparin. She was transferred to the cardiology unit the following day. On further review of her ECG by the cardiologists it was noted that she had extensive changes, which could not be explained by the occlusion of a single artery suggesting that they might not reflect an ACS. She was also noted to be progressively more confused. An urgent CT head was arranged and took place on the day following admission. The CT scan revealed an extensive SAH (figure 2A,B).

Her ACS treatment was stopped and she was transferred to neurosurgery where her right posterior communicating artery aneurysm was coiled.

OUTCOME AND FOLLOW-UP

She made a full neurological recovery and was discharged home. A repeat 12 lead ECG showed some resolution of her changes with only minor ST segment flattening in V5-6. An echocardiogram showed overall good left ventricular systolic function with no regional wall abnormalities.

DISCUSSION

SAH is a serious neurological disorder, which is associated with ECG changes in 50–100% of patients. These mostly consist of ST changes, QT prolongation and U wave abnormalities,^{1 2} which can mimic changes due to myocardial ischaemia or infarction and if misinterpreted can lead to inappropriate or harmful treatment. SAH is also associated with a predisposition to serious potentially life threatening arrhythmias³ and can be associated with neurogenic

Figure 1 ECG Q waves in leads V1-V3 and marked T wave inversion in leads II, III, aVF and V1-V6.

Figure 2 (A) CT of head demonstrating blood in the quadrigeminal cistern. (B) CT of head demonstrating blood in the sylvian fissure.

cardiac dysfunction. A diminished left ventricular ejection fraction (<50%) is observed in 8–15% patients; although cardiogenic shock rarely occurs.^{4 5} Wall motion disorders usually occur in the first days after SAH, do not match coronary artery distribution and generally show spontaneous improvement.⁴ Altogether, 20–40% of cases of SAH demonstrate troponin T release and this positively correlates with the severity of neurological symptoms.^{5 6} The explanation for the effects of SAH on the heart is unclear. One hypothesis is that they are driven by raised intracranial pressure causing stimulation of the hypothalamus with a resultant increase in sympathetic activation and catecholamine release.¹ However, in our case the absence of hypertension and tachycardia on presentation would not support this hypothesis.

Several case reports have previously documented the confounding effect that ECG abnormalities can have in the management of patients with SAH: Beard *et al*⁷ described a case of a 37-year-old woman admitted with a brief syncope prior to admission and stupor. She had no neurological signs, but had extensive ST segment elevation and T wave inversion. Myocardial infarction was diagnosed and anticoagulation treatment was started. The patient died several days later and autopsy revealed a SAH, an intracranial aneurysm and a normal heart. Another report described the postponement of surgery on a patient with a SAH because of ECG changes consistent with anterior myocardial infarction. This patient died and again autopsy findings included a ruptured aneurysm and no cardiac abnormalities.³

A careful history and examination is essential in the management of a patient presenting with collapse. Sudden collapses, contrary to popular belief, are rarely due to ACS. Patients presenting with a collapse without cardiac sounding symptoms should not be treated for an ACS unless there is hard evidence beyond an abnormal ECG.

Neurologically mediated ECG changes may be misdiagnosed as myocardial ischaemia or infarction resulting in delayed or harmful treatment of the primary problem.

Learning points

- ▶ ECG changes associated with SAH can mimic changes associated with coronary ischaemia or infarction.
- ▶ Anticoagulation of a patient with SAH may be fatal.
- ▶ ECG changes not conforming to a coronary artery distribution may reflect extra-cardiac pathology.
- ▶ Sudden collapses, contrary to popular belief, are rarely due to ACS.

Competing interests None.

Patient consent Obtained.

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