CASE REPORT

The ischaemic ECG: is it all in the head?

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SUMMARY

Acute cerebrovascular events including ischaemic stroke and subarachnoid haemorrhage are known to be associated with left ventricular dysfunction and significant electrocardiographic abnormalities that can mimic cardiac ischaemia. The authors present a case in which an intracerebral haemorrhage was diagnosed during the treatment of pneumonia in a 62-year-old woman, with repolarisation abnormalities on the ECG acting as the key diagnostic clue in the absence of clinical or radiological evidence of ventricular dysfunction. The pneumonia was treated without complication, and there were no long-term respiratory, cardiac or neurological sequelae. Diagnostic cardiac imaging, including angiography at follow-up, confirmed the absence of significant coronary artery disease, suggesting a non-coronary aetiology for these ECG changes. The authors emphasise that when presentation is atypical for acute coronary syndrome, non-specific ECG changes could point to a primary cerebrovascular pathology, even in the absence of neurological or cardiac symptoms.

BACKGROUND

The ECG is one of the most frequently performed investigations in clinical practice. Cardiac ischaemia can manifest on the ECG as ST segment elevation, ST segment depression, and T-wave flattening or inversion. This case highlights the association of an acute cerebrovascular pathology with diffuse 'ischaemic' changes on the ECG. It demonstrates that electrocardiographic changes can in fact serve as an important diagnostic clue to non-cardiac pathology. Treating the ECG rather than the patient in these cases could worsen the prognosis, particularly if antiplatelet therapy is initiated on the background of an intracranial haemorrhage.

CASE PRESENTATION

A previously fit, normotensive 62-year-old woman presented to our emergency department (ED) with a 2-day history of shortness of breath, productive cough and subjective fevers. The patient had no significant medical history or family history, and no cardiovascular risk factors. She did not take any regular prescription or over-the-counter medication. On arrival to the ED, the patient was dyspnoeic, with a respiratory rate of 22/min and oxygen saturations of 89% on air. The patient was haemodynamically stable and without fever. Physical examination revealed crackles on auscultation of the right lung.

INVESTIGATIONS

An arterial blood gas revealed type I respiratory failure (pO_2 7.29 kPa, pCO_2 5.00 kPa) and blood tests revealed a raised white cell count (15.4×10^3 /mL). Chest X-ray (figure 1) showed patchy right middle lobe shadowing. A CT pulmonary angiogram was performed and excluded pulmonary embolism while confirming findings of pneumonia. As part of the diagnostic work-up, an ECG was performed, which demonstrated T-wave inversion in leads I, aVL and all precordial leads (figure 2). However, the patient did not have any symptoms of chest pain or other signs of cardiac ischaemia. She was treated with intravenous antibiotics for pneumonia, while a cardiac troponin T (cTnT) was requested to further investigate the abnormal ECG findings.

The cTnT result was raised at 80 ng/L and the decision was made, in the absence of any cardiac symptoms, to continue to monitor ECGs and repeat the cTnT test. Ten hours later, the ECG revealed deepening T-wave inversion (figure 2), and repeat cTnT was 100 ng/L. The patient again denied any chest discomfort and her vital signs had stabilised, but the ECG changes and raised troponin remained unexplained. On the second day of admission, a cardiology specialist performed a transthoracic echocardiogram which revealed no regional wall motion abnormalities or left ventricular dysfunction. Given these findings, and the absence of symptoms in the patient, suspicion for an underlying myocardial infarct was low. However, the patient's ECGs continued to show dynamic changes suggestive of cardiac ischaemia.

Figure 1 Chest X-ray demonstrating right middle lobe consolidation.

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Figure 2 Initial 12-lead ECG.

At this point, on further questioning, the patient recalled suffering from an occipital headache on the day prior to admission. She described the headache as severe and with sudden onset, but admitted that she had been distracted from it by her respiratory symptoms. At the time of questioning, the headache had completely resolved. The patient reported some mild neck stiffness, but admitted that this had been long-standing. There were no focal neurological signs on examination. Given the history of headache, the possibility of a subarachnoid haemorrhage as the underlying cause for the ECG abnormalities was considered.

A CT head was performed 36 h after admission, and revealed a small, acute, intracerebral haemorrhage (ICH) within the right basal ganglia. Examination and repeated observations revealed normal blood pressure and no neurological deficits and the patient was treated conservatively. Coagulation studies and platelet count were within normal limits. Serial ECGs (figure 3) showed continuing dynamic changes over the following days with inverted T-waves and QTc prolongation, before resolution to baseline.

OUTCOME AND FOLLOW-UP

The patient improved clinically on antibiotic treatment for her pneumonia over the following days and made a full recovery. She was discharged with cardiology and neurology follow-up. MRI scans carried out at 1 week (figure 4) and 1 month confirmed appropriate evolution of a small right basal ganglia haemorrhage. One month postdischarge, a coronary angiogram revealed a normal left main coronary artery and circumflex artery, with 20–30% proximal atheroma of the left anterior descending artery. The dominant right coronary artery had 20–30% proximal atheroma. These findings were thought to represent only minor coronary artery disease which would have been extremely unlikely to be the cause of the ECG abnormalities, supporting the relative lack of troponin rise and the normal echocardiogram. At 1 year postdischarge, the patient continued to be well and was discharged from outpatient follow-up.

DISCUSSION

ECG changes in association with acute cerebrovascular pathology are recognised, and in this case were likely to have been secondary to an ICH. Results of angiography excluded

obstructive coronary artery disease as a cause for the findings. The aetiology of the patient's ICH remains unclear and its presentation in association with an otherwise uncomplicated pneumonia is interesting. ICH has previously been reported in the setting of mycoplasma pneumonia and thrombocytopenia.¹ In our patient, serological tests were not performed but her platelet count remained within normal range.

ECG changes and elevated cTnT levels are more commonly seen in association with subarachnoid haemorrhage² and ischaemic stroke³ than with intracerebral haemorrhage. These phenomena are often associated with a transient left ventricular dysfunction, which is observed in several other clinical situations such as after suffering severe emotional stress (Takotsubo's cardiomyopathy or 'apical ballooning syndrome'), during acute illness and in association with pheochromocytoma.²

The underlying pathogenesis of these cardiac complications is not fully understood. It has been suggested that neurocardiogenic influences characterised by increased sympathetic nervous system activity on the heart are responsible for the observed ECG changes after acute intracranial events, and they may be explained by a catecholamine-induced effect.^{2 4 5} It is likely that the size and location of strokes correlate with cardiac ischaemic changes and cardiac arrhythmias, reflecting the varying roles of different areas of the brain. In an attempt to better understand the neuroanatomical basis for stroke-related myocardial injury, Ay *et al*⁶ analysed regions of infarction in patients after ischaemic stroke, and found that strokes within the right insula and right inferior parietal lobule were associated with elevated cTnT indicative of myocardial injury. Right insular involvement has also been shown to be an independent predictor of early mortality following stroke.⁷ A neurobiological basis for this observation may be the role of the right insula in autonomic modulation of cardiac activity. In this particular case, the ICH was located within the right basal ganglia, in close approximation to the insular cortex.

There are a limited number of case reports recognising cardiac complications in ICH. One case series found increased cTnT levels in 20% of supratentorial haemorrhages⁸ without confirmatory ECG changes. In a study by Hasegawa *et al*⁹ 41% of patients with an ICH had ischaemic appearing ECG changes. In our patient, the rise in cTnT was associated with profound

Figure 3 ECG recordings over the first 5 days of admission showing dynamic changes with deepening T waves over the first 24 h: (i) 0 h, (ii) 10 h, (iii) 24 h, (iv) day 2, (v) day 3 and (vi) day 5.

repolarisation abnormalities on the ECG, but an absence of ventricular dysfunction or haemodynamic compromise. The elevated cTnT probably reflects subclinical myocardial damage.

This case highlights that reversible cardiac ischaemic signs on the ECG can occur in the setting of an ICH which is clinically 'silent'. Such brain–heart interactions can have a profound effect on the ECG even in the absence of other clinical cardiac signs or symptoms, thereby posing a difficult diagnostic conundrum.

Figure 4 T₂-weighted fluid attenuated inversion recovery MRI at 1 week showing a small right basal ganglia haemorrhage.

Learning points

- ▶ Acute intracranial pathology can cause ECG changes which mimic cardiac ischaemia.
- ▶ These ECG changes may be explained by a catecholamine-induced effect.
- ▶ Physicians should consider an intracranial haemorrhage or ischaemic stroke in the patient with ECG changes in the absence of cardiac signs or symptoms.
- ▶ The absence of significant neurological signs or symptoms does not preclude acute intracranial pathology, which could be potentiated by antiplatelet therapy.

Contributors All authors were involved in the care of the patient. The case report was written by APP and BII. The case report was revised and approved by FD.

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