Case Report

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Acute ECG changes in a woman presenting to coronary care with fluctuating consciousness

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Abstract

We present the case of a 67-year-old woman brought into the coronary care unit (CCU) with a suspected ST-segment elevation myocardial infarction (STEMI) due to lateral ST-segment elevation on her 12-lead electrocardiogram (ECG) and a significant troponin rise, but no reported chest pain and a fluctuating consciousness level. Whilst in CCU, she deteriorated further with a reduction in consciousness and sluggish pupillary reflexes, warranting urgent computed tomography (CT) of her brain, which confirmed extensive subarachnoid haemorrhage (SAH) with early evidence of hydrocephalus. She was therefore transferred to the local tertiary neurosurgical centre for endovascular coiling. ECG changes alongside a raised troponin are not uncommon findings in SAH and clinicians should exercise vigilance and consider urgent brain imaging in the absence of chest pain and presence of neurological deficit, to prevent adverse events from unnecessary antiplatelet or anticoagulant therapy, and invasive coronary angiography. SAH is a medical emergency and prompt recognition and referral for neurosurgical intervention is integral for optimal patient outcome.

Keywords

Arrhythmias < cardiovascular medicine < CLINICAL, other neurology < neurology < CLINICAL, headache (including migraine) < neurology < CLINICAL

ECG changes alongside fluctuating consciousness are not uncommon findings in subarachnoid haemorrhage (SAH), and clinicians should therefore consider urgent brain imaging.

Case presentation

A 67-year-old woman was referred as a pre-alert to our Institution's coronary care unit (CCU) with 1–2mm of ST elevation in the lateral leads on 12-lead electrocardiogram (ECG) alongside vomiting and diaphoresis. She was given 300mg of aspirin by paramedics, who subsequently noted that she was drowsy in the ambulance, however in view of a possible ST-segment elevation myocardial infarction (STEMI), she was transferred to the CCU. Prior to admission, this lady was independent in activities of daily living, using a walking stick to mobilise due to her arthritis. Her medical history consisted of rheumatoid arthritis, osteoarthritis, chronic leg ulcers, and non-alcoholic fatty liver disease.

On arrival to CCU, the patient's drowsiness temporarily resolved, and she was orientated to time, place, and person. She denied chest pain, dyspnoea, or palpitations. Clinical observations and examination were unremarkable, with a clear chest and no audible murmurs on auscultation. Neurologically, pupils were equal and reactive to light, with no focal neurology of the limbs or cranial nerves and a Glasgow Coma Scale (GCS) score of 15.

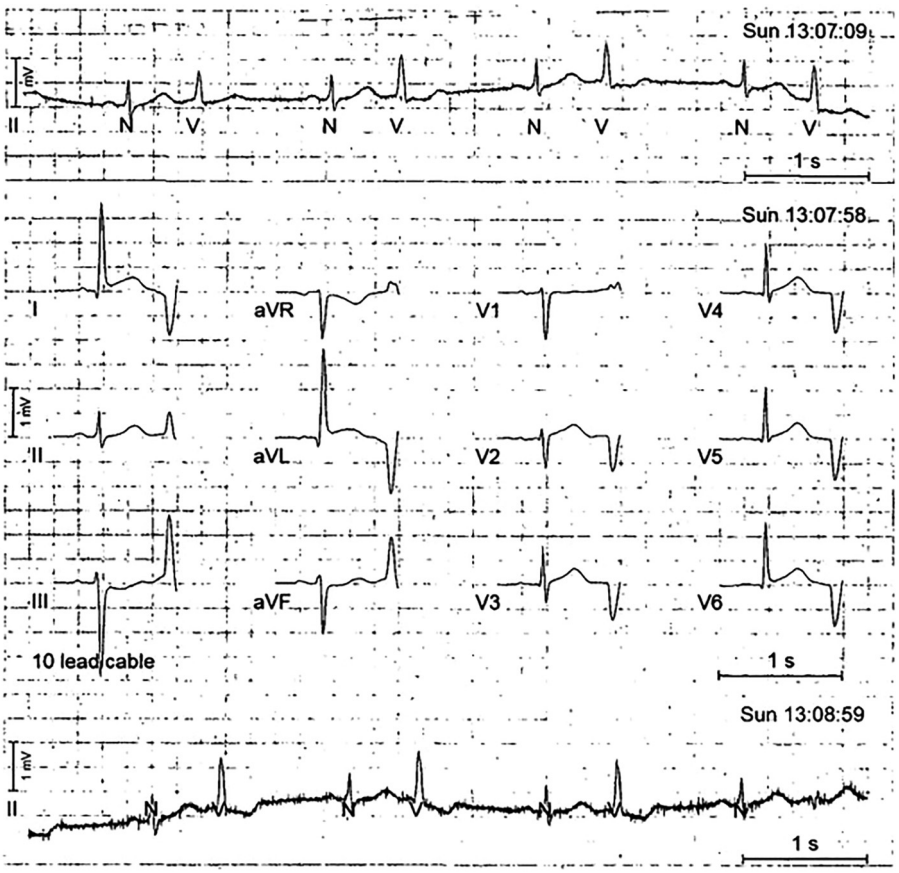
On re-assessment an hour later, nursing staff noted a reduced consciousness level. GCS was now 12: scoring 3 for eye opening response, 4 for verbal response, and 5 for motor response. Pupillary reflexes were reactive but sluggish, and the remainder of the examination was unchanged. Collateral history was obtained from a family member. They described how this patient had phoned earlier in the day complaining of a sudden onset headache, radiating to the neck, which was associated with vomiting.

Investigations

A 12-lead ECG showed sinus rhythm, normal R wave progression, and left axis deviation; with Q-waves and 1mm ST-segment elevation in the leads V5, V6, I, and aVL and reciprocal ST-segment in III and aVR (Figure 1). QRS duration and corrected QT interval were within normal limits. Furthermore, there was evidence of ventricular bigeminy on the patient's rhythm strip (Figure 1). Blood tests demonstrated a raised high-sensitivity troponin of 180ng/L. A bedside transthoracic echocardiogram demonstrated mild left ventricular systolic impairment with apical septal hypokinesia.

However, in view of the fluctuating consciousness level, an urgent non-contrast computed tomography (CT) head was requested which demonstrated clear evidence of an extensive SAH filling the basal cisterns, sylvian fissures, and the subarachnoid spaces along the

Figure 1. Pre-hospital Ambulatory ECG demonstrating lateral ST elevation in leads V5-V6, I, and aVL; with reciprocal ST depression in leads III and aVF. There is also evidence of sinus rhythm with ventricular bigeminy on the associated rhythm strip.



medial aspects of frontal lobes; with associated early hydrocephalus (Figures 2(a) and 2(b)). A CT angiogram clarified the source of this bleed to be a 3 mm saccular aneurysm extending from the anterior communicating artery (Figure 2(c)).

Management and outcome

Discussion with neurosurgery led to urgent transfer to the local tertiary centre where endovascular coiling of the anterior communicating artery aneurysm was performed (Figure 2(d)), with subsequent admission to the neuro-intensive care unit.

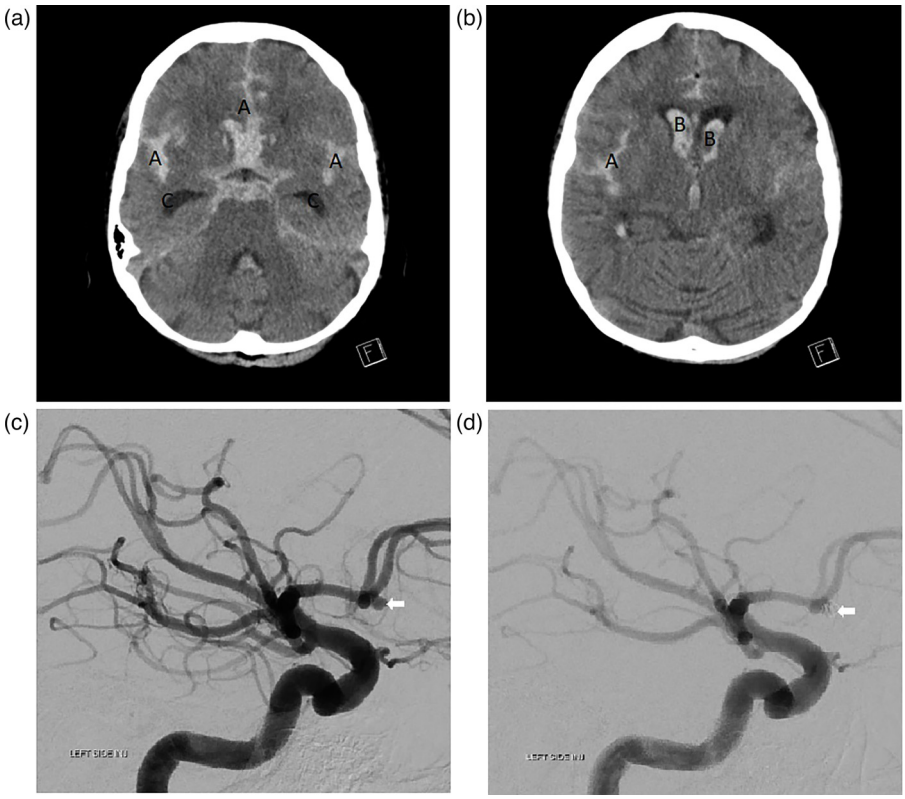
The patient underwent a further 3-month hospital stay with ongoing neurorehabilitation and was subsequently discharged home, with a formal package of care to assist with personal care and household tasks. Unfortunately, she experiences ongoing fatigue, with new short-term memory difficulties, and now requires a Zimmer frame to mobilise.

Discussion

SAH is an uncommon yet severe medical emergency, most frequently caused in 85% of cases following rupture of an intracranial aneurysm. A classical history of sudden onset severe occipital headache is present in 50% of cases.[1] Other associated symptoms include nausea and vomiting, transient loss of consciousness, fluctuating conscious level, and focal neurological deficits.

SAH results in brain injury due to transient global ischemia and the direct toxic effect of subarachnoid blood on brain tissue.[2] Current literature suggests that the combination of anterior hypothalamic stimulation and a catecholamine surge during aneurysm rupture leads to increased sympathetic tone, causing coronary artery vasospasm.[3] This leads to a neurogenic stunned myocardium, that mimics an acute coronary syndrome (ACS) by causing reversible left ventricular systolic dysfunction, decompensated heart failure, and even cardiogenic shock.[4] Therefore, neurological insults such as

Figure 2. a: CT brain demonstrating extensive subarachnoid haemorrhage, with blood filling the subarachnoid spaces (A). b: There is also evidence of intraventricular haemorrhage, involving the lateral, third, and fourth ventricles (B) and the presence of early hydrocephalus with dilatation of the temporal horns (C). 2c: Lateral Projection digital subtraction angiography (DSA) image demonstrating an anterior communicating artery aneurysm prior to embolization (arrowed). 2d: Lateral Projection DSA image demonstrating the anterior communicating artery aneurysm following embolisation (arrowed).



SAH can present with apparent left ventricular dysfunction and could be mistaken for a primary cardiac pathology if alternative causes of non-specific ECG changes and troponin rise are not considered, particularly if there is no history of chest pain, and presence of neurological deficit or fluctuating level of consciousness.

ECG changes are thought to be present in over 50% of patients with SAH.[5] These changes are most prevalent within the first 48 h and commonly include QT prolongation, T wave inversion, and ST segment changes.[6] In addition, various arrythmias have been associated with SAH, including ventricular bigeminy as in our case.[7] Previous studies suggest ECG changes and SAH associated cardiac arrhythmia are independent predictors of adverse outcome.[4,7] Elevated troponin is apparent in 20–40% of individuals with SAH and is associated with increased mortality and morbidity from SAH.[8] Moreover, transient localised left ventricular hypokinesis on echocardiogram has been reported in approximately 10% of SAH patients, often with no evidence of significant coronary artery disease (CAD) on coronary angiography. In this case, the apical septal pattern of regional wall motion abnormalities fits the distribution of the myocardial sympathetic nerve terminals, rather than significant CAD.[8]

Due to the considerable mortality and morbidity from SAH, a low threshold for investigation is recommended, with a non-contrast CT head advised first-line.[9] If a CT scan shows no evidence of pathology, and SAH is still suspected, then a lumbar puncture should be undertaken to look for xanthochromia. Once a definitive diagnosis has been made, referral to a neurosurgical centre is required for management of the bleed and complications including hydrocephalus.[10] A good awareness of neurologically mediated ECG changes, in conjunction with good history taking and clinical examination will aid timely diagnosis and management and is therefore critical to patient outcome.

Key points

- Although neurologically mediated ECG changes and troponin elevation in SAH are well established, it is not uncommon for these cases to be misdiagnosed as

myocardial ischaemia, resulting in delayed or harmful treatment of the primary problem.

- Careful history and examination can help to differentiate SAH from ACS: a fluctuating conscious level alongside intermittent confusion in the context of a troponin rise and non-specific ECG changes should prompt consideration of an alternative diagnosis, such as a neurological event.
- Consider a low threshold for a CT head in patients with these neurological symptoms in the absence of clear features of an ACS prior to initiating antiplatelet therapy and/or invasive coronary angiography.

Declarations

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Patient consent: Obtained from the patient for publication.

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