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THE GREAT MIMICKER: A UNIQUE CASE OF DIFFUSE SUBARACHNOID HAEMORRHAGE SIMULATING ACUTE MYOCARDIAL INFARCTION

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ABSTRACT

Subarachnoid haemorrhage (SAH) is a rare yet consequential medical emergency that may mimic an acute myocardial infarction (MI). SAH causes enhanced sympathetic activity, culminating in the development of neurogenic stunned myocardium (NSM), which presents as ST-segment deviations, prolonged QT intervals, T-wave inversions or Q-waves. Reperfusion therapy is contraindicated for SAH because of an increased risk of bleeding and death. Therefore, a prompt diagnosis is crucial. Here, we report a unique case of massive SAH presenting as diffuse ST-segment deviation simulating an acute MI. Our patient was brought to the emergency department after a cardiac arrest and died on day 2 of admission.

KEYWORDS

subarachnoid haemorrhage, acute myocardial infarction, neurogenic stunned myocardium, electrocardiogram, ST-segment depression

LEARNING POINTS

- Subarachnoid haemorrhage (SAH) can present with EKG changes and significant rise in troponin, mimicking acute coronary syndrome.
- SAH should be included in the differential diagnosis for patients presenting with neurological symptoms, ST-segment deviations or prolonged QT intervals.
- Misdiagnosis of SAH as acute coronary syndrome can lead to inappropriate use of anticoagulants or delays in necessary neurological interventions.

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INTRODUCTION

Subarachnoid haemorrhage (SAH) is a rare, yet significant medical emergency primarily caused by a ruptured intracranial aneurysm[1]. SAH leads to brain damage due to transient global ischaemia and the toxic effects of blood on the brain tissue[1]. In 1953, Levine et al. reported the first case of SAH with abnormal electrocardiogram (ECG) changes mimicking acute coronary syndrome (ACS)[2], and few cases have since been reported. Brain aneurysm rupture causes anterior hypothalamic activation and catecholamine surge, leading to neurogenic stunned myocardium (NSM)[3,4], which simulates ACS[1]. This is an extremely rare and highly critical event; a misdiagnosis can lead to inappropriate use of anticoagulants or delay in neurosurgical intervention[3]. We present a unique case of massive SAH that was initially misdiagnosed as acute MI due to persistent ST-segment depression on ECG and a significant rise in troponin levels. This case contributes to the limited literature on SAH simulating an ACS event.

CASE DESCRIPTION

A 44-year-old Hispanic female with a history of obesity, iron deficiency anaemia and hypertension was brought to the emergency department (ED) after cardiac arrest. According to the advanced life support team, the patient collapsed at a bar after consuming two alcoholic beverages. Family and friends denied any chest pains, palpitations or seizure-like activities before the collapse. The initial rhythm was pulseless electrical activity, and a return of spontaneous circulation (ROSC) was achieved after an unknown period. The team administered one round of epinephrine, 50 amps of sodium bicarbonate and 250 cc of intravenous fluids during the code. The patient was intubated with an 8.0 tube in the field, and manual resuscitation was performed via a bag-and-valve mask until arrival at the ED.

On initial evaluation in the ED the patient was afebrile, normotensive and tachycardic at 144 beats per minute. Using an Accu-Chek® in the field revealed a blood sugar level of 244 mg/dl and upon examination, the patient was found to be unresponsive. The pupils were 5 mm bilaterally, fixed and non-reactive to light or corneal reflexes. Oculocephalic and oculovestibular reflexes were absent. The patient had absent gag and cough reflexes, and her extremities were flaccid bilaterally. The initial laboratory values were as follows: sodium 145 mEq/l, potassium 2.3 mEq/l, phosphorus < 1.0 mEq/l, creatine kinase 640 U/l, lactic acid 4.2 mmol/l and a high sensitivity troponin at 11,904 pg/ml. The urine drug screening results were unremarkable. Arterial blood gas revealed a pH of 7.21, carbon dioxide partial pressure of 47 mmHg, oxygen of 67 mmHg, bicarbonate of 11.6 mEq/l and lactic acid of 10.5 mmol/l.

The patient was coded twice in the ED, and ROSC was achieved in less than two minutes each time. The patient became bradycardic and hypotensive, and she was started on norepinephrine and later switched to epinephrine, after central venous access was established. An emergent ECG

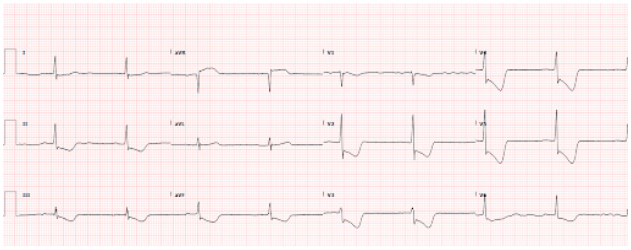

Figure 1.

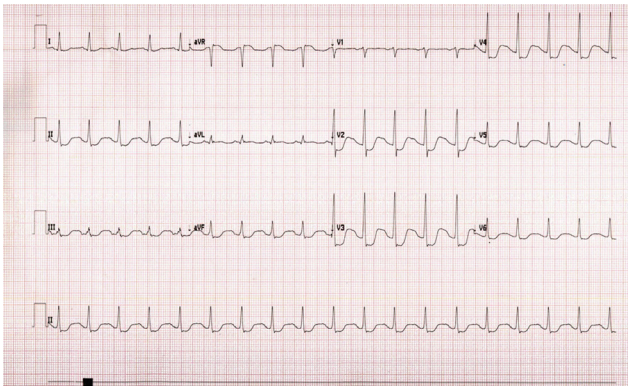

Figure 2.

revealed sinus bradycardia, global ST-segment depressions and a single ST elevation in lead aVR, indicating possible left main coronary artery disease *(Fig. 1)*. ST depressions in the anterior leads with R>S were suggestive of posterior wall involvement. Serial ECGs consistently showed ST-segment depression, which was suggestive of coronary artery disease *(Fig. 2)*. An ST-elevation myocardial infarction (STEMI) code was activated, and the patient was administered aspirin and ticagrelor, and started on a heparin drip. The patient was immediately taken for left heart catheterization, which revealed normal coronary angiography findings and normal left ventricular function.

After this, the patient was transferred to the cardiac care unit where she coded again and required a shock. The initial rhythm was ventricular fibrillation, and ROSC was attained within 8 minutes. A computed tomography (CT) scan of the brain and head without contrast revealed a diffuse subarachnoid haemorrhage and significant cerebral oedema with crowding of the basilar cisterns and a mass effect on the ventricles *(Fig. 3)*. The patient's neurological examination result was very poor, with a Glasgow coma scale score of 3. Neurosurgery was consulted and surgical intervention was deferred, given the patient's clinical status. A CT angiography of the brain/head revealed no intracranial arterial flow or perfusion, suggesting brain death, which was confirmed by a nuclear medicine study. We notified the family about the patient's neurological prognostication, and she was pronounced dead.

DISCUSSION

ACS is a medical emergency and is the leading cause of out-of-hospital cardiac arrest[5]. ACS is caused by cardiac ischaemia, resulting in myocardial injury or necrosis[1,6]. Once ROSC is attained, reperfusion therapy is recommended

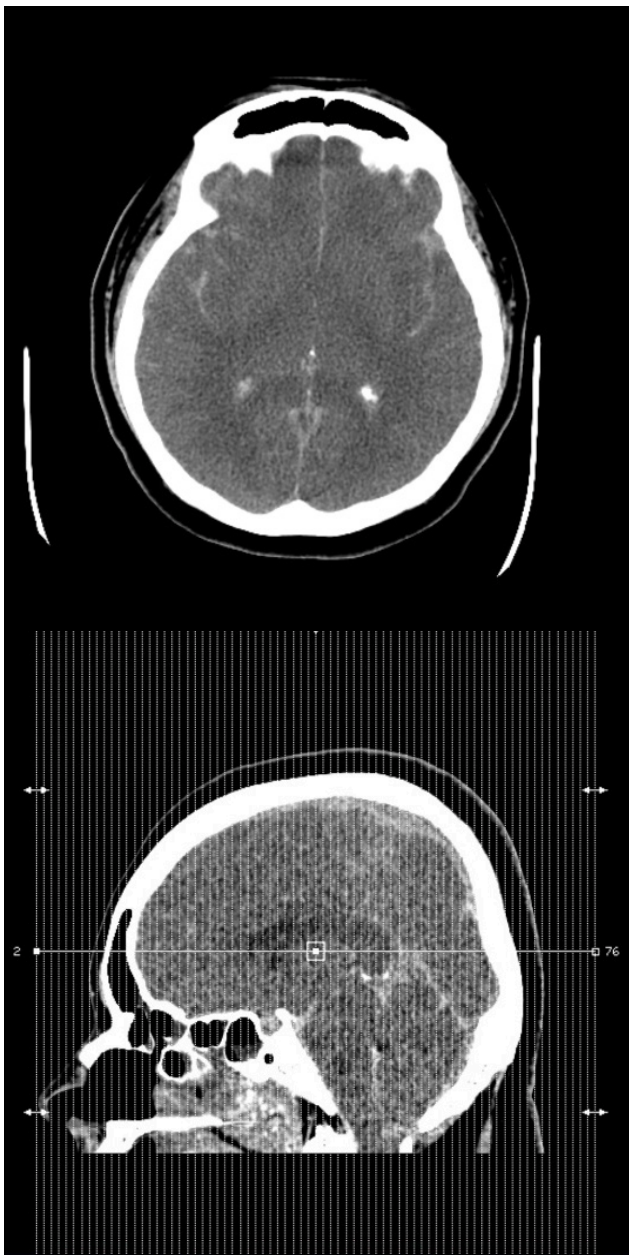

Figure 3.

according to the Advanced Cardiac Life Support guidelines[5]. Rarely, cardiac arrest may be due to SAH, an absolute contraindication to antiplatelet or anticoagulant use[5], due to an increased risk of rebleeding and worsening neurological outcomes. An ECG is essential for diagnosing ACS and localising cardiac injuries[6]. Various neurological disorders have been observed to mimic the ECG changes observed in ACS[6]. These conditions include SAH, intracranial haemorrhage, subdural haematoma, brain tumours, brain abscesses, tuberculoma and meningitis[3,6,7]. However, these observations are infrequent in clinical practice and the literature. Our patient exhibited ST-segment depression in leads V2–V6 and a slight ST-segment elevation in lead aVR, which was initially misinterpreted as ACS. Similarly, Yogendranathan et al. observed an unusual case of STEMI and T-wave inversions on a 12-lead ECG in a patient with a large meningioma[6].

SAH is a catastrophic event with an annual incidence of six cases per 100,000 individuals[8]. Potential complications include hydrocephalus, seizures, rebleeding, hyponatraemia, infections and global brain ischaemia[8]. It has been postulated that the excess catecholamine released during SAH induces NSM. NSM leads to myocardial ischaemia, which often manifests as ST-segment deviations, QT interval prolongation or T-wave inversions on a 12-lead ECG[1,3,9]. Elevated serum cardiac markers, ventricular and atrial arrhythmias[3,9] and motion abnormalities have also been reported[1]. It has also been suggested that catecholamines have toxic effects on the myocardium, as evidenced by the subendocardial lesions noted in patients with SAH, a phenomenon known as contraction band necrosis[7,10,11]. Epinephrine infusions have successfully replicated this phenomenon in animal models[7].

In most instances, SAH initially presents as a severe occipital headache[1]; however, nausea, vomiting and altered mentation have also been reported in some cases[12]. In post-cardiac arrest patients, distinguishing between ACS and SAH can be a diagnostic challenge, even for seasoned clinicians. Therefore, it is essential to handle such cases with care and pay close attention to any signs of neurological abnormalities such as paralysis or severe headache, as well as to consider the likelihood of cerebrovascular disease, necessitating the need for cautious use of antiplatelet agents[7,10]. Delayed neurosurgical intervention may also worsen the patient's prognosis. Serial ECGs, emergent coronary angiography and non-contrast CT scans of the brain may help to differentiate ACS from SAH.

CONCLUSION

SAH is a severe, yet rare, medical emergency that warrants urgent intervention. In a few of the patients, SAH may cause ECG changes and significant troponin rise, thus simulating ACS. SAH should be considered in the differential diagnosis of patients with neurological symptoms, ST-segment deviations or prolonged QT intervals. Misdiagnosis can lead to the inappropriate use of anticoagulants or delays in neurosurgical intervention.

REFERENCES

1. Parker H, El-Medany A, Crossley R. Acute ECG changes in a woman presenting to coronary care with fluctuating consciousness. *JRSM Open* 2022;**13**:2054270422112348.
2. Levine HD. Non-Specificity of the electrocardiogram associated with coronary artery disease. *Am J Med*. 1953 Sep;**15**(3):344-55.
3. Weintraub BM, McHenry LC Jr. Cardiac abnormalities in subarachnoid hemorrhage: a resumé. *Stroke* 1974;**5**:384–390.
4. Aikhionbare I, Gruber M, Gopalakrishnan P. Subarachnoid hemorrhage masquerading as ST elevation. *JACC* 2022;**79**:2477.
5. Wei Ting W, Lai PF, Chang H. Subarachnoid hemorrhage–induced out-of-hospital cardiac arrest patients with ST-segment elevation from the electrocardiogram: two case reports. *Hong Kong J Emerg Med* 2019;**28**:320–323.
6. Yogendranathan N, Herath HM, Pahalagamage SP, Kulatunga A. Electrocardiographic changes mimicking acute coronary syndrome in a large intracranial tumour: a diagnostic dilemma. *BMC Cardiovasc Disord* 2017;**17**:91.
7. Heo WJ, Kang JH, Jeong WS, Jeong MY, Lee SH, Seo JY, et al. Subarachnoid hemorrhage misdiagnosed as an acute ST elevation myocardial infarction. *Korean Circ J* 2012;**42**:216–219.
8. Amodio S, Bouzat P, Robba C, Taccone FS. Rethinking brain injury after subarachnoid hemorrhage. *Crit Care* 2020;**24**:612.
9. Sakamoto H, Nishimura H, Imataka K, Ieki K, Horie T, Fujii J. Abnormal Q wave, ST-segment elevation, T-wave inversion, and widespread focal myocytolysis associated with subarachnoid hemorrhage. *Jpn Circ J* 1996;**60**:254–257.
10. Köklü E, Yüksel IÖ, Bayar N, Küçükseymen S, Arslan Ş. Subarachnoid hemorrhage that electrocardiographically mimics acute coronary syndrome: a case report. *Turk Kardiyol Dern Ars* 2015;**43**:730–733.
11. Chen HY. Angiographic coronary spasm in a case of spontaneous subarachnoid hemorrhage mimicking acute myocardial infarction. *Cardiol Res* 2013;**4**:74–77.
12. Rouanet C, Silva GS. Aneurysmal subarachnoid hemorrhage: current concepts and updates. *Arq Neuropsiquiatr* 2019;**77**:806–814.