

Case Report

Subarachnoid haemorrhage mimicking unstable angina: a case report

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ABSTRACT

Subarachnoid haemorrhage (SAH) is medical emergency caused by bleeding into the subarachnoid space. It is caused by rupture of an aneurysm or arteriovenous malformations (AVM). Symptoms of SAH are severe headache, nausea, vomiting, impaired consciousness and seizures. Neck stiffness and neck pain are relatively uncommon. Risk factors are high blood pressure, smoking, family history, alcoholism and even cocaine use. Diagnosis is mainly made by CT scan of head which should be done within six hours of the onset of symptoms and occasionally lumbar puncture can also be done. An electrocardiogram (ECG) of all patients with subarachnoid should be done because patient with SAH can have myocardial ischemia due to increased level of circulating catecholamines or due to autonomic stimulation of the brain. ECG changes associated with SAH primarily reflect repolarisation abnormalities involving ST segment, T wave, U wave and QTc interval. Myocardial ischemia or infarction is often suspected in patients with SAH. Even troponin levels may be raised in these patients. However, suspicion of SAH is a contraindication for thrombolytic and anticoagulant therapy. This is a case of SAH which was initially treated for acute coronary syndrome (ACS) on the basis of symptoms and gradually changing ECG findings but on CT Head, patient was having SAH.

Keywords: Subarachnoid space, SAH, ECG

INTRODUCTION

SAH is the bleeding in the subarachnoid space which is the area between arachnoid matter and pia matter surrounding the brain. SAH may be due to ruptured aneurysm, AVM or a consequence of head injury. It is a life-threatening type of stroke may cause death in one third of patients.

SAH has long been known to be associated with ECG changes¹. ECG changes associated with SAH primarily reflect repolarization abnormalities involving the ST segment, T wave, U wave, and QT interval. Because of the combination of ST-segment elevation or depression and abnormal T-wave morphology, myocardial ischemia or infarction is often suspected in patients with SAH.

This may cause physicians to be confused between SAH and ACS.²

In this article we report a case of 62 years old female presented to hospital with chief complaint of vomiting, giddiness and epigastric discomfort. Based upon serial ECG changes she was diagnosed to be having unstable angina but later on turned out to be SAH.

CASE REPORT

A 62-year female presented to emergency department with chief complaints of epigastric discomfort, nausea and dizziness for one day. Pain was acute in onset with no radiation, there was no relationship with meals, pain was not relieved specific posture. There was no history of headache.

At presentation she was conscious, oriented to time, place, and person. Her vitals were blood pressure 160/90 mm Hg, pulse rate 90 per minute, oxygen saturation 98% on room air. There was mild epigastric tenderness. Cardiovascular examination was normal. Patient was treated for dyspepsia.

Blood investigations revealed haemoglobin of 10.5g/dl, TLC 14,200, urea 20 mg/dl, creatinine 0.9mg/dl, random blood sugar 136 mg /dl. ECG was done in the emergency department which was normal

Patient was given antiemetics and proton pump inhibitors. However, patient didn't improve. ECG was repeated which showed dynamic ECG changes in leads V1 to V6 (Figure 2). Possibility of ACS in the form of unstable angina was kept and patient was admitted to cardiac care unit and treatment of ACS started.

Patient remained stable the next day and repeat troponin T was also negative. Treatment for unstable angina was continued. However, by the evening patient complained of worsening of dizziness and nausea with decreased consciousness. Non contrast computed tomography was done, which revealed SAH and intraventricular haemorrhage (Figure 3). Treatment for unstable angina was stopped. Patient was then advised neurosurgery consultation and improved with conservative management and had no complaints during follow-up visit.

SAH associated with ECG changes are seen in many studies but serial ECG changes associated with SAH are rarely seen making this case more interesting.



Figure 1: ECG of Patient.



Figure 2: Dynamic ECG changes in leads V1 to V6.

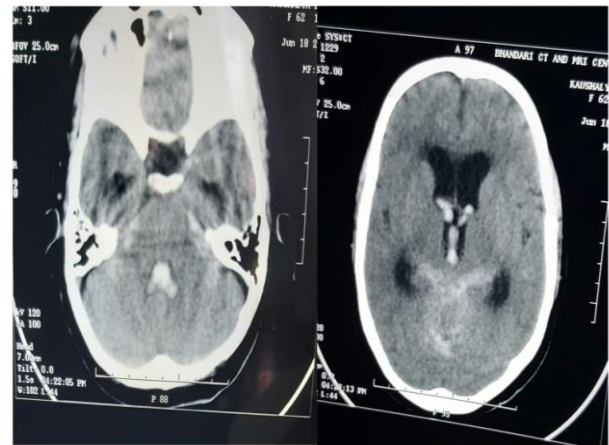


Figure 3: SAH and intraventricular haemorrhage.

DISCUSSION

SAH is a medical emergency caused by bleeding into the subarachnoid space. Symptoms caused by SAH are headache, nausea, vomiting, loss of consciousness and seizure.³

The association of ECG changes in SAH has been known since long. Spontaneous SAH is also a common neurological disorder that leads to out-of-hospital cardiac arrest.⁴

The reported prevalence of ECG changes in patients of SAH ranges from 27%-100%.⁵ Electrocardiographic changes may include QT prolongation, Q waves, cardiac dysrhythmias, ST and T wave changes.

There are two mechanisms that might mediate ECG changes in patients with SAH, i.e. autonomic neural stimulation from the hypothalamus or elevated levels of circulating catecholamine.⁶ Hypothalamic stimulation may cause ECG changes without associated myocardial damage whereas elevated catecholamine levels have been correlated with QT-interval prolongation and myocardial damage.

SAH patients have often been misdiagnosed to have cardiac abnormalities based on their ECGs when in many of those instances the ECG change had been the result of the SAH itself. Beard et al diagnosed myocardial ischemia in a 37-year-old female and started anticoagulants.⁷ There was only short-term history of syncope and stupor and later on ruptured aneurysm was found on autopsy.

ECG changes in SAH are challenging to differentiate from ACS so any patient who present with nonspecific ECG changes SAH may also be kept as differential diagnosis. Because most of the ECG abnormalities that occur in SAH are benign and reversible, differentiating true myocardial infarction from benign changes is important. Two-dimensional echocardiography is more

sensitive in detecting myocardial ischemia than ECG and thus will be more helpful.

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