

Subarachnoid Hemorrhage Mimicking Myocardial Infarction

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Citation

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Abstract

Electrocardiographic changes due to subarachnoid hemorrhage (SAH) are seen frequently and this can lead to erroneous examinations and treatment, like thrombolytic or antiaggregant, which can increase the mortality. A 42-year-old female was admitted to our emergency. While planning with primary percutan transluminal coronary angioplasty (PTCA) the ECG changed to narrow QRS complex supraventricular tachycardia. Due to the normal echocardiography of the heart, lack of coronary artery disease medical history, physical examination was made again and we decided to take a non-contrast cranial computer tomography(CT) to exclude intracranial hemorrhage which could explain the electrocardiographic changes and clinical situation of the patient We presented a case with SAH whose electrocardiograph mimicked myocardial infarction.

INTRODUCTION

Subarachnoid haemorrhage accounts for only 5% of strokes, but it generally occur in the young age. Hypertension, hypoxaemia, and electrocardiographic (ECG) changes, which can mimic acute myocardial infarction and lead to erroneous examinations and treatment are associated with subarachnoid haemorrhage in the acute phase⁽¹⁾.

Electrocardiographic changes can be responsible for life-threatening arrhythmias. They are found responsible for 8 to 15% prehospital mortality rate of patients with SAH. Of deaths within the first 24 hours after SAH, 75% occur suddenly and are presumed also to be cardiac in origin⁽²⁾.

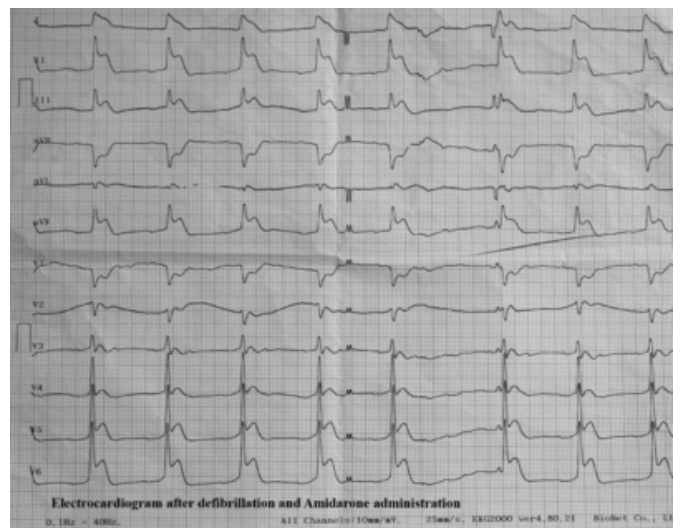
Aim: We presented a case with SAH, who mimicked myocardial infarction, to avoid misdiagnoses and thrombolytic therapy.

CASE REPORT

A 42-year-old female was admitted to our emergency service by paramedics with asystole rhythm. After 10 minute cardiopulmonary resuscitation with intermittently epinephrine administration (3mg total) cardiac rhythm turned to torsades de pointes. She was defibrillated with 200j (biphasic defibrillator) and also 1gr MgSO₄, and 300mg amiodarone were administered intravenously. The ECG changed to third degree AV block, 3 mm ST-segment elevation in DI, aVL, DII, DIII and aVF limbs and ST segment depression, T wave negativity in V1 and ST-segment elevation in V2-6 precordial limbs (Figure 1).

Figure 1

Figure-1. The ECG demonstrated to third degree AV block and ST-segment elevation in leads DI, DII, DIII, aVL, aVF and V.



The patient's blood pressure raised to 80/60mmHg, and pulse beat rate was 40/min. The patient was diagnosed as anterolateral and inferior myocardial infarction, based on the results of ECG, and cardiology consultation was requested. For her third degree AV block 1mg atropin was administered with intervals (total 3mg) and her ECG returned to normal sinus rhythm, with a 130/90mmHg blood pressure, 150 beats per minute pulse rate and SPO₂: 90%. The patient's pathologic laboratory results were as follows; glucose 346mg/dl, Ca: 7.8 mg/dL, Troponin: 0.233ng/ml. While planning with primary percutan transluminal coronary

angioplasty (PTCA) the ECG changed to narrow QRS complex supraventricular tachycardia. Due to the normal echocardiography of the heart, lack of coronary artery disease medical history, physical examination was made again and we decided to take a non-contrast cranial computer tomography(CT) to exclude intracranial hemorrhage which could explain the electrocardiographic changes and clinical situation of the patient. Subarachnoid hemorrhage had been revealed with cranial CT (Figure 2).

Figure 2

Figure-2. Cranial CT shows hemorrhage in subarachnoid space.



During the planning time for hospitalisation to neurosurgery intensive unit, cardiopulmonary arrest occurred again. The patient failed to respond cardiopulmonary resuscitation.

DISCUSSION

Electrocardiographic changes due to SAH were analysed in the literature. Autonomic neural stimulation from the hypothalamus or elevated circulating catecholamines are held responsible for this changes. Hypothalamic stimulation may cause ECG changes without associated myocardial damage, whereas elevated catecholamines may result in myocardial damage^(3,4). Oppenheimer⁽⁵⁾ reported that pathologies within the left insular cortex produce ST-segment depression, QT prolongation, third degree heart block, like our case, and culminating in death in asystole. Electrocardiographic changes are approximately seen in %50 of the patients with subarachnoid hemorrhage. Although this

suggests a myocardial damage, no macroscopic evidence of injury is demonstrated in a postmortem study. The possible changes are prolongation or shortening of PR distance, pathological Q wave, ST-segment elevation or depression, prolongation or shortening of QT, arised U waves, sinus bradycardias or tachycardias, supraventricular tachycardias, and life-threatening ventricular fibrillation, torsades de pointes, ventricular tachycardia may be present⁽⁶⁻⁸⁾. Brouwers et al. revealed that this changes can mimic myocardial ischemia or infarctions. Also they found supraventricular and ventricular arrhythmias which can threat the patients life in the first hours⁽⁹⁾. In about 3% of patients, cardiac arrest occurs at onset of the subarachnoidal haemorrhage due to a fatal cardiac rhythm; resuscitation is essential, because half the survivors regain independent existence⁽²⁾. Also there are reports that SAH patients are misdiagnosed and treated as myocardial infarction⁽¹⁰⁾. Musuraca et al reported with a case that there are pitfalls for prehospital thrombolysis among the patients with intracranial haemorrhage due to this electrocardiographic changes⁽¹¹⁾.

CONCLUSION

This case report showed us that all physicians, especially emergency physicians, should always be aware of cardiac manifestations of acute cerebrovascular events such as SAH. Patients who were admitted with ST-segment elevation should not diagnosed immediately as acute coronary syndrome and differential diagnosis must be made particularly among patients who are unconscious without chest pain and headache.

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