

## Subarachnoid hemorrhage mimicking acute myocardial infarction: case presentation

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**Abstract:** We present a patient with a subarachnoid hemorrhage (SAH) on the computed tomography and electrocardiogram changes compatible with myocardial infarction. SAH is a medical emergency, but it can be initially misdiagnosed. Diagnosis may be delayed due to atypical presentations of SAH and this may endanger the life of the patient. Electrocardiogram abnormalities have been described previously in SAH and may obscure the correct diagnosis.

**Key words:** subarachnoid hemorrhage - SAH, acute myocardial infarction

### Introduction

Subarachnoid hemorrhage (SAH) represents a life-threatening medical emergency; therefore patients suffering from it are generally admitted to an intensive care unit (1, 4, 7). With a worldwide incidence of 9 per 100.000 persons per year, this condition has an average mortality of 51%, with one third of survivors needing lifelong care (1, 4). Although approximately 70% of deaths are due to direct complications of SAH, such as primary bleeding (about 15% die at the time of the ictus), rebleeding and vasospasm; up to 23% of deaths are mainly caused by cardiopulmonary complications (1, 4, 7, 16).

Cardiac dysfunction has been frequently described in conjunction with intracranial pathologies, such as ischemic stroke,

meningitis and epilepsy. Yet, SAH is the most common intracranial condition associated with cardiac abnormalities (1, 2, 5, 6, 10-12, 16, 18). The mechanisms of these abnormalities are not yet completely understood, but they are thought to be related to the autonomic imbalance, in particular to direct autonomic discharge to the heart or to increased levels of circulating catecholamines induced by the intracranial hemorrhage (1, 2, 4-9, 12). Injury in the insular area has particularly been noted to be associated with ECG abnormalities (2, 5, 6, 8, 10). The absence of significant coronary lesions in such patients has been demonstrated by coronary angiography (1, 4, 17). Byer - cited by (1, 3) is the first to describe ECG abnormalities associated with cerebrovascular diseases, since 1947. It has been estimated that around 50% to 100% of patients suffering from

SAH present ECG abnormalities, including T wave abnormalities, peaked P waves, prolonged QTc (greater than 440 ms), ST-segment elevations/depressions and in some cases, even Q waves (1, 3, 4, 6, 8, 10, 12).

Prolongation of QT interval is one of the most common changes during SAH (1, 2).

These changes are often observed in patients without a known preexisting heart disease, therefore they represent a real challenge to cardiologists, anesthetists and intensivists, since the variety of these ST/T changes during SAH are indistinguishable from those identified during acute myocardial ischemia or injury (1, 2-4, 10, 16). A misdiagnosis may lead to unfortunate consequences, since treatment of myocardial ischemia is aimed at reperfusion of the myocardium with double antiplatelet therapy, anticoagulants, with or without acute coronary angioplasty or fibrinolytic drugs (2, 10, 16).

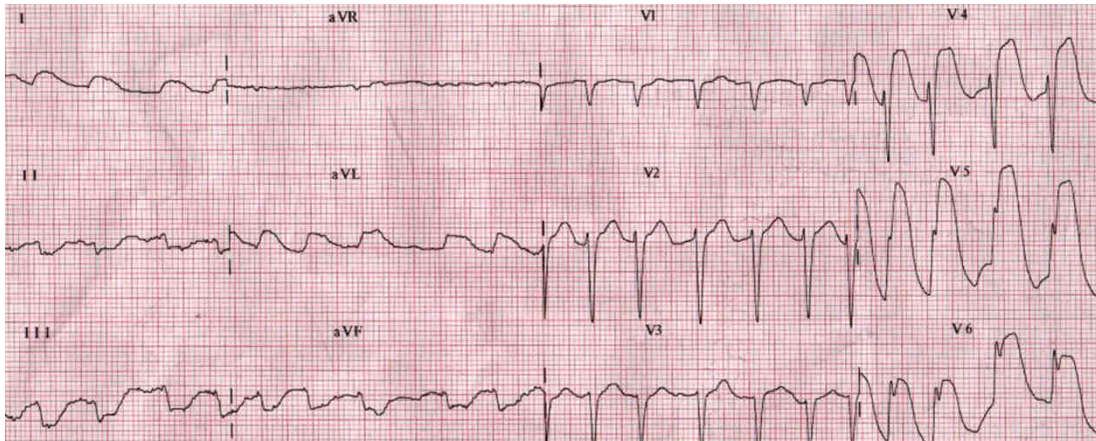
### **Case presentation**

A 80-year-old-man, with a medical history of hypertension, type 2 diabetes mellitus and dementia, was found drowsy by his family. At the emergency department of the first hospital he was brought to, neurological examination showed a Glasgow Coma Scale of 8 points (E2M5V1), with normal isochoric pupils and bilateral reactive light reflexes. He had spontaneous breathing with no need of intubation or ventilatory support.

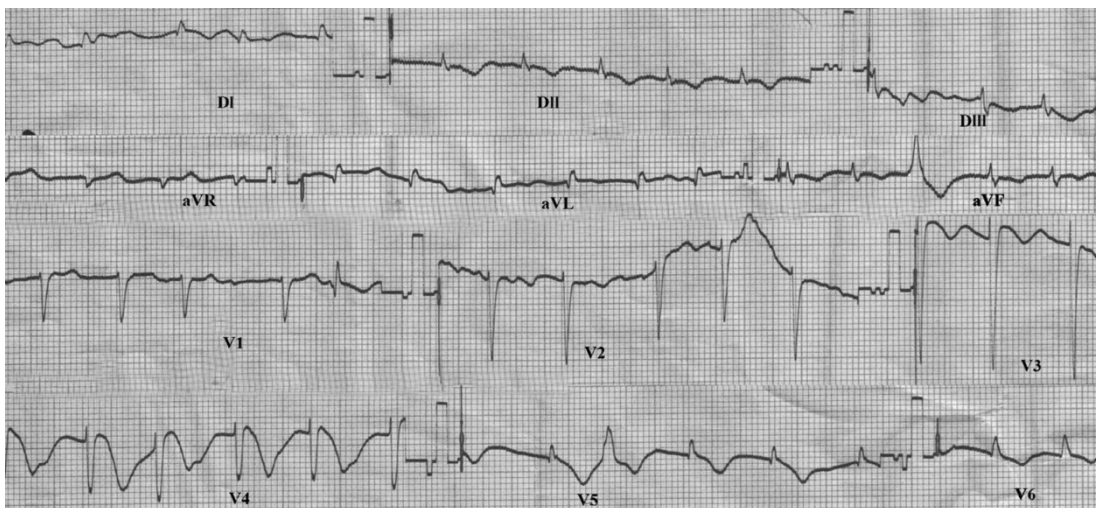
His physical examination revealed a heart rate of 70 beats per minute and a blood pressure of 110/65 mmHg.

His first ECG (Figure 1) showed atrial fibrillation and ST elevation in lateral leads D I, aVL, V 4-V 6, with ST depression in leads D II, D III, aVF, which indicated an acute antero-lateral myocardial infarction. Next, an echocardiography was performed, showing diffuse hypokinesis of the left ventricle, with an ejection fraction of 40%. His cTnI value was 1.9 ng/ml (normal value < 0.4 ng/ml). Creatinine and serum potassium were within normal range (1,1 mg/dl and 4,2 mmol/l respectively). Based on the abnormalities seen on the patient's ECG, the result of the echocardiography and his cTnI value, he was diagnosed with acute antero-lateral myocardial infarction. An emergency coronary angiography was performed but there was no significant stenosis. Next, an emergency cerebral CT - scan was done. It revealed the presence of SAH and a 5 mm subdural hematoma, without the need of an urgent neurosurgical intervention.

A few hours later, the patient's family decided to transfer him at the Emergency University Hospital in Bucharest. Here a cerebral angiography could not be performed. During the patient's follow-up his left ventricle ejection fraction improved (45-50%) and his cTnI value decreased (0,9 ng/ml on day 4). Moreover his ECG traces improved (Fig. 2) and negative/biphasic T waves appeared (day 4). A control cerebral CT - scan showed some resorption of the intracranial blood. Unfortunately, the patient died the fifth day following a respiratory arrest which could not be resuscitated. The family refused the necropsy.



**Figure 1** - Atrial fibrillation: D I, aVL, V4-V6 ST elevation; D II, D III, aVF ST depression



**Figure 2** - ST elevations in ECG traces improved and negative/biphasic T waves appeared (after 4 days)

**Discussion**

Although electrocardiographic abnormalities are not uncommon in patients with cerebrovascular disease including subarachnoid hemorrhage, subdural hematoma and ischemic stroke, typical ST - segment elevation is rare. Up to date, there are only a few cases of intracranial pathologies

mimicking acute myocardial infarction described in the literature (13-17).

As described earlier, the physiopathology is still not known. There are two mechanisms thought to produce such ECG changes in patients with cerebral lesions: autonomic neural stimulation from the hypothalamus/insula and/or elevated circulating catecholamines. Hypothalamic

stimulation might cause ECG changes without associated myocardial damage, whereas elevated circulating catecholamines might induce tachycardia, coronary vasospasm and direct myocardial toxicity due to increased intracellular calcium (1-5)(8)(9).

In the present case, the coronary angiography excluded an acute myocardial infarction. Thus the left ventricular dysfunction seen on echocardiography may have a toxic effect upon the myocardium leading to contractile dysfunction, necrosis and apoptosis.

During a follow-up period, the cardiovascular status of the patient showed almost complete recovery. Serial ECG revealed resolution of the ST elevations, repeated echocardiography demonstrated quasi-normal left ventricular function. Reversible left ventricular systolic dysfunction associated with intracranial hemorrhage may be due the neurogenic stunned myocardium.

The present case tries to emphasize the fact that there may be diagnostic problems in differentiating between intracranial hemorrhage and acute myocardial infarction among unconscious patients. Differentiating myocardial infarction from neurogenic left ventricular dysfunction associated with intracranial hemorrhage is very important for initiating appropriate therapy for the relevant condition. Antiplatelet, anticoagulant and fibrinolytic drugs are essential in STEMI treatment, while they are contraindicated in intracerebral hemorrhages.

In conclusion, doctors should always be aware of the cardiac manifestations of acute

cerebrovascular events such as SAH, especially in unconscious patients, where coronary angiography might prove extremely important in the differential diagnosis.

## References

- 1.Okabe T., Kanzaria M., et al. - Cardiovascular Protection to Improve Clinical Outcomes After Subarachnoid Hemorrhage: Is There a Proven Role?, *Neurocrit Care* 2013, 18, 271-284
- 2.Takeuci S., Nagatani K. et al. - Electrocardiograph abnormalities in intracerebral hemorrhage, *Journal of Clinical Neuroscience* 2015, 22, 1959-1962
- 3.Sakr Y.L., Lim N. et al. - Relation of ECG changes to neurological outcome in patients with aneurysmal subarachnoid hemorrhage, *International Journal of Cardiology* 2004, 96, 369-373
- 4.Urbaniak K., Merchant A.I. et al. - Cardiac complications after aneurysmal subarachnoid hemorrhage. *Surgical Neurology* 2007, 67, 21-29
- 5.Juntilla E., Vaara M. et al. - Repolarization Abnormalities in Patients with Subarachnoid and Intracerebral Hemorrhage: Predisposing Factors and Association with Outcome. *Neuroscience in Anesthesiology and Perioperative Medicine* 2012, 116 (1), 190-197
- 6.Huang C.C., Huang C.H. et al. - The 12-lead electrocardiogram in patients with subarachnoid hemorrhage: early risk prognostication. *The American Journal of Emergency Medicine* 2012, 30, 732-736
- 7.Diringer M.N., Citerio G. - Understanding the disease: aneurysmal subarachnoid hemorrhage. *Intensive Care Med* 2014, 40, 1940-1943
- 8.Chen W-L., Huang C-H. et al. - ECG abnormalities predict neurogenic pulmonary edema in patients with subarachnoid hemorrhage, *American Journal of Emergency Medicine* 2016, 34, 79-82
- 9.Schuiling W.J., Algra A. et al. - ECG abnormalities in predicting secondary cerebral ischemia after subarachnoid haemorrhage, *Acta Neurochirurgica* 2006, 148, 853-858
- 10.Van Bree M.D.R., Roos Y.B.W.E.M. et al. - Prevalence and Characterization of ECG Abnormalities After Intracerebral Hemorrhage, *Neurocrit Care* 2010, 12, 50-55

- 11.Crago E., Kerris K. et al. - Cardiac Abnormalities After Aneurysmal Subarachnoid Hemorrhage: Effects of  $\beta$ -Blockers and Angiotensin-Converting Enzyme Inhibitors, *Am J Crit Care* 2014, 23(1), 30-39
- 12.Brouwers P.J.A.M., Wijdicks E.F.M. et al. - Serial Electrocardiographic Recording in Aneurysmal Subarachnoid Hemorrhage, *Stroke* 1989, 20, 1162-1167
- 13.Van der Bilt I. A. C., Raaphorst J. et al. - Ischemic stroke mimicking acute myocardial infarction, a diagnostic dilemma, *International Journal of Cardiology* 2006, 109, 127-128
- 14.Hamilton J.C., Korn-Naveh L., Crago E.A. - Case Studies in Cardiac Dysfunction After Acute Aneurysmal Subarachnoid Hemorrhage, *Journal of Neuroscience Nursing* 2008, 5 (40), 269-274
- 15.Heo W.J., Kang J.H. et al. - Subarachnoid Hemorrhage Misdiagnosed as an Acute ST Elevation Myocardial Infarction, *Korean Circ J* 2012, 42, 216-219
- 16.Benninger F., Raphaeli G., Steine I. - Subarachnoid hemorrhage mimicking myocardial infarction, *Journal of Clinical Neuroscience* 2015, 22, 1981-1982
- 17.Köklü E., Yüksel I. Ö. et al. - Subarachnoid hemorrhage that electrocardiographically mimics acute coronary syndrome: a case report, *Türk Kardiyol Dern Ars* 2015, 43(8), 730-733