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Subarachnoid haemorrhage imitating acute coronary syndrome as a cause of out-of-hospital cardiac arrest – case report

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Abstract

Background: Severe subarachnoid haemorrhage (SAH) is a common cause of cardiac arrest. The survival of patients with out-of-hospital cardiac arrest (OHCA) due to SAH is extremely poor. Electrocardiographic and echocardiographic changes associated with SAH may mimic changes caused by acute coronary syndromes (ACS) and thus lead to delayed treatment of the primary disease. Misdiagnosed SAH due to ACS mask can have an influence on patient outcomes.
Case report: A 47-year-old man presented with a history of out-of-hospital cardiac arrest due to asystole. He had a medical history of hypertension, smoking, and a diffuse, severe headache for one week. The ECG showed atrial fibrillation, 0,2 mV ST-segment elevation in leads aVR and V1-V3 and 0.2 mV ST-segment depression in leads I, II, aVL and V4-V6. Echocardiography revealed left ventricular function impairment (ejection fraction < 20%). The CK-MB activity was 98 U L^{-1} and the troponin I concentration was 0.59 µg L^{-1}. ACS was suspected. Coronarography did not reveal any changes in the coronary arteries. An urgent CT of the head was arranged and showed an extensive SAH.
Conclusion: It appears that an urgent CT of the head is the most effective method for the early identification of SAH-induced OHCA, especially in patients with prodromal headache, no history of the symptoms of ACS and CA due to asystole/pulseless electrical activity (PEA).

Key words: cardiac arrest, subarachnoid haemorrhage, acute coronary syndrome

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Out-of-hospital cardiac arrest (OHCA) predominantly develops due to acute coronary syndrome (ACS). Extra-cardiac causes, e.g., subarachnoid haemorrhage (SAH), are less common. The purpose of the present case report was to describe a patient with OHCA due to subarachnoid haemorrhage imitating acute coronary syndrome.

CASE REPORT

A 47-year-old patient with a history of arterial hypertension and smoking complaining of an increasing, diffuse compression/throbbing headache persisting for 7 days, experienced nocturnal, out-of-hospital sudden cardiac arrest (OHCA) due to asystole. Cardiopulmonary resuscitation was performed by the witnesses to the incident and continued by paramedics for 30 minutes, according to the current standards. Spontaneous circulation was restored, and the patient was transferred to the Department of Cardiology with the suspicion of acute coronary syndrome (ACS) due to electrocardiographic ischaemic changes. On admission, the patient was mechanically ventilated and sedated (midazolam and fentanyl). The features of cardiogenic shock were observed with atrial fibrillation (a ventricular rate of approximately 150 min^{-1}) and ST elevation in leads aVR and V1-V3, as well as 0.2 mV ST depression in leads I, II, aVL and V4-V6 leads (Fig. 1). The significant laboratory abnormalities demonstrated an increased CK-MB activity (98 U L^{-1}) and an increased troponin I level (0.59 µg L^{-1}). The first ECG revealed generalised left ventricular hypokinesis with an ejection fraction of approximately 20% (Figs 2, 3).

Considering haemodynamically significant atrial fibrillation, electrical cardioversion was performed. A continuous infusion of pressor amines was initiated (noradrenaline and dobutamine). The ECG after electrical cardioversion demonstrated a sinus rhythm of 112 min^{-1} and 0.1 mV ST

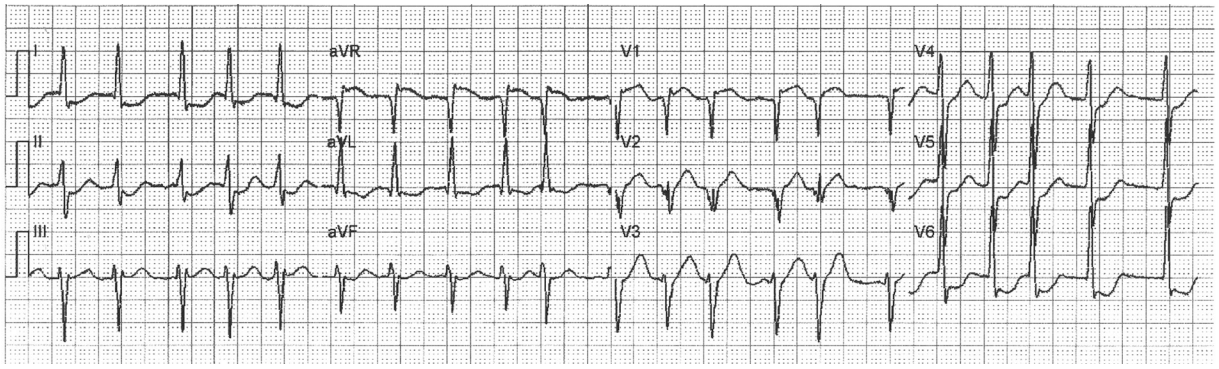

Figure 1. Electrocardiogram

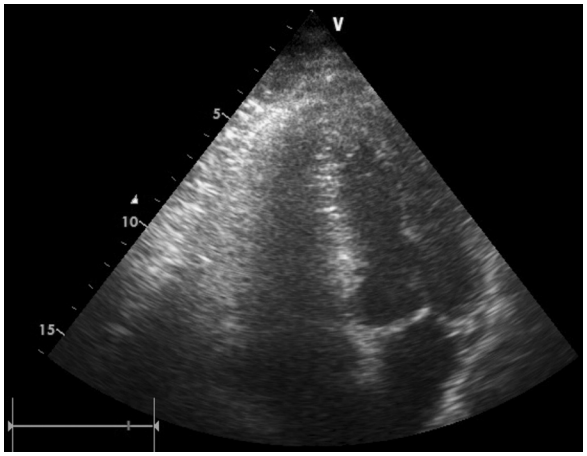

Figure 2. Four-cavity projection during systole

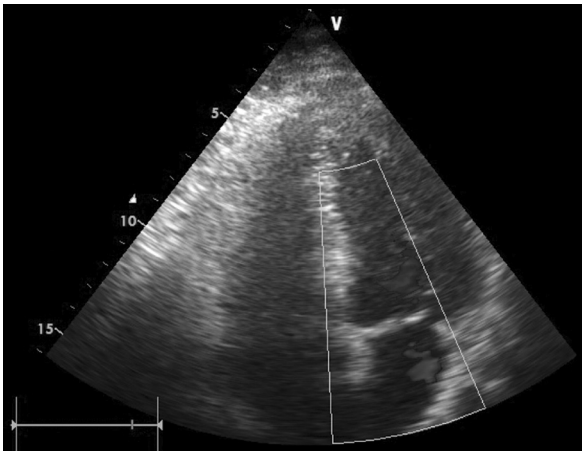

Figure 4. Four-cavity projection during systole

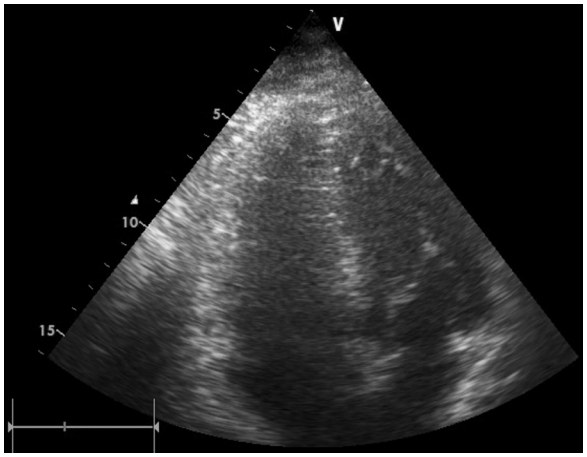

Figure 3. Four-cavity projection during diastole

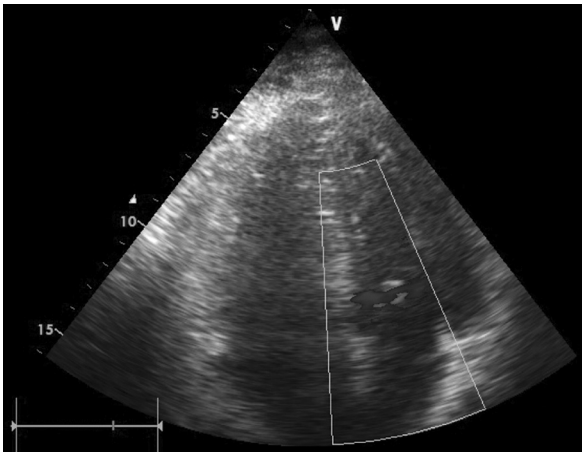

Figure 5. Four-cavity projection during diastole

depression in leads I, II and V4-V6. Repeated echocardiography demonstrated generalised left ventricular hypokinesis with an ejection fraction of approximately 40% (Figs 4, 5). Once the patient's general condition was stabilised, urgent coronarography was performed, which did not reveal pathological changes in the coronary vessels.

Several hours later, the patient developed a gastrointestinal (GI) haemorrhage. Endoscopy of the upper GI tract was

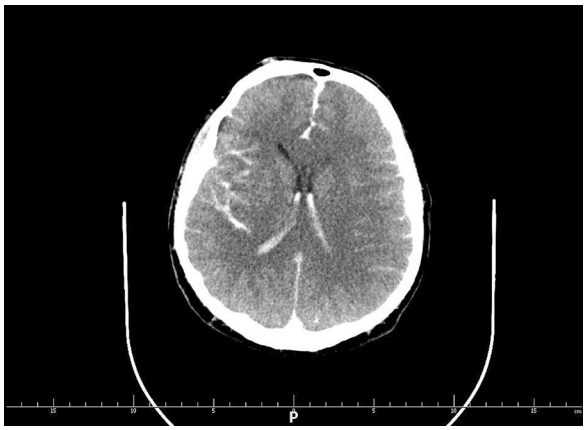

Figure 6. CT scan of the head

carried out which did not show the source of the haemorrhage, yet revealed erosive lesions in the gastric cardia and inflammation of the duodenal bulb. Because the patient's condition was grave, he was transferred to the Intensive Care Unit (ICU). Before admission, a CT of the head was performed which demonstrated a massive subarachnoid haemorrhage with cerebral oedema, corresponding to a grade 4 haemorrhage according to the Fisher scale (Fig. 6). On admission, the patient was unconscious with a GCS score of 3 (sedation was no longer used). His circulation was inefficient, supported by the infusion of pressor amines. The neurological examination showed total areflexia. The patient was disqualified from neurosurgical intervention. An angio-CT was performed followed by cerebral angiography, which demonstrated lack of cerebral blood flow. After the legally binding tests were made, the patient was collectively pronounced dead.

DISCUSSION

The failure of one of the heart functions (electrical or mechanical), or lack of their coupling, leads to sudden cardiac arrest caused by ventricular fibrillation, pulseless ventricular bradycardia, asystole or pulseless electrical activity. The most common rhythm mimicking out-of-hospital cardiac arrest is VF/VT (51%, 43%, respectively) [1, 2]. According to the Utstein-style examination, the primary (cardiac) aetiological cause is observed most commonly, accounting for 73% of cases [3]. A newer study, supplemented with CT, demonstrated that an extra-cardiac cause is more common (62.5%) [4].

The most common primary cause of OHCA is acute coronary syndrome [4]. According to the guidelines of the Polish Cardiac Society, acute coronary syndrome is diagnosed based on increased and/or decreased concentrations of a cardiac biomarker (preferably cardiac troponin — cTn), with at least one of the values above the 99th centile of the upper reference limit, or at least one of the following: ischaemic symptoms, new or possibly new significant ST-T changes, new left bundle branch block (LBBB), pathological Q waves, new loss of the viable myocardium, new segmental contractility impairments on imaging examinations, or angiography- or autopsy-revealed coronary artery clots [5]. In patients with ST elevation in ECG recordings undergoing cardiopulmonary resuscitation, the treatment of choice is urgent coronarography with the intent to perform revascularisation [6].

The secondary causes of OHCA include acute aortic dissection, pulmonary embolism, hypoxia due to pneumonia, asthma and exacerbation of COPD, cerebrovascular diseases, malignant neoplasms, constricted airways and exogenous causes [4]. One of the vascular diseases of the brain is subarachnoid haemorrhage (SAH); 85% of SAH cases are caused by aneurysmal SAH, 10% by non-aneurysmal perimesencephalic haemorrhage, and the remaining 5% by other rare causes [7]. In the majority of cases, cardiac arrest secondary to SAH is caused by a ruptured aneurysm [8], as in our case. The incidence of SAH-induced sudden cardiac arrest amongst all of the cases of out-of-hospital cardiac arrests ranges between 4 and 8% in patients who died before admission and between 4–18% in those who were admitted. The survival to discharge of these patients is 0–2% [9]. Thus, SAH as a cause of OHCA is a serious problem.

ECG changes in patients with SAH are common and occur in 27–100% of cases. They can be both disorders of cardiac repolarisation and arrhythmias. The former often resemble those observed in ACS (ST-segment and T-wave morphology abnormalities) [10]. Echocardiography shows changes meeting the criteria of the Tako-Tsubo syndrome, also called apical balloting syndrome or stress-induced cardiomyopathy. The above criteria include transient akinesis or dyskinesis of the central left ventricle with or without apex involvement extending over the region of vascularisation of more than one coronary artery; the stressogenic factor is common, yet not always present. The other criteria are as follows: lack of significantly constricted coronary vessels or angiographic features of atheromatous plaque rupture, new ECG changes (ST segment elevation or negative T waves), or slightly increased concentrations of cardiac troponins. The differential diagnosis of this syndrome also includes pheochromocytoma and myocarditis. All four criteria must be present to establish the diagnosis.

The ECG and echocardiographic changes observed in our patient suggested ACS. The standard management in such cases is reperfusion therapy, which requires anticoagulant and antiplatelet drugs. The therapy is associated with an increased risk of bleeding and is contraindicated in patients diagnosed with SAH as an underlying disease. When the ECG and echocardiographic abnormalities in patients with SAH have been misinterpreted ACS, the treatment of

the underlying disease is likely to be improper, hence harmful and delayed. Moreover, in cases of concomitant SAH and AE, coronary intervention after total body heparinisation is contraindicated.

Determination of cTN concentrations and echocardiography can be useful for differentiating between SAH and AE. In 15% of patients with SAH, a left ventricular ejection fraction below 50% was found, whereas in 13% of such cases, the segmental disorders of left ventricular contractility were observed. The segmental changes often do not correlate with the region supplying the coronary arteries [12]. Echocardiography that is markedly inconsistent with ECG findings suggests SAH-related left ventricular dysfunction (stunned myocardium — SM)**.** The concentrations of cTn < 2.8 μg L^{-1} in patients with ejection fraction below 40% are characteristic of SM [13]. The other valuable diagnostic elements include the CA mechanism and presence of headache, or pre-SCA history of other neurological symptoms. In the population of patients with SAH, the most common cause of SCA is pulseless electrical activity (PEA) (43-63%)/asystole (44–57%). Ventricular fibrillation is rare (0–18% of cases) [10]. Headache is an axial symptom of SAH, which is not a typical symptom preceding OHCA and affects only 5% of individuals with OHCA. The majority of those with headache preceding cardiac arrest develop intracranial haemorrhage. However, 50% of patients with OHCA secondary to intracranial haemorrhage do not report headaches; therefore, a lack of headache is not necessarily indicative of the absence of intracranial haemorrhage as a cause of OHCA [14].

Based on findings in the literature and the case described, it should be emphasised that the differential diagnosis of acute coronary syndrome requires increased vigilance of clinicians while making decisions about diagnostic and therapeutic management. The symptoms found on physical examination preceding sudden cardiac arrest and its mechanism can be valuable and induce the clinician to perform suitable additional examinations to confirm the diagnosis of the underlying disease and to institute specific therapy. In order to diagnose SAH in patients with OHCA as quickly as possible, an urgent CT of the head seems most effective, especially in patients with headaches, without pre-CA history of symptoms of acute coronary syndrome (ACS) and asystole/PEA as the mechanism of sudden cardiac arrest. Moreover, it should be remembered that the absence of headache is not a contraindication for CT examination.

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