SUBARACHNOID HEMORRHAGE ASSOCIATED WITH ACUTE MYOCARDIAL INFARCTION AND SUDDEN DEATH

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Abstract

We describe a patient presenting as sudden death with electrocardiographic evidence of transmural myocardial infarction. Cardiac enzymes were elevated and there was severe localized hypokinesis on echocardiography. Brain CT showed subarachnoid hemorrhage. We believe that in subarachnoid hemorrhage, there is concurrent myocardial injury and dysfunction producing acute myocardial infarction. Simultaneous monitoring of both cardiovascular and neurological conditions is recommended.

Key words: Subarachnoid hemorrhage, Sudden death, Myocardial infarction

Electrocardiographic abnormalities have long been reported in patients with central nervous system disorders.[1] Indeed electrocardiographic changes simulating an evolving acute myocardial infarction have been observed in patients with spontaneous subarachnoid hemorrhage (SAH).[2] Moreover, SAH is frequently complicated by cardiopulmonary episodes, including sudden death.[3] In this report, we describe a patient who presented to our emergency room in sudden death, with the subsequent electrocardiographic changes of an evolving acute transmural anterior wall myocardial infarction occurring in association with a SAH.

Case Report

A 52-year-old man with no history of heart disease was found comatose and brought to the emergency room. On arrival, no vital signs were detected. Cardiopulmonary rescusitation was immediately performed and he was subsequently transferred to the intensive care unit.

At the intensive care unit, he was comatose and on mechanical ventilation. The temperature was 36°C, the pulse rate was 136/min and the blood pressure was 120/77 mmHg. The head and neck were normal, with no nuchal rigidity. There were no signs of head trauma. The breathing sound was coarse with bilateral basal rhonchi. Heart rhythm was regular and a grade 2/6 systolic murmur was heard over the left sternal border. The abdomen was soft. The liver and spleen were not felt, and no masses were detected. The peripheral pulses were intact. There was no peripheral edema. There was no Babinski sign.

The electrocardiogram revealed signs of an evolving Q wave myocardial infarction involving the anterior and lateral walls (Figure 1, top). An echocardiogram demonstrated hypokinesis over

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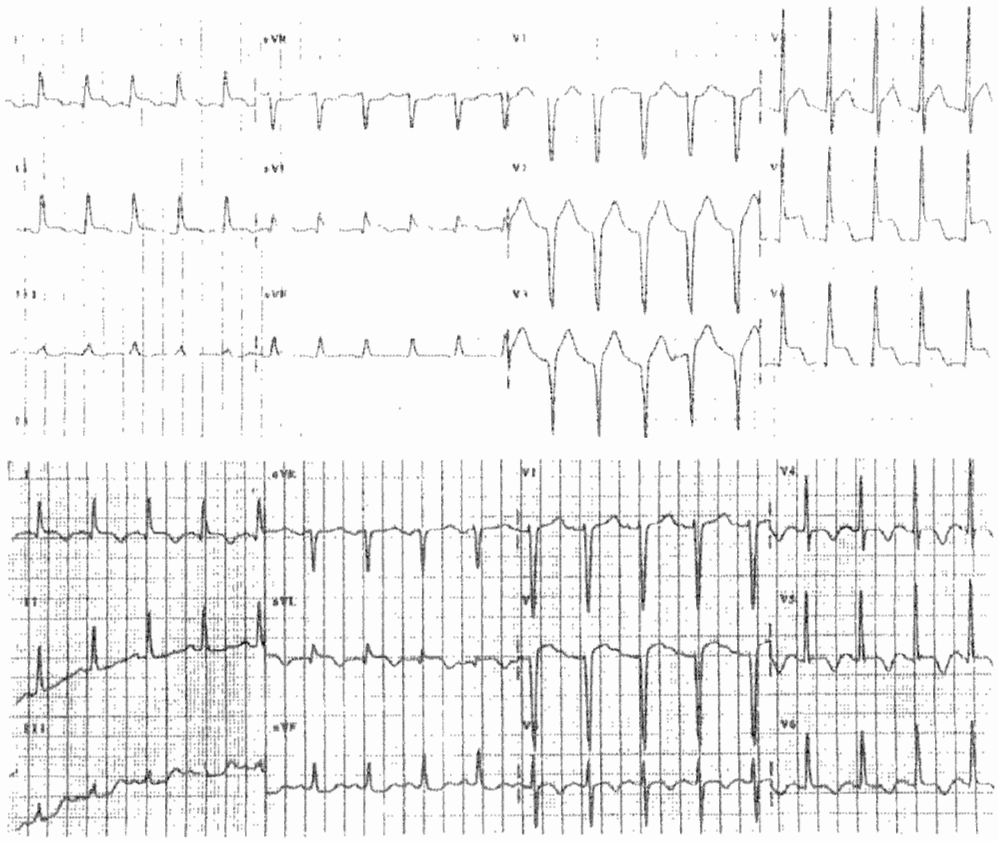

Fig. 1. Top: Electrocardiogram at admission showing sinus tachycardia with deep Q waves in leads V1 to 3 and ST segment elevation in leads I, aVL and V1 to V6 with reciprocal ST segment depression in leads II, III and aVF suggesting acute transmural anterior myocardial infarction. Bottom: Electrocardiogram 1 day later showing sinus tachycardia with declining ST elevation.

the anterior wall with severely impaired ventricular systolic function (ejection fraction 27%). Creatine kinase and CKMB were 76 and 48 IU/liter on admission; 119 and 44 IU/liter at 6 hours; and at 12 hours, 158 and 13 IU/liter, respectively. Troponin was positive (8.8 ug/ml).

In the second hospital day, the electrocardiogram showed sinus tachycardia with promptly declining ST elevation (Figure 1, bottom). A computed tomography (CT) scan of the brain was consistent with a subarachnoid hemorrhage. The patient was treated conservatively. His clinical course was punctuated by recurrent hypotension unresponsive to increasing doses of inotropic agents and worsening neurologic status. Five days after admission, the patient died in electromechanical dissociation.

Discussion

It is well known that cardiac arrhythmias and sudden death are most frequently caused by preexisting heart disease. However, they may also be a first symptom of an acute neurological event.[4] Our patient with SAH collapsed in ventricular fibrillation. It has been reported that life-threatening arrhythmias such as ventricular tachycardia or ventricular fibrillation resulted from myocardial damage due to catecholamine release secondary to an extraordinary transient enhancement of sympathetic nervous system in patients with SAH.[3] On the other hand, our

patient had no history of chest discomfort nor heart disease. There were no signs of head trauma. No abrasion, bruising or hematoma were found upon admission. Therefore, the possibility of acute myocardial infarction complicated by ventricular fibrillation and head trauma producing SAH was less likely.

Following resesuitation, our patient presented with marked electrocardiographic changes indistinguishable from acute myocardial infarction. Serial electrocardiograms showed the combination of abnormal Q waves and ST segment elevation in leads I, aVL and V1 to 6, with reciprocal ST segment depression in leads II, III and aVF, which strongly suggested acute transmural anterior myocardial infarction. It has been reported that the most frequent electrocardiographic abnormalities in the course of SAH were lengthening of QT interval, very negative or positive deep T waves, elevation or depression of the ST segment and the presence of U waves.[5] The arrhythmias were believed to be due to increased sympathetic activity or by a damage of cerebral areas with arrhythmogenic capacity.[6]

Myocardial injury in SAH as evidenced by elevated CKMB has been reported.[3] In our patient, cardiac enzymes were elevated, with increased levels of CK, CKMB and troponin. The pathophysiology of cardiac injury after SAH remains controversial. Animal studies suggested that catecholamine-mediated injury is the most likely cause of myocardial injury after SAH.[3] Postmortem examination in a patient with SAH who presented with an electrocardiogram simulating myocardial infarction revealed widespread focal myocytolysis of the myocardium which was unrelated to vascular distribution but similar to that induced by exogenous catecholamines.[7] Thus, myocardial injury in SAH could be due to neurohumoral damage to the myocardial cells, caused either by focal ischemia from vasoconstriction of the myocardial microcirculation or by a direct toxic effect of catecholamines.

Elevation of troponin is associated with a higher incidence of myocardial dysfunction.[8] In our patient, there was severe hypokinesis of the anterior wall, with an ejection fraction of 27%. Animal studies showed that regional wall motion abnormalities in SAH dogs were not related to myocardial hypoperfusion nor coronary artery spasm.[9] Postmortem examination in a patient with SAH simulating myocardial infarction revealed that the coronary arteries and the myocardium were macroscopically normal.[10] Thus, myocardial stunning may account for the myocardial dysfunction in SAH.

In conclusion, we believe that in SAH, there may be concurrent myocardial injury and dysfunction, thereby producing acute myocardial infarction. A careful simultaneous monitoring is recommended to prevent cardiac complications, as well as to evaluate the neurologic pathology. The possibility of SAH or another central nervous system event needs to be considered in patients with altered mental status.

References

1. Yamour BJ, Sridharan MR, Rice JF, Flowers NC. Electrocardiographic changes in cerebrovascular hemorrhage. Am Heart J 1980;99:294-6.
2. Gascon P, Ley TJ, Toltzis RJ, Bonow RO. Spontaneous subarachnoid hemorrhage simulating acute transmural myocardial infarction. Am Heart J 1983; 105(3):511-3.
3. Sato K, Masuda T, Isumi T. Subarachnoid hemorrhage and myocardial damage: clinical and experimental studies. Jpn Heart J 1999;40(6):683-701.
4. Van der Kleij FG, Henselmans JM, Van de Loosdrecht AA. Cardiac arrhythmia as initial presentation of aneurysmal subarachoid hemorrhage. Netherlands J Med 1999;55(5)242-6.
5. Salvati M, Cosentino F, Artico M et al. Electrocardiographic changes in subarachnoid hemorrhage secondary to cerebral aneurysm. report of 70 cases. Italian J Neurol Sci 1992;13(5):409-13.
6. Martorano PP, Bini G, Tanara L, Sinkovets L, Pelaia P, Pietropaoli P. Subarachnoid hemorrhage and the heart. Minerva Anestesiologica 1998;64(5):231-3.
7. 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 Cir J 1996;60(4):254-7.
8. Parekh N, Venkatesh B, Cross D, Leditschke A, Atharton J, Miles W, Winning A, Claque A, Richard

C. Cardiac troponin I predicts myocardial dysfunction in aneurysmal subarachnoid hemorrhage. JACC 2000;36(4):1328-35.

9. Zaroff JG, Rordorf GA, Titus JS, Newell JB, Nowak NJ, Torchiana DF, Aretz HT, Picard MH, Macdonald RL. Regional myocardial perfusion after experimental subarachnoid hemorrhage. Stroke 2000;31(5):1136-43.
10. Soman P, Senior R. Severe ventricular dysfunction secondary to subarachnoid hemorrhage. Clin Cardiol 1997;20(4):402-3.

蜘蛛膜下出血併急性心肌梗塞及猝死

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摘要

一猝死病人心電圖檢查發現急性心肌梗塞，心肌激酶上升及超音波發現嚴重局部性心臟收縮功能衰竭，腦部電腦斷層掃瞄發現蜘蛛膜下出血。我們相信在蜘蛛膜下出血時，會有心臟傷害及功能異常引致急性心肌梗塞，建議在此類病人需同時觀察其心臟及神經功能狀況。

關鍵詞：蜘蛛膜下出血，急性心肌梗塞，猝死

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