Coronary vasospasm following subarachnoid hemorrhage as a cause of stunned myocardium

Case report

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A patient with subarachnoid hemorrhage was found to have electrocardiographic abnormalities resembling an acute myocardial infarction as well as left ventriculographic findings of cardiac dysfunction. These cardiac abnormalities resolved following surgical clipping of the aneurysm and the patient recovered well from the operation. She died 2 months later from cancer and a postmortem examination at that time revealed no evidence of myocardial necrosis. In this report, the authors discuss coronary vasospasm and reversible postischemic “stunned myocardium,” a condition that has not been considered previously in relation to subarachnoid hemorrhage.

KEY WORDS • subarachnoid hemorrhage • myocardial infarction • coronary vasospasm • stunned myocardium

Electrocardiographic (EKG) abnormalities, which are indistinguishable from those due to an acute myocardial infarction, have occasionally been reported in cases of subarachnoid hemorrhage (SAH). We recently encountered a patient with an SAH who exhibited findings similar to an acute myocardial infarction on EKG examination, as well as findings of anterior wall akinesis on left ventriculography. These cardiac abnormalities recovered. The disorder in our patient seems to correspond to the condition of “stunned myocardium,” which was defined by Braunwald and Kloner in 1982 as reversible postischemic myocardial dysfunction. The cause of the EKG changes resembling an acute myocardial infarction in patients with an SAH remains unknown. Coronary vasospasm is considered to have been the main etiology of the cardiac abnormalities in our patient and is discussed in this report as the possible cause for “stunned myocardium.”

Case Report

This 70-year-old woman was referred to our hospital because of disturbances of consciousness. A diagnosis of maxillary cancer had been made 6 months earlier for which she received radiation therapy.

Examination. Examination conducted 3 hours after onset of symptoms revealed the patient to be disoriented and complaining of severe headache and vomiting. Paraparesis and bilateral pathological Babinski reflexes were noted. Electrocardiography was normal except for a right bundle branch block. Computed tomography (CT) revealed an SAH, and cerebral angiography showed two saccular aneurysms: one arising from the anterior communicating artery and the other at the basilar artery bifurcation.

Surgery for the aneurysms was not performed because of the patient’s age and poor general condition. She did well until the 26th day after presentation, when she suffered a generalized seizure. During the period of recovery from unconsciousness, she complained of chest pain and the electrocardiographic monitor showed ST elevation. An EKG examination conducted after the pain had subsided showed ST-T changes suggestive of an acute myocardial infarction in the anterior leads (Fig. 1). Another EKG examination on the following day showed abnormal Q waves in leads V₅, V₆, and V₇.
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(Fig. 1). Serial determinations of creatine phosphokinase levels along with MB isoenzyme fractions, serum glutamic-pyruvic transaminase, and lactate dehydrogenase values were all within normal limits during the next 4 days. A CT scan obtained shortly after the seizure revealed SAH, mild hydrocephalus, and a hematoma in the fourth ventricle.

Selective coronary arteriography performed 30 days after the SAH for preoperative evaluation purposes did not show any narrowing or spasm of the coronary artery; however, left ventriculography revealed anterior wall akinesis although the overall cardiac function was not severely depressed. A decision was made to operate as an emergency measure regardless of the risks because of a gradual deterioration in the patient's level of consciousness.

Operation. On the 37th day after the onset of SAH, the neck of the aneurysm of the anterior communicating artery was clipped with placement of a ventricular drain. A transient ST elevation lasting a few minutes was observed several times, which was treated by nitroglycerin administration. After aneurysmal clipping, a ventriculoperitoneal shunt was placed, and excellent recovery was achieved.

Postoperative Course. Postoperative EKG examination revealed negative T waves in leads V1 to V6; the abnormal Q wave and ST elevation had returned to normal (Fig. 1). Left ventriculography showed normal left ventricular wall motion, and a nuclear scan using pyrophosphate showed no localized or diffuse increase in tracer uptake by the myocardium.

After a period of rehabilitation, the patient was discharged from the hospital, experiencing no neurological deficits at that time. She was readmitted 2 months later because of the recurrence of cancer and died of pneumonia. An autopsy showed no gross evidence of myocardial infarction and, although the coronary artery was mildly arteriosclerotic, no stenosis or thrombi of the artery could be detected. Microscopic examination of the anterior ventricular wall, which had shown akinesis, revealed no findings of individual myocyte drop-out, no myocyte lysis, none of the characteristic changes of catecholamine overload, and no focal scarring.

Discussion

EKG Abnormalities Following SAH

Electrocardiographic abnormalities are often seen in association with central nervous system disorders, and have been reported with a 70% to 90% frequency in association with SAH. 9,10,14,20 The most common patterns noted are a broad or inverted T wave, a prolonged QT interval, an ST segment elevation or depression, a prominent U wave, and various rhythm abnormalities. 2,4,10 Ischemic EKG changes simulating an acute myocardial infarction are, however, rare. 3,5,7,8 Although reference has occasionally been made to the cause of the pathological Q wave, the etiology still remains unknown.

J. Neurosurg. / Volume 75 / August 1991
Several explanations have been suggested for EKG alterations in SAH, some relating the role of catecholamines, hypercortisolism, or hypokalemia. Certain recent studies have unequivocally demonstrated that catecholamines play the principal role in EKG changes and furthermore that ischemic stimulation of the posterior hypothalamus following an SAH is responsible for an increase in sympathetic tone, which has been implicated as the cause.

Several reports have described a normal myocardium at autopsy, as was seen in our patient. Since the demonstration by Connor that subtle areas of necrosis might be overlooked, careful postmortem examinations have shown focal myocytolysis in 8% to 12% of patients dying from acute cerebral lesions. Kolin and Norris reported that transmurally scattered foci of damaged myocardial fibers are more common in patients with intracranial lesions than in control subjects (p < 0.01). Norepinephrine may cause myocardial damage either by systemic hypertensive effects that lead to left ventricular strain or by direct tissue toxicity.

**Coronary Vasospasm**

In view of the foregoing, the cardiac disorder associated with an SAH may be due not only to neurogenic factors but also to the myocardial damage itself. The cause for coronary vasospasm in patients with primary cardiovascular disease is unclear even now, and is still less clear in cases of SAH. Although the mechanisms of SAH were not given, some reports have suggested that coronary vasospasm may be responsible for the cardiac ischemic changes associated with SAH. Toyama, et al., reported the occurrence of Prinzmetal's variant angina in a patient with SAH.

Although coronary arteriography did not show vasospasm, the ischemic EKG changes and reduced contractility in our patient are thought to be due to a coronary spasm for the following reasons (even though ergonovine challenge was not performed): 1) no narrowing of the coronary artery was observed; 2) repeated sudden ST-T changes occurred which did not follow tachycardia or hypertension; and 3) there was good response to the use of nitroglycerin.

Such factors as severity, duration, and frequency of coronary vasoconstriction may play an important role in coronary artery disturbances. Various factors involved in coronary vasospasm following SAH can cause a wide spectrum of clinical syndromes ranging from transient ischemia, through "stunning" of the myocardium without necrosis or scattered myocytolysis to an irreversible infarction. Our patient's situation was intermediate between these extremes; she manifested reversible postischemic left ventricular dysfunction and a return of the EKG changes to normal.

**Stunned Myocardium**

Reduced contractile function and diastolic compliance following coronary occlusion may persist for several days after re-establishment of coronary perfusion. Even though this phenomenon was not a new find, it was termed "stunned myocardium" by Braunwald and Kloner. The mechanism involved in the development of this condition is still unknown. Braunwald and Kloner attributed stunned myocardium to a delayed recovery of adenosine triphosphate (ATP) concentration because the process of a recovery of postischemic intracellular ATP depletion following the re-establishment of perfusion was found to occur in parallel with the recovery of myocardial contractility. On the other hand, reduced myocardial contractility following the re-establishment of perfusion is alleviated by the administration of free radical scavengers, and electron paramagnetic resonance spectroscopy has shown that a burst of free radical production can occur in such reversible ischemic conditions. These findings suggest that reversible cellular disturbances due to free radicals may be the cause of stunned myocardium.

Because transient electrical instability may accompany coronary vasospasm, profound ischemia without cell death may cause a part of the myocardium to become electrically inert; consequently, electrically stunned myocardium may be associated with a transient Q wave. Therefore, coronary spasm is believed to have caused the EKG abnormalities and stunned myocardium in our patient. The manifestation of these various changes in association with SAH may be dependent upon the degree of a coronary vasospasm.

**Suggested Management**

To avoid unnecessary delays in brain- and life-saving surgical intervention, it is very important for neurosurgeons and anesthesiologists to evaluate expeditiously whether these EKG abnormalities are electrical or ischemic. Echocardiography or radionuclide ventriculography is currently considered to be most useful and essential for such cardiac evaluations. In addition, coronary arteriography should be performed when there is any abnormal motion of the arterial wall; this study should assist in the differential diagnosis.

**References**

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Manuscript received November 7, 1989. Accepted in final form December 19, 1990. Address reprint requests to: Kiyoshi Yuki, M.D., Department of Neurosurgery, Kure National Hospital, 3-1 Aoyama-cho, Kure, Hiroshima 737, Japan.