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Immediate Coronary Vasospasm upon the Onset of Subarachnoid Hemorrhage during Cardiac Catheterization

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Summary. **The possible mechanisms leading to acute cardiopulmonary arrest (CPA) soon after a subarachnoid hemorrhage (SAH) are still controversial. Cardiac arrest, which has been thought to be mainly related with arrhythmia triggered by the onset of SAH, is less common than respiratory arrest without circulatory collapse but is not a negligible cause of sudden death after SAH. A 69-year-old man with angina pectoris suffered cardiac arrest immediately after the loss of consciousness during coronary angiography. His angiogram revealed a vasospasm of the left coronary arteries which had induced the cardiac arrest. The cause of loss of consciousness was diagnosed as a SAH by brain computed tomography (CT). It is strongly suggested that the onset of the SAH triggered the coronary vasospasm which in turn induced cardiac arrest. Our case clarified that sudden death soon after SAH may be partly caused by cardiac arrest induced by coronary vasospasm. This case report may resolve some aspects of the controversy over the cause of primary cardiac arrest following SAH.**

Key words—coronary vasospasm, sudden death, cardiac arrest, myocardial ischemia, subarachnoid hemorrhage.

INTRODUCTION

Sudden death associated with the onset of subarachnoid hemorrhage (SAH) is not uncommon. Approximately 10% of sudden deaths in cases of cardiopulmonary arrest (CPA) on arrival in Japan are directly attributable to SAH. Respiratory arrest without circulatory collapse is more common than primary cardiac arrest as an initial cause of sudden death. From clinical evaluation of CPA cases, we previously determined that the direct cause of death following SAH was primary respiratory arrest in 56% of cases, cardiac arrest in 22%, and unknown in 22%[1]. However, diagnosis in our study was based on indirect evidence such as electrocardiogram (ECG) at the onset scene and ECG on admission. Although many authors have discussed the mechanism of sudden death after SAH, it remains controversial and possibly varied. The role of primary cardiac arrest has also yet to be clarified. Our unique case with SAH during coronary angiography presented direct evidence of one mechanism of sudden cardiac arrest.

CASE REPORT

A 69-year-old man with a history of angina pectoris after myocardial infarction underwent coronary angiography at Kawasaki Chuo Hospital on July 20, 1994. He had also suffered from hypertension, hypercholesterolemia, and atrial fibrillation, without any history of neurological deficit or syncope. His initial coronary angiogram showed a 50%-diameter stenosis in the proximal left anterior descending artery (LAD) and subtotal occlusion of the distal left circumflex artery (LCX) (Fig. 1). On the basis of the findings of the initial angiogram, percutaneous transluminal coronary angioplasty was scheduled for July 29.

The patient experienced no neurological deficit or headache in the catheter laboratory. After the oral administration of 5 mg tablet of nitrazepam and an intravenous infusion of heparin sodium at a rate of 400 unit per hour, his blood pressure was 173/83

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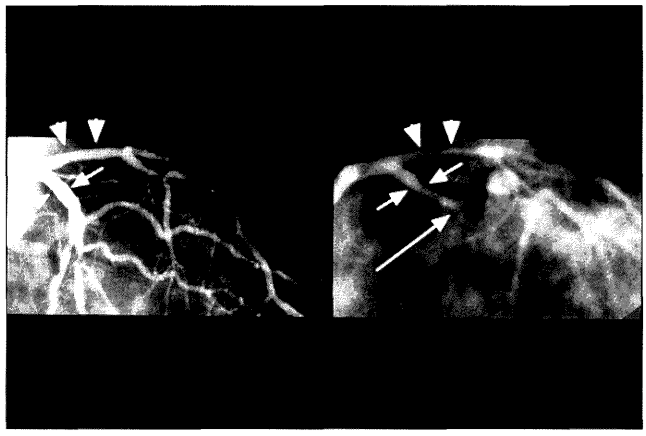

Fig. 1. Left coronary angiogram of the patient on July 20 shows multiple stenotic lesions, and this finding was duplicated in the angiogram before the onset of SAH on July 29. (*left*) The angiogram after the recovery from cardiopulmonary arrest following the onset of SAH reveals spastic stenosis in the proximal LAD (*arrows*) and LCX (*arrow heads*), and subtotal occlusion in the distal LAD (*long arrow*) (*right*). The real time fluoroscopy demonstrating total occlusion in the left main artery which was performed simultaneous with circulatory collapse could not be filmed.

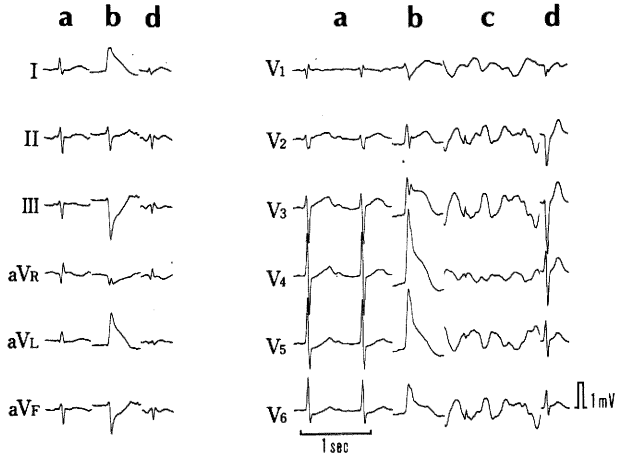

Fig. 2. Electrocardiogram of the patient on July 29 before the onset of SAH (**a**), upon loss of consciousness due to the onset of SAH during the catheterization (**b**), immediately after the onset of SAH (**c**), and after the cardiopulmonary resuscitation (**d**).

mmHg and pulse rate was 74 beats/ min with atrial fibrillation (Fig. 2a). The findings of the angiogram taken prior to the balloon angioplasty were the same as those of the initial angiogram and showed no vasospasm. When a guide wire for angioplasty was inserted in the LCX in the next procedure, he immediately began snoring and lost consciousness without any complaint of headache or convulsion. Simultaneously, ECG monitor revealed an elevation of ST-segment in leads I, a VL and V2-V6 (Fig. 2b), and blood pressure dropped remarkably. The real time coronary fluoroscopy with contrast injection which was performed simultaneously with the circulatory collapse of the patient showed a total occlusion of the left main coronary artery. He went into an electromechanical dissociation, intubated, resuscitated, and was given intravenous administration of 9.6 million international unit of tissue plasminogen activator, inotropic agents and vasodilator material. In spite of pacemaking, ventricular fibrillation was observed 8 min after the loss of consciousness (Fig. 2c). As a result of resuscitation and cardioversion, cardiac output and spontaneous respiration were recovered, and the elevated ST- segment diminished on the ECG monitor (Fig. 2d), but unconsciousness without focal neurological deficit remained. The contrast injection and angiogram of the left coronary system performed after recovery of systemic circulation revealed that spastic stenosis remained in the proximal LAD and LCX (Fig. 1). Brain computed tomography (CT) on the next day demonstrated a broad diffusion of blood in the subarachnoid space (Fig. 3). Analyses of cerebrospinal fluid, cerebral angiography and brain CT on the onset day were not carried out because of his poor condition. The patient died following brain death on August 1. Autopsy was not permitted.

The cause of the rapid deterioration of the patient's consciousness was identified as SAH by a brain CT the next day. This conclusion was reached because the loss of consciousness preceded the circulatory collapse, his symptoms did not appear arrhythmogenic, and it was unlikely that the SAH had developed between end of the catheterization and the brain CT. Moreover, the onset of SAH was strongly implicated as a trigger of the marked vasospasm of the coronary artery, since the insertion of a guide wire into the coronary artery seldom induces vasospasm and the patient had no history of spastic angina. Thus, on the basis of these facts, we speculate that SAH triggered coronary vasospasms which in turn induced cardiac arrest. Additionally, although it is difficult to reach a conclusion on the etiology of the SAH because of the absence of cerebral angiography and autopsy, the CT findings failure to depict any abnormal structures in the cerebral parenchyma suggests that a ruptured aneurysm was the most probable cause for the SAH.

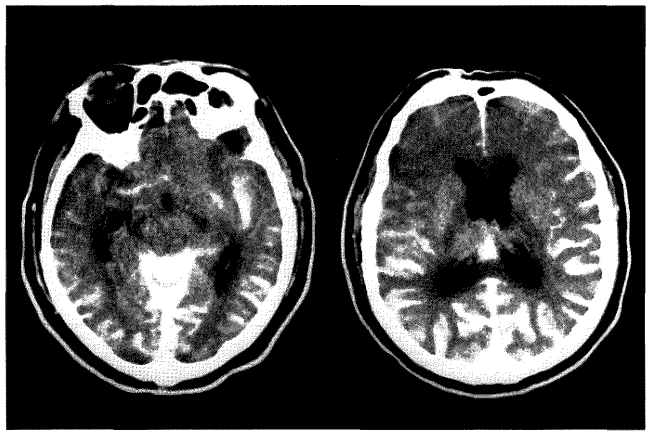

Fig. 3. Non-contrast brain CT of the patient on July 30 reveals a broad diffusion of blood in the subarachnoid space.

DISCUSSION

SAH is one of the most common causes of acute CPA, and it has been reported that 10% to 15% of patients with SAH died before hospitalization[2]. In spite of the importance of SAH in acute CPA, the etiology and mechanism of acute CPA after SAH have been described in only a few reports.

Conservative opinions have focused on respiratory arrest as a primary cause of acute CPA after SAH. Shapiro[3] documented that all of his 26 cases progressed to acute CPA after SAH by respiratory arrest. Thompson et al.[4] documented that dynamic axial brain stem distortion from acute intracranial pressure differences between the supratentorial compartment and infratentorial compartment can lead to respiratory arrest. Based on many findings including our own[1,5,6], a large majority of the cases undoubtedly fall into CPA as a consequence of primary respiratory arrest. However, the clinical courses of some cases ending in 'sudden death' after SAH are too fulminant to be explained by respiratory mechanisms.

Recent opinions have focused on cardiac arrest as a cause of acute CPA after SAH, and several authors[7,8,9] reported the association of SAH with arrhythmia and myocardial ischemia. Nakamura et al. described in their report on ST-segment elevation in SAH that these electrocardiographic changes could be explained by alterations of the autonomic activity in the coronary arteries or the myocardium. Yuki et al. discussed coronary vasospasms and reversible myocardial ischemia following SAH, but in their case there was no acute CPA, the onset of myocardial ischemia was 26 days after the SAH, and coronary angiography performed 4 days after the ischemia did not show any spasm of the coronary artery.

Our unique, valuable case partly resolved one unanswered question pertaining to this focus on myocardial ischemia as a primary cause of CPA after SAH. Coronary vasospasm with direct evidence was clearly triggered by the onset of SAH and induced cardiac arrest.

The association between the onset of SAH and coronary vasospasm remains unclear. The most probable mechanism proposed is that a vasospasm is induced by a sympathetic overactivity: a sudden release of catecholamines triggered by SAH. This mechanism is also thought to induce ventricular tachyarrhythmia and direct myocardial damages. Yasu et al[10] refuted coronary vasospasm as a cause of ECG abnormality and ventricular asynergy in their report of a case with SAH presenting normal coronary findings during ongoing ST elevation on the ECG. Although their case is not referable to ours for the two reasons that the coronary angiography of the case was not performed coincident with the onset of SAH and their case had no circulatory disaster, the authors also referred to increased sympathetic activity triggered by ischemia of the hyporthalamus and of midbrain reticular formation as a cause of ECG abnormality.

This report presents a strong argument that some cases with acute CPA after SAH are caused by coronary vasospasm, and that these patients should be resuscitated with the presupposition that myocardial ischemia is present.

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