Case Report

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Subarachnoid Hemorrhage Misdiagnosed as an Acute ST Elevation Myocardial Infarction

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Without significant coronary artery stenosis, ischemic electrocardiographic change including ST segment elevation, segmental wall motion abnormality and elevated serum cardiac-specific markers (creatine kinase-MB, Troponin-T) may develop after central nervous system injuries such as subarachnoid, intracranial or subdural hemorrhage. Misdiagnosing these patients as acute myocardial infarction may result in catastrophic outcomes. By reporting a case of a 55-year old female with subarachnoid hemorrhage mimicking acute ST elevation myocardial infarction, we hope to underline that careful attention of neurologic abnormality is critical in making better prognosis. **(Korean Circ J 2012;42:216-219)**

KEY WORDS: Myocardial infarction; Subarachnoid hemorrhage.

Introduction

Although, electrocardiographic change is not uncommon in patients with cerebrovascular disease including subarachnoid hemorrhage, subdural hematoma, and ischemic stroke,[1-3] typical ST-segment elevation is rare. The precise mechanism is not clear, but it is widely accepted that excess releasing of cathecholamine may influence myocardial contractility and necrosis.[2][4][5] Here, we intend to emphasize that careful attention of neurologic abnormality can enable better prognosis.

Case

A 55-year-old female patient presented with severe headache and loss of consciousness. She was previously healthy and had no symptoms. On arrival, she was drowsy and her blood pressure, pulse rate, respiratory rate and body temperature were 73/50 mm Hg, 80 beats per minute, 20 per minute and 36.0°C, respectively. There was no head trauma, cardiac murmur or abnormal respiration sound. Electrocardiogram on arrival presented ST segment elevation in lead II, III, aVF, V 5, 6 and depression in V 1, 2 (Fig. 1). Laboratory examination showed her creatine kinase-MB was 1.21 ng/mL and troponin-T <0.010 ng/mL. Assuming acute myocardial infarction, we conducted emergency coronary angiography but there was no significant stenosis (Fig. 2). Echocardiogram exhibited hypokinesia on septum and apex and left ventricular ejection fraction was 43%. During coronary angiography, the patient vomited once and became stuporous. We then checked brain computed tomography immediately, and found a large amount of subarachnoid hemorrhage and small intracranial hemorrhage in the medial temporal lobe base (Fig. 3). Upon cerebral angiographic computed tomography, a small saccular aneurysm in the right posterior communicating artery area was detected (Fig. 3). After taking right pterional craniotomy and aneurysmal neck clipping, the patient improved and was discharged in 30 days without any neurological sequela.

Discussion

It has been reported that abnormalities of electrocardiogrphy, echocardiography and serum cardiac specific markers are associated with cerebrovascular disease.[4-8] The most common cause is subarachnoid hemorrhage,[9-11] but additional causes include head injury, meningi-

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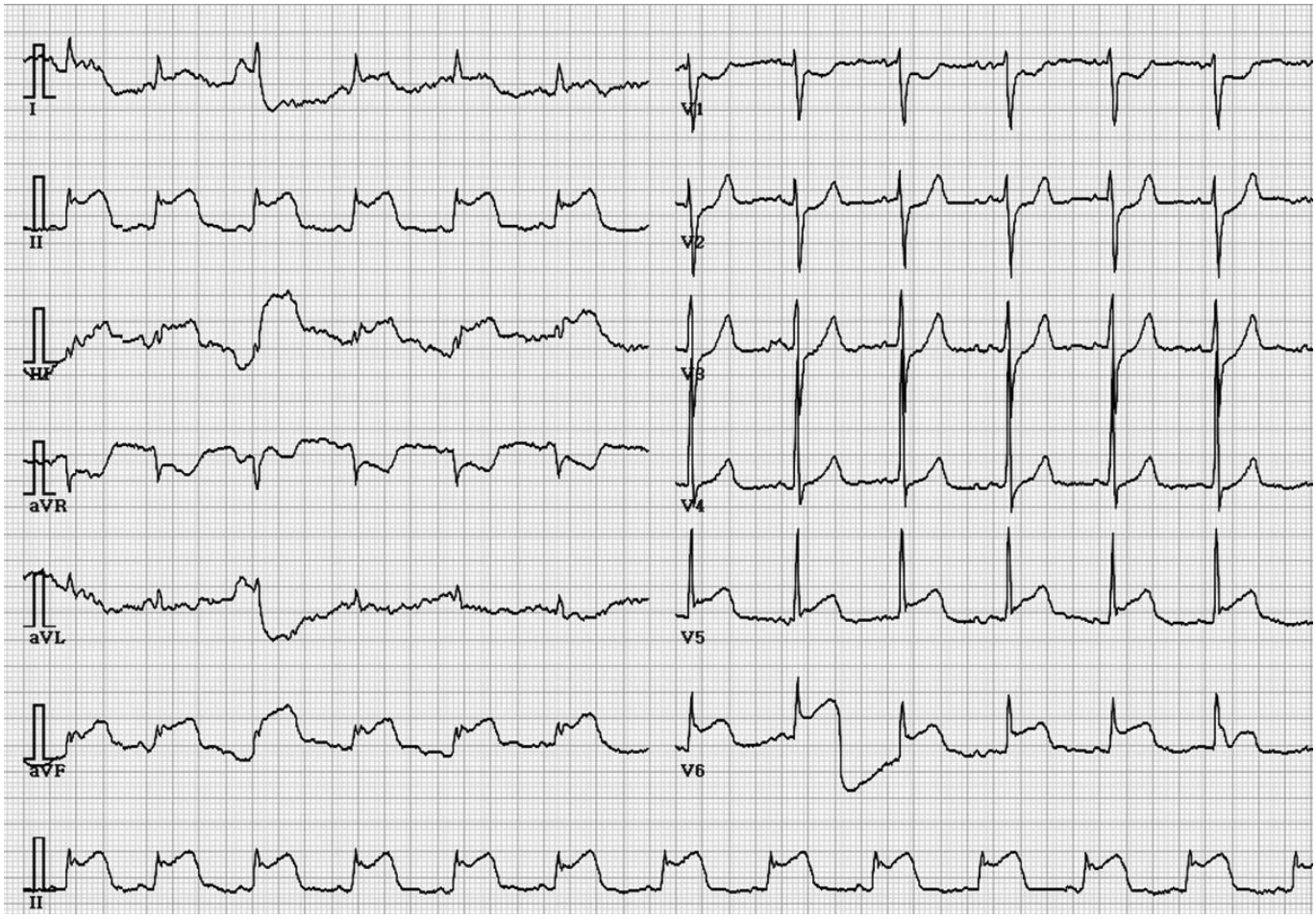

Fig. 1. Twelve-lead surface electrocardiogram showing extensive ST elevation (II, III, aVF) and ST depression ($V_{1,2}$).

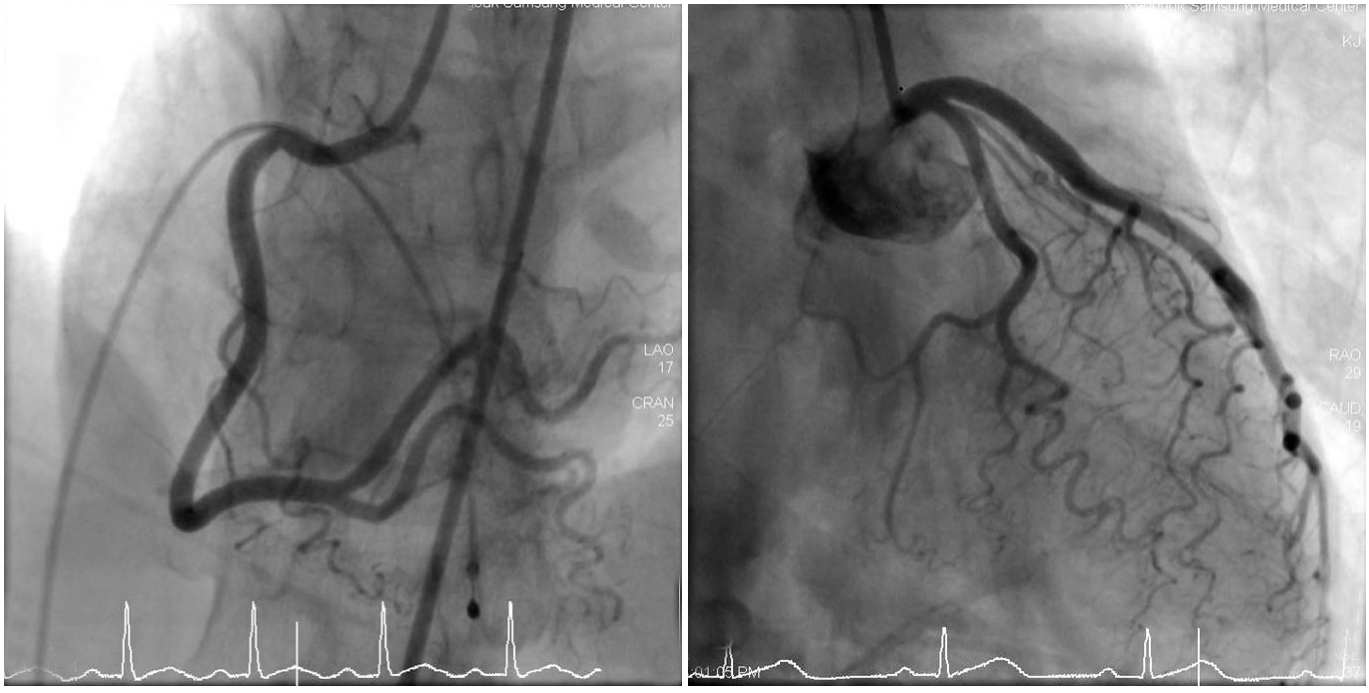

Fig. 2. There is no significant stenosis in coronary angiography.

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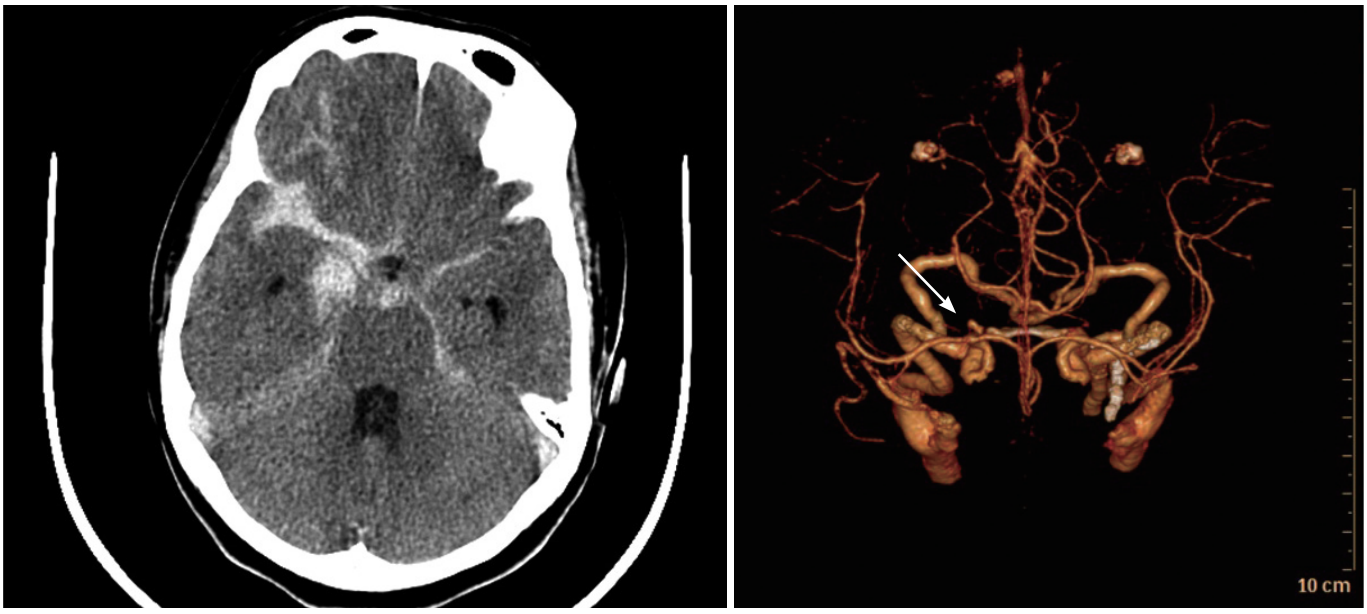

Fig. 3. A large amount of the SAH with a small ICH in the medial temporal lobe base. Small saccular aneurysm in the right posterior communicating artery area (arrow). SAH: subarachnoid hemorrhage, ICH: intracranial hemorrhage.

tis and brain tumor. Previous investigators has suggested that severe stress such as ischemic stroke and cerebral hemorrhage may provoke high catecholamine induced neurogenic-stunned myocardium.[2)4-5)] Increased release of catecholamines from local nerve endings in the heart may mediate these cardiac abnormalities. Transient severe coronary vasoconstriction leads to ischemia followed by postischemic ventricular failure and subendocardial myocardial damage. In addition, a direct cardiotoxic effect of catecholoamine may cause the development of subendocardial damage.[4)5)] Autopsies have revealed areas of characteristics subendocardial myocardial lesions, called contraction band necrosis, in the hearts of patients with subarachnoid hemorrhage (SAH).[14-16)] The myocardial damage resembles lesions produced in animal experiments by infusion of norepinephrine.[4)] The characteristic pattern of myocardial lesions has suggested that damaging catecholamines are released from intramyocardial nerve endings rather than from the general circulation.[17)] Despite all these studies, the precise mechanism of action is still unclear and future studies are required.

When unconscious cerebral hemorrhage patients with electrocardiograms showing ST segment elevation come to the emergency room, we have a tendency to misdiagnose as acute myocardial infarction and treat with multiple antiplatelet and anticoagulate agents, which can cause harmful effects. Furthermore, delaying accurate diagnosis may result in catastrophic outcome.[12)13)] Therefore, any neurological abnormalities including paralysis or severe headache should be dealt with careful attention and the possibility of cerebrovascular disease should be considered, prompting the need to be prudent with the use of antiplatelet agents. Even if a patient is misdiagnosed as acute ST elevation myocardial infarction, we should consider a cerebral cause if there is no significant stenosis in coronary angiography, and act promptly to save the life of a patient. It is well known that stress induced cardiomyopathy may develop by a cerebrovascular accident, but typical ST elevation is rare. By reporting this case, we wish to share our uncommon experience and hope that it may be helpful in future cases.

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