Concurrent Subarachnoid Hemorrhage and Acute Myocardial Infarction: A Case Report

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Concurrent subarachnoid hemorrhage and acute myocardial infarction are rare, and the therapeutic strategies are seldom discussed. Herein, we report a 58-year old man with subarachnoid hemorrhage and acute myocardial infarction who was successfully treated with primary percutaneous coronary intervention for the myocardial infarction with supportive treatment for the subarachnoid hemorrhage.

Key words: acute myocardial infarction, primary percutaneous coronary intervention, subarachnoid hemorrhage

Introduction

Subarachnoid hemorrhage (SAH) is frequently associated with myocardial injury and dysfunction¹⁻³. The myocardial damage may be minor (evidenced by elevated cardiac enzymes) or major (causing hemodynamic instability and left ventricular dysfunction)³. But concurrent SAH and acute myocardial infarction (AMI) are unusual and difficult to manage⁴⁻⁵. We report a 58 year-old man who presented with head injury and chest pain.

Case Report

A 58-year-old diabetic man had a history of an extensive anterior AMI and had received coronary angioplasty and stenting in the left anterior descending (LAD) coronary artery in 2003. He received no regular medications afterward. In 2006, he fell from a ladder from the second floor, resulting in a head injury with muscle weakness and transient loss of consciousness. He was sent to our emergency room, where brain computerized tomography (CT) revealed a SAH (Fig. 1). After admission, the patient complained of chest pain and electrocardiography (EKG) showed ST-segment elevation in the anterior leads (Fig. 2). Initial cardiac enzyme levels were creatine kinase (CK) 101 IU/l, CK-MB (creatine kinase-MB) 0.2 ng/dl and troponin I 0.06 ng/ml, which then increased to CK 1076 IU/l, CK-MB 53.2 ng/dl and troponin I 30.2 ng/ml several hours later. The impression was extensive anterior wall AMI and traumatic SAH. Fibrinolytic therapy was contraindicated. Coronary angiography was performed, demonstrating total occlusion of the LAD artery (Fig. 3). Percutaneous coronary intervention (PCI) was carried out without heparinization and no use of devices for protection against distal embolization or thrombosuction. A bare metal stent was deployed in the LAD (Fig. 4).
Mannitol was given via intravenous infusion for the SAH. Acetylsalicylic acid and clopidogrel were administrated on the 6th day of hospitalization. The maximum CK, CKMB and troponin I were, respectively, 3880 IU/l, 199.6 ng/dl and over 100 ng/ml. He was discharged in stable condition on the 8th day of hospitalization without any sequelae from the SAH. Three months later, follow-up coronary angiography revealed a patent LAD.

Discussion

Myocardial damage occurring in association with SAH is a well-described phenomenon\(^{(1-3)}\). The
myocardial damage is believed to result from an associated catecholamine surge. In most cases, the myocardial injury is transient. However in our patient, the myocardial infarction was extensive and dominant but the SAH was mild. Both should be managed at the same time. First, the risks of peri- and post-operative cardiac complications from SAH surgery need to be considered. Second, bleeding form the SAH may be exacerbated after treatment for AMI. In the event of absolute contraindication of lytic therapy, prompt coronary reperfusion can be achieved with primary PCI, probably resulting in a smaller infarct and better left ventricular function\(^{(4)}\). Treatment of concurrent SAH and AMI has
been reported by Barcena et al\textsuperscript{5}\textsuperscript{5}. But in that report, heparin, acetylsalicylic acid, and ticlopidine were given during the procedure and coil embolization for a ruptured intracranial aneurysm was done 1 hour after PCI. In our case, primary PCI was performed. Heparin, acetylsalicylic acid, ticlopidine and clopidogrel were not used in the early stage, even during the PCI procedure, to prevent exacerbation of the SAH. We began administration of antiplatelet agents after stabilization of the cardiac condition. The LAD remained patent in the follow-up coronary angiogram. No acute or subacute stent thrombosis developed before heparin and antiplatelet drug therapy was initiated.

In summary, patients with concurrent SAH and AMI are more difficult to manage than those with either condition alone. The most common causes of SAH are head trauma and rupture of an intracranial aneurysm. Many patients with SAH resulting from rupture of an aneurysm have diffuse distribution of blood in the subarachnoid spaces and basal cisterns on brain CT. In our patient, brain CT showed that the SAH localized to the peripheral subarachnoid cisterns. This finding favors traumatic SAH, rather than a ruptured aneurysm\textsuperscript{6}\textsuperscript{6}. In this case, primary PCI for AMI may have reduced the infarct area and prevented LV dysfunction; heparin and antiplatelet drugs were not used to avoid exacerbated bleeding from SAH. These patient need to be closely monitored for acute or subacute in-stent thrombosis. There was no heavy thrombous burden in the LAD coronary artery, so the above complications didn’t occur in our case.

References

蜘蛛腦膜下出血合併急性心肌梗塞：病例報告

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蜘蛛腦膜下出血合併急性心肌梗塞是一個災難性事件，臨床上也很少探討到處理上述問題的方針。這裡我們提出一個合併上述兩個情況，原先未使用抗凝血劑和抗血小板藥物下，以冠狀動脈介入性治療方式治療急性心肌梗塞，及以支持性療法治療蜘蛛腦膜下出血成功之個案。

關鍵詞：急性心肌梗塞，冠狀動脈介入性治療，蜘蛛腦膜下出血

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