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Case Report

Management of Patients with Stunned Myocardium Associated with Subarachnoid Hemorrhage

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Summary: Cardiac complications are well known after aneurysmal subarachnoid hemorrhage. Electrocardiographic changes occur in 50% to 100% of such cases. Arrhythmias, left ventricular dysfunction, and frank myocardial infarction are infrequently observed. Myocardial infarction must be differentiated from neurogenic stunned myocardium, which is a reversible condition. From 1996 to 2001, 105 patients with aneurysmal subarachnoid hemorrhage underwent endovascular treatment at the University of Michigan. Of these, four patients with no history of cardiac disease experienced cardiac failure related to neurogenic stunned myocardium. All had signs of left ventricular dysfunction, electrocardiographic changes, and elevated cardiac enzymes. Three had pulmonary edema at presentation. All were diagnosed with myocardial infarction. One underwent coronary angiography, which was normal. All were considered poor surgical candidates and underwent endovascular treatment of the aneurysms. Three of four patients developed symptomatic vasospasm, and two required balloon angioplasty. Three patients achieved good outcomes. The eldest died from severe vasospasm that was unresponsive to angioplasty. Reversible cardiac failure associated with subarachnoid hemorrhage may be due the neurogenic stunned myocardium. Frequent symptomatic vasospasm occurs, possibly related to poor cardiac output and the inability to optimize hyperdynamic hypervolemic therapy, particularly with compromised volume status. These patients can be treated with endovascular therapy of the aneurysms and balloon angioplasty as needed. With aggressive management, patients can recover from these reversible cardiac complications.

Electrocardiographic changes associated with aneurysmal subarachnoid hemorrhage (SAH) are well known, occurring in 50% to 100% of such cases (1). Prolongation of QT interval, depression or elevation of ST segments, and T wave inversion are the commonly encountered electrocardiographic alterations. Left ventricular dysfunction and frank myocardial infarction are infrequently observed. Reduced cardiac output due to impaired left ventricular contractility in severely affected patients might increase the risk of delayed cerebral ischemia from vasospasm. However, myocardial infarction must be differentiated from "neurogenic stunned myocardium," which is a reversible condition.

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Case Material

From 1996 to 2001, 105 patients with aneurysmal SAH underwent endovascular treatment. Of these, four patients (Table) with no history of cardiac disease experienced cardiac failure related to neurogenic stunned myocardium. The three female and one male patient ranged in age from 32 to 83 years (mean age, 51 years). One patient presented with Hunt and Hess grade II and the remainder with Hunt and Hess grades III–IV. All had signs of left ventricular dysfunction, electrocardiographic changes, and elevated cardiac enzymes. Three had pulmonary edema at presentation. All were diagnosed with myocardial infarction. One patient also underwent coronary angiography, which showed normal results. Cardiac enzymes reverted to normal in 4 to 9 days (mean, 5 days). Left ventricular dysfunction improved in 5 to 9 days (mean, 7 days). All patients were considered poor surgical candidates because of their cardiac status and underwent endovascular treatment of the aneurysms. Three of the four patients developed symptomatic vasospasm, and two required balloon angioplasty. Three patients achieved good outcomes. The eldest died as a result of severe vasospasm that was unresponsive to angioplasty. Our Institutional Review Board exempted this study from review.

Case 1

A 44-year-old man with no significant medical history presented with Hunt and Hess grade IV SAH. CT of the head revealed Fisher grade IV SAH. The patient had an irregular cardiac rhythm, and electrocardiography showed sinus tachycardia, ST abnormality, and T wave abnormality. Systolic blood pressure was very labile, ranging from 120 to 200 mm Hg. Cardiac enzymes were markedly elevated: CK, 389 IU/L; CK-MB, 6.5% of total CK; and troponin, 49.4 ng/mL. Echocardiography revealed left ventricular ejection fraction of <20% and akinesis of the apex and anterolateral wall. Coronary angiography did not show any significant coronary artery disease to account for the markedly depressed cardiac function. The same day, the patient underwent cerebral angiography, which revealed a 2-cm-diameter right supraclinoid carotid aneurysm at the origin of the posterior communicating artery. The patient continued to have depressed cardiac function and hypotension during the ensuing days and was given a regimen of inotropic agents. He was not considered to be fit for surgery and hence underwent endovascular occlusion of the aneurysm on the third day. His cardiac enzymes and left ventricular ejection fraction returned to normal levels by the fifth day, and he was doing well from a cardiac standpoint without any inotropic agents. However, he did develop right side weakness and extensor posturing. CT of the head did not show any obvious infarction, but transcranial Doppler study showed elevated velocities in left middle anteroposterior cerebral artery territories, suggestive of vasospasm. The patient's condition improved clinically with careful management of fluid status and blood pressure

Summary of the cases

	Age/ Sex	Admission Hunt & Hess Grade	Admission Fisher CT Grade	Aneurysm Location and Day of Coiling	Poor LVEF (Days)	Pulmonary Edema	Vasospasm	Balloon Angioplasty	Outcome
Case 1	44/M	IV	IV	Pcom, day 3	1–5	N.A.	Day 5	N.A.	Good
Case 2	32/F	III	III	Pcom, day 1	1–9	Day 1	Day 7	+	Good
Case 3	83/F	II	I	Acom, day 1	1–9	Day 6	Day 6	+	Died
Case 4	44/F	III–IV	III	Vertebrobasilar, day 1	1–4	Day 1	N.A.	N.A.	Good

Note.—Pcom = Posterior communicating artery, Acom = Anterior communicating artery, LVEF = Left ventricular ejection fraction.

during the next 2 days; transcranial Doppler velocities returned to normal. The patient was discharged after 2 weeks and did well after that. At the 3-month follow-up examination, cerebral angiography revealed some coil compaction with a residual patent aneurysm, which was successfully treated with further coiling. The patient was doing well at the time of follow-up.

Case 2

A 32-year-old previously healthy woman presented with severe headache, partial left cranial nerve III palsy, and Hunt and Hess grade III SAH. CT of the head showed Fisher grade III SAH. The patient's blood pressure was very labile, intermittently high (190/110 mm Hg), and occasionally low (78/50 mm Hg). Electrocardiography showed sinus tachycardia, prolonged QT interval, and nonspecific ST and T wave changes. CK (891 IU/L) and troponin (12.4 ng/mL) levels were elevated, but CK-MB (1.7% of total CK) was within normal limits. Echocardiography revealed left ventricular ejection fraction of 10% and global hypokinesis of the left ventricle, and the patient was thought to have acute myocardial infarction. Radiography of the chest showed pulmonary edema. Cerebral angiography performed on the same day showed an approximately 8-mm left posterior communicating aneurysm, which was treated with endovascular coil occlusion at the same time because the patient was not considered to be a good surgical candidate because of her poor cardiac status. Her cardiac enzymes returned to normal on the third day. On the seventh day, the patient's mental status deteriorated. Transcranial Doppler and xenon CT showed global ischemia; hypervolemia, hemodilution, and hypertensive ("Triple-H") therapy was begun. The patient underwent balloon angioplasty of bilateral distal internal carotid arteries, A1 and M1 segments, P1 and P2 segments, and distal basilar artery, and good angiographic results were achieved. The left ventricular ejection fraction returned to 55% by the ninth day. The patient was weaned off the hyperdynamic therapy from the 14th day onward and was discharged to inpatient rehabilitation 3 weeks after SAH. One-year follow-up angiography showed no residual or recurrent aneurysm, and the patient was doing well at the time of follow-up.

Case 3

An 83-year-old woman with no history of cardiac complaints presented with severe headache, photophobia, nausea, vomiting, and Hunt and Hess grade II at admission. CT of the head revealed Fisher grade I SAH. A silent acute myocardial infarction was suggested by electrocardiography in the inferior leads, although the patient had no complaints of chest pain or other cardiovascular symptoms. Her cardiac enzymes were markedly deranged (CK, 905 IU/L; troponin, 73.6 ng/mL; CK-MB, 14.6% of total CK). She had mildly decreased left ventricular systolic function with EF of 40% to 45% and also had inferior and posterior hypokinesis. Cerebral angiography revealed a complex anterior communicating artery aneurysm, which was treated by endovascular coiling on the same day. The patient had left hemiparesis 4 hr after endovascular treatment. Although repeat angiography did not show any apparent intraluminal thrombus or branch occlusion, it was thought to be related to an embolic event. On the sixth day, the patient's echocardiographic findings returned to normal but she developed right hemiparesis. Xenon CT showed global ischemia, most pronounced in the left middle cerebral artery territory, and balloon angioplasty of the left anterior and middle cerebral artery branches was performed. Radiography of the chest revealed pulmonary edema. Triple-H therapy was begun but only to the extent tolerated because of the patient's poor myocardial status. Her cardiac enzymes remained high until the ninth day. MR imaging performed at that stage showed multiple areas of cerebral infarction, and the patient died 18 days after SAH.

Case 4

A 44-year-old woman with no previous cardiac complaints presented with headache, sudden unresponsiveness, and Hunt and Hess grade III–IV SAH. CT of the head showed Fisher grade III SAH, and radiography of the chest showed extensive pulmonary edema. Electrocardiography showed sinus rhythm but prolonged QT interval. Echocardiography was positive for severe left ventricular dysfunction, with an EF of 25% and hypokinesia. The patient's cardiac enzymes on the first day were markedly abnormal (CK, 354 IU/L; troponin, 15 ng/mL; CK-MB, 5.8% of total CK). Cerebral angiography revealed a wide necked right vertebrobasilar junction aneurysm. It was treated with endovascular coiling by using a balloon remodeling technique (2) because the patient was considered to be a poor surgical candidate because of her cardiac status. Her cardiac enzymes and echocardiographic parameters returned to normal levels by the fourth day. She had an uneventful hospital course and was discharged to home after 3 weeks and was doing well at the time of follow-up.

Discussion

The cardiac changes that occur in association with SAH are thought to result from increased central sympathetic activity, which typically results in a hyperdynamic cardiovascular state (1). Small myocardial enzyme elevations reflect relative cardiac decompensation and failure of the left ventricle to meet these inotropic demands. Massive peripheral vasoconstriction may also further aggravate the left ventricular decompensation by increasing the cardiac afterload. Transient increase in sympathetic nervous activity induces myocardial damage, referred to as *neurogenic stunned myocardium*, which is hypothesized to be caused by oxygen-derived free radicals or transient calcium overload (3). Both free radical and calcium overload is associated with decreased responsiveness of contractile filaments to calcium, which is secondary to selective troponin I proteolysis (4). Myocardial stunning has been shown to be reversible within 48 hr in experimental coronary occlusion in a

canine model (5). The delayed recovery is thought to be due to resynthesis of the damaged proteins (3).

Neurogenic stunned myocardium associated with SAH is characterized by a transient nature (ie, postischemic dysfunction is fully reversible), the dysfunction's not being caused by a primary defect in myocardial perfusion (ie, normal coronary arteries), peak CK-MB levels of ≥2%, poor neurologic grade, and female sex. Neurogenic stunned myocardium most likely resembles exercise-induced myocardial stunning and is probably due to a dramatic increase in cardiac sympathetic drive resulting from SAH. This probably causes the myocardial oxygen demands to exceed oxygen supply (6). However, it is very important to differentiate neurogenic stunned myocardium from myocardial dysfunction caused by coronary artery disease in patients with SAH. Noninvasive technetium-99m pyrophosphate myocardial infarct imaging could help differentiate myocardial dysfunction secondary to SAH (ie, neurogenic stunned myocardium from that caused by coronary artery disease in patients with SAH) (7). Elevation of cardiac enzymes, particularly cardiac troponin, has been found to be a highly sensitive and specific indicator of myocardial dysfunction in cases of aneurysmal SAH (8). In the present study, cardiac troponin levels at the time of initial presentation were high (range, 12.4–73.6 ng/mL) and returned to normal as the cardiac status improved. However, in some cases, an invasive procedure such as coronary angiography may become necessary to rule out myocardial dysfunction due to coronary artery disease. Coronary angiography was performed in case 1 of the present study, which did not show any coronary artery disease. Echocardiography performed during the acute phase may show abnormal left ventricular wall motion, particularly of the apex that reverts back to normal within a few days.

The electrocardiographic abnormalities have been attributed to increased circulating and local myocardial tissue catecholamines in conjunction with low myocardial intracellular potassium. Hypothalamic stimulation in animals can induce cardiac changes similar to those observed after SAH (9), and most patients dying as a result of SAH have been shown to have both hypothalamic and myocardial lesions at autopsy (10, 11). Impaired myocardial contractility related to contraction band necrosis occurs in animals after excessive cardiac sympathetic stimulation (12), and this reversible form of cardiac pathologic abnormality is found in ≤50% of patients with fatal SAH at autopsy (10, 13, 14).

Neurogenic stunned myocardium due to nontraumatic SAH is probably associated with more incidence of symptomatic vasospasm because of reduced cardiac output and hypotension due to impaired myocardial contractility. The prevention and management of vasospasm is dependent on maintaining the fine balance of cerebral blood flow and perfusion pressure. This requires close monitoring of intracranial pressure and blood pressure. The patients are managed in intensive care units with continuous invasive hemodynamic monitoring and support. Calcium channel blockers have been considered to be useful in the management of patients before vasospasm occurs and in the management of patients with symptomatic vasospasm. Transcranial Doppler sonography is used frequently to monitor and detect vasospasm before the patient suffers ischemic neurologic deficit or infarct. Elevated Transcranial Doppler velocities often initiate the use of Triple-H therapy and subsequently guide it. Recently, perfusion CT has shown some promise in detecting early vasospasm (our unpublished data) and may become a valuable and easily accessible imaging technique, particularly for monitoring vasospasm. However, impaired myocardial function due to stunning can significantly limit the effectiveness of Triple-H therapy because such therapy depends on optimal cardiac function. Continuous monitoring of central hemodynamic variables, such as pulmonary artery occlusion pressure and cardiac index, allow aggressive but watchful fluid management while monitoring the cardiac status. Positive inotropic agents and pressors have been used with significant success in these patients. Intra-aortic balloon pump counterpulsation has also been used to optimize cardiac performance to allow continuation of Triple-H therapy and to maintain adequate cerebral perfusion in an attempt to reduce the risk of delayed vasospasm (6, 15, 16).

In the present study, three patients (age range, 32–44 years) achieved good outcomes and the eldest patient (age, 83 years) died as a result of complications related to vasospasm. Although the patients with good outcomes had poorer Hunt and Hess grades, they recovered completely. Compromised cerebral circulation and neural plasticity with advancing age add to unfavorable outcomes for elderly patients after aneurysmal SAH (17).

Conclusion

In summary, reversible cardiac failure associated with SAH may be due to neurogenic stunned myocardium. Associated high incidence and severity of delayed cerebral vasospasm is related to poor cardiac output and the inability to optimize hyperdynamic hypervolemic therapy. Most of these patients are not considered to be good surgical candidates and can be treated with endovascular therapy and balloon angioplasty as needed. With aggressive management, patients can recover from these reversible cardiac complications.

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