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Case Studies in Cardiac Dysfunction After Acute Aneurysmal Subarachnoid Hemorrhage

Jason C. Hamilton, BSN RN CNIM,

neurovascular ICU nurse and neurophysiology technologist at the University of Pittsburgh Medical Center in Pittsburgh, PA

Lauren Korn-Naveh, BSN RN, and

neurovascular ICU nurse at the University of Pittsburgh Medical Center. She also is a graduate student at the University of Pittsburgh School of Nursing in Pittsburgh, PA

Elizabeth A. Crago, MSN RN [research associate]

University of Pittsburgh, Pittsburgh, PA, ecrago@pitt.edu

Abstract

Patients with acute aneurysmal subarachnoid hemorrhage (SAH) often present with more than just neurological compromise. A wide spectrum of complicating cardiopulmonary abnormalities have been documented in patients with acute SAH, presenting additional challenges to the healthcare providers who attempt to treat and stabilize these patients. The patients described in this article presented with both acute aneurysmal SAH and cardiopulmonary compromise. Education and further research on this connection is needed to provide optimal care and outcomes for this vulnerable population. Nurses play a key role in balancing the critical and diverse needs of patients presenting with these symptoms.

Myocardial dysfunction after subarachnoid hemorrhage (SAH) can be challenging, even for the most experienced clinicians. In the neurovascular intensive care unit (NVICU), priorities historically have focused on preserving brain tissue and subsequently assessing secondary complications such as myocardial damage. Recent research suggests that cardiac dysfunction often accompanies SAH, and it may have a significant effect on outcomes. The purpose of this article is to present three cases of aneurysmal SAH with subsequent cardiac dysfunction and identify how nurses in the NVICU can monitor and intercede to promote optimal outcomes.

Case Presentations

Patient A

Patient A, M. M., was a 33-year-old white woman who was 6 weeks postpartum after a caesarean section (gravida 2, para 2) with a past medical history of migraine headaches. Paramedics responded to her home after she complained of the “worst headache of her life.” She initially was arousable, but her mental status deteriorated and she was electively intubated to protect her airway. Upon arrival in the emergency department (ED), she had evidence of neurological deficits including poor pupillary response and posturing to painful stimuli. Her initial Hunt and Hess (HH) grade was V and her Glasgow Coma Scale (GCS)

score was 5. An admission computed tomography (CT) scan of the brain revealed SAH with intraventricular hemorrhage (Fisher grade IV). M. M. was immediately taken to the NVICU for further evaluation and treatment.

M. M. required immediate placement of an external ventricular drain (EVD) for cerebrospinal fluid (CSF) drainage and intracranial pressure (ICP) monitoring, which resulted in an initial improvement in her clinical neurological exam. A cerebral angiogram revealed a 5-mm anterior communicating artery aneurysm, which was obliterated by inserting endovascular coils.

In addition to her neurological symptoms, M. M.'s clinical course was complicated by significant cardiac dysfunction described in the paragraphs to follow. Table 1 shows cardiac enzyme levels for the first week of her hospitalization.

Because of her elevated troponin levels and hypertension (systolic blood pressure [BP] >200 mm Hg), M. M. initially was placed on a nicardipine drip. Tests were performed to evaluate the possibility of heart damage. Her initial electrocardiogram (ECG) showed sinus bradycardia but no S-T segment changes. However, an echocardiogram (ECHO) revealed a severely hypokinetic, nondilated left ventricle with a significant decrease in left ventricular function and an estimated ejection fraction (EF) of 20%–25% (normal EF = 65%).

After initial stabilization and coil embolization, M. M. was extubated and the nicardipine drip was discontinued. Over the course of her 3rd and 4th days, M. M.'s condition became increasingly unstable. She had neurological changes including increasing headache and ICP and agitation. She also was febrile (103.4 °F/39.7 °C). The neurosurgical team ordered a one-time dose of mannitol for cerebral edema, fentanyl for headache, and a norepinephrine drip to maintain the systolic BP >180 mm Hg. An intravenous cooling catheter was inserted using a subclavian catheter line and a stat CT scan of the head was ordered to look for any new intracranial processes. Her transcranial Dopplers, a reflection of cerebral blood flow, were extremely elevated (middle cerebral artery [MCA] systolics >200 ml/sec)—findings often associated with cerebral vasospasm. Given her neurological decline, an angiogram was performed, which was negative for vasospasm.

In addition to her neurological changes, she had cardiopulmonary changes that included nonspecific T-wave changes on her 12-lead ECG, an elevated heart rate (160 beats per minute), and a rapid drop in her BP. She was tachypneic and acidotic and required reintubation. In an attempt to stabilize her hemodynamically, M. M. was placed on numerous inotropic and vasoconstrictive agents in addition to the norepinephrine drip, which included epinephrine, phenylephrine, vasopressin, and amiodarone. She also was given intravenous sodium bicarbonate and calcium chloride. To monitor cardiac output (CO), a pulmonary artery catheter was inserted. Initial CO was 9.2 L/min (normal CO = 4–8 L/min), and pulmonary capillary wedge pressure (PCWP) was 27 mm Hg (normal PCWP is 8–12 mm Hg). A transesophageal ECHO revealed decreased left ventricular function with an estimated EF of 20%–25%. She was noted to have positive blood cultures and required antibiotic therapy.

M. M.'s overall condition improved after day 6. Her neurological status slowly improved, and she became oriented, followed commands bilaterally, and was able to be extubated. She did not experience vasospasm. Her heart function improved; she was weaned off vasopressors and her pulmonary arterial catheter was removed. A repeat ECHO performed on day 6 showed slightly worse dilation of the left ventricle, decreased systolic function, and a small pericardial effusion; however, her EF increased to 40%–45%.

M. M.'s length of stay in the hospital was 20 days. She returned to her home, where she received physical and occupational therapy. Her function continued to improve and she remained at home spending much of her time with her children. She reported persistent memory loss and fatigue.

Patient B

J. N., a 56-year-old white woman, was found by her husband at the bottom of two steps in their home. She had complained of intermittent, moderate-to-severe headaches for 1 month before this incident, but a magnetic resonance imaging scan was inconclusive. She had complained of a severe headache just before being found and was reported to be apneic and pulseless by her husband. Paramedics were called and cardiopulmonary resuscitation was performed with success and she began breathing on her own. Upon arrival in the ED, J. N.'s GCS was 15, but her level of consciousness deteriorated during her ED stay and she was observed to have an episode of "seizure-like" activity. J. N.'s heart rate became labile, ranging from 40 to 140 beats per minute. She required endotracheal intubation before her heart rate normalized into the 70s. Her admission ECG was negative, and she was taken for a CT scan of the head. The scan showed a diffuse SAH with intraventricular hemorrhage (Fisher grade III). Based on her clinical presentation including the seizure-like activity, she was scored an HH grade III. J. N. was transferred to the NVICU for further neurological evaluation and treatment.

An EVD was placed upon arrival to the NVICU based on hydrocephalus seen on her admission CT scan. Her next 12-lead ECG showed significant S-T segment elevation, which, in addition to her abnormal cardiac enzymes from routine initial lab work, was indicative of myocardial injury. The next morning, J. N. was taken for an angiogram, and tests suggested a left posterior communicating artery aneurysm. Because she remained hemodynamically unstable, it was not possible to verify the aneurysm site or perform a repair. She became acidotic and hypotensive (systolic BP in the 80s) and required aggressive management with multiple intravenous inotropes and vasopressors including dopamine, dobutamine, and norepinephrine (phenylephrine). J. N. was transferred from interventional radiology to the cardiac catheterization lab (or interventional cardiology lab), where a pulmonary artery catheter was inserted to monitor her cardiac function including cardiac output. Due to the severity of her cardiac compromise, a cardiac catheterization was performed, which revealed an EF of 25% with normal coronary arteries. She returned to the NVICU on dopamine and norepinephrine drips.

Ongoing hemodynamic monitoring with the pulmonary artery (PA) catheter showed a significantly low CO (2.5 L/min; normal CO = 4–8 L/min). Her CO gradually improved over the next few days, reaching 5.8 L/min on day 2 and 7.2 L/min on day 3. She continued

to require intravenous norepinephrine to maintain a systolic blood pressure between 90 and 120 mm Hg through day 3. On day 3, her PA catheter was removed and she was weaned from vasopressor support. She was evaluated by both neurosurgery and critical care medicine and cleared as sufficiently stable to undergo surgery. On day 4, she was taken to surgery for clipping of a right posterior communicating artery aneurysm without complications and she returned to the NVICU.

J. N.'s treatment was complicated significantly as a consequence of myocardial injury following her SAH. Her unstable cardiac status prevented immediate treatment and her cerebral aneurysm could not be repaired until day 4 of her hospital stay. Table 2 shows troponin and cardiac enzyme levels for 1 week postadmission.

Although J. N.'s cardiopulmonary status improved during the first week after experiencing her SAH, she had periods of hypoxemia with arterial oxygen saturation between 88% and 90% and was placed on biphasic positive airway pressure ventilation. On the 8th day after her SAH, she became confused, tachypneic, and hypoxemic. She was reintubated and evaluated for a pulmonary embolus, which was negative. She became febrile and had an elevation in her transcranial Doppler velocity measurement above 200 ml/sec systolic. An angiogram showed cerebral vasospasm. The vasospasm was treated with induced hypertension and hypervolemia, using norepinephrine and dobutamine drips to maintain a systolic BP of 160–180 mm Hg as well as packed red blood cells and isotonic fluids to keep her central venous pressure (CVP) >8 mm Hg. She continued to be febrile and received oral antipyretics (acetaminophen) and intravenous cooling through a subclavian catheter line.

Nearly 2 weeks after her initial bleed, J. N. was hemodynamically stable and was extubated. Her ECHO and neurological status improved to a point at which she was alert and oriented, and generally weak without focal deficits or problems with swallowing, speaking, or toileting. After 19 days, she was discharged to a rehabilitation facility at which she continued to recover and gain independence. Her follow-up visits included monitoring by cardiology and neurosurgery. At neurosurgery follow-up 3 months after her initial bleed, J. N. rated her disability as slight with some residual weakness and memory problems.

Patient C

J. T., a 64-year-old white woman, initially presented to the ED after a 2-week complaint of moderate-to-severe headaches that culminated in a syncopal episode. J. T. had no prior history of chronic disease or drug abuse. She was neurologically intact with a GCS score of 15. A CT scan revealed Fisher grade III SAH, and her neurological symptoms of severe headache, nausea, vomiting, and neck stiffness were graded as an HH II.

J. T. immediately was taken to the NVICU, where she required insertion of an EVD for ICP monitoring and CSF drainage secondary to hydrocephalus. She required endotracheal intubation for acute respiratory failure, which was attributed to acute pulmonary edema. Attempts to control her extreme agitation with a propofol drip were not successful due to hypotension, and a decision was made to therapeutically paralyze her with a vecuronium drip. Cardiac enzymes and troponin levels were significantly increased. J. T.'s ECG showed sinus tachycardia (101 beats per minute) with nonspecific T-wave changes in inferior leads,

denoting a possible lateral wall injury. Table 3 shows J. T.'s cardiac enzyme trends over 5 days in the NVICU.

An ECHO performed on the day of admission was suspicious for a significant myocardial event. Results showed left ventricle function had moderately to severely decreased, with an estimated EF of 26%–30%. Mild pulmonary hypertension was discovered, with an estimated pulmonary artery systolic pressure of 34 mm Hg (normal 15–30 mm Hg). J. T.'s cardiopulmonary compromise was treated as cardiogenic shock. Her BP remained low with systolic readings in the 90s, and the critical care medicine team decided to place her on both dobutamine and norepinephrine drips to maintain her blood pressure and optimize tissue perfusion. After initial stabilization, she was taken to the angiography suite. An anterior communicating aneurysm was found and treated by endovascular coiling.

A repeat ECHO performed on day 3 demonstrated improvement; overall left ventricular function was normal, with an estimated EF of 61%–65%. She remained on norepinephrine for several days and required continuous dobutamine infusion through the fourth day of her stay, when she was weaned off the medication and remained hemodynamically stable with systolic BP greater than 130 mm Hg.

On the evening of the fifth day following her SAH, J. T. stopped following commands and had an elevation in her transcranial Doppler reading on the right side with a Lindegaard ratio greater than 4 (a mean MCA of 122 ml/sec and mean internal carotid artery [ICA] of 25 ml/sec). A subsequent angiogram showed vasospasm, and she was placed back on norepinephrine for hypervolemic-hypertensive-hemodilution (HHH) therapy.

Despite improvements in her overall hemodynamic status (blood pressure, heart rate, and ejection fraction), J. T. continued to show cardiac dysfunction as late as 18 days after her SAH. Her ECG readings showed S-T and marked T-wave abnormality, indicating anterolateral wall injury. Her ECHO results showed mild pulmonary hypertension of 48 mm Hg (normal 15–30 mm Hg). She continued to have neurological deficits through the remainder of her hospitalization with confusion and agitation, and required tracheostomy for persistent ventilator dependency. J. T. was weaned from ventilator support, but still had a tracheostomy tube for secretion management when she was transferred to an in-patient rehabilitation facility 19 days after admission. Six months after sustaining SAH, J. T. rated her recovery as good without disability.

Discussion

These cases serve as exemplars of the types of cardiac dysfunction that frequently accompany aneurysmal SAH (at our institution, we identified a 33% incidence of cardiac dysfunction following SAH, with arrhythmias and pulmonary edema as the most common complications [Crago et al., 2004]). No universally accepted explanation of the link between aneurysmal SAH and cardiac dysfunction currently exists. Ongoing investigations are examining causal factors and outcomes for cardiac dysfunction in this patient population. Zaroff and colleagues (2006) suggest that genetic predisposition may play a role in identifying patients at risk for related cardiac dysfunction. The authors found that certain

genetic combinations (polymorphisms of the adrenoceptors β -1, β -2, and α -adrenoreceptors, which may affect cardiac responsiveness to catecholamines) resulted in a 10–15-fold increase in the likelihood of developing major cardiac dysfunction following SAH (Zaroff et al., 2006). Similarly, it would seem logical that those with preexisting cardiac disease would have increased vulnerability to this kind of catecholamine surge, although a link between preexisting cardiac disease and the presence of cardiac dysfunction following SAH has not been established in our work (Crago et al.).

A commonly accepted theory suggests that a catecholamine surge occurs at the time of the aneurysm rupture, setting off a cascade of events that includes cardiopulmonary compromise. However, the exact catecholamine(s) responsible for initiating this sequence of events has not been identified, and the events that cause catecholamines to be released during or after an initial insult of an SAH remain unclear. Regardless of initiating factors, the catecholamine surge is thought to stimulate a sympathetic storm (Parr, 1996). A sympathetic storm occurs when a rapid increase in ICP (such as that which occurs following aneurysm rupture) triggers a catecholamine surge. The sympathetic storm is manifested by episodic alterations in body temperature, blood pressure, heart and respiratory rate, pupil size, and level of consciousness coinciding with hyperhydrosis, excessive salivation, and extensor posturing (Young, Finn, Pellegrini, Soloaga, & Bruetman, 2006). After these physiologic alterations begin, the patient is at risk for cardiopulmonary dysfunction. Unfortunately, clinical management for patients with SAH often includes maintaining systolic BP under 120 mm Hg to prevent further rupture of the unsecured aneurysm during the period of time before the repair. Some theorize this therapy may increase the risk to cardiac muscle by preventing much needed oxygen from perfusing the heart (Parr, 1996).

Regardless of the cause, cardiopulmonary dysfunction may manifest in several ways. Troponin elevation, a sensitive and widely accepted marker of cardiac injury, has been associated with an increased risk of cardiopulmonary complications following SAH, as well as an increased risk for delayed cerebral ischemia and poor functional outcome at discharge (Naidech et al., 2005). Elevated cardiac troponin I (cTnI) values have been noted frequently in intracranial hemorrhage and are independently associated with higher in-hospital mortality (Hays & Diringer, 2006). Cardiopulmonary dysfunction also may manifest following SAH as abnormal ECG readings that may show bradycardia, cardiac output prolongation, diffuse S-T changes, and ventricular ectopy (Zaroff, Rordorf, Newell, Ogilvy, & Levinson, 1999). All of this article's case study subjects had early and significantly elevated troponin, creatine phosphokinase (CPK), and CPKMB levels as well as abnormal ECG readings.

Elevated laboratory values and abnormal ECG readings often signal the time at which transthoracic ECHO should be performed to fully evaluate the extent of myocardial dysfunction. This test typically reveals areas of ischemia and dilation of the heart and evaluates heart function by determining EF and wall motion. In each of the case studies presented here, patients underwent ECHO evaluation, which demonstrated varying degrees of inadequate cardiac function. Inadequate cardiac function in this situation often is referred to as "stunned myocardium" and describes low cardiac output that initially occurs after SAH and then resolves (Donaldson & Pritz, 2001). Interestingly, despite the incidence of

cardiopulmonary dysfunction in persons with SAH, it is rare to find actual coronary artery disease (Donaldson & Pritz).

Cardiopulmonary dysfunction is just one of many complications that may occur following SAH. Patients with a ruptured aneurysm are at increased risk for vasospasm, pneumonia, and skin breakdown. M. M., for example, demonstrated indications of septic shock, increased cardiac output, positive blood cultures, and decreased blood pressure, but also had evidence of myocardial ischemia with ECG and enzyme changes (Bernardini & DeShaies, 2001). In patients with SAH, the complication of primary concern to neuroscience practitioners is vasospasm, which can extend the hospitalization period following an “uncomplicated” SAH beyond 2 weeks (Kosty, 2005; Urbaniak, Merchant, Amin-Hanjani, & Roitberg, 2007). Some theorize that patients who experience cardiac complications are at greater risk for cerebral vasospasm (Lee et al., 2006), although the cause for this relationship is unknown. Regardless, cardiopulmonary dysfunction complicates the treatment of vasospasm because medications that treat cerebral vasospasm can worsen cardiopulmonary dysfunction and vice versa. Vasopressors, for example, are known to elevate heart rate and cause vasoconstriction. This may cause increased workload or oxygen demand on an already irritated myocardial muscle. Using HHH therapy for vasospasm becomes increasingly difficult or impossible if there is continued pulmonary vascular congestion or low cardiac output (Kosty). On the other side, hypoperfusion from cardiopulmonary function affects the brain’s ability to maintain tissue perfusion necessary to prevent additional damage to an already acutely injured cerebrum (Yarlagadda et al., 2006). The challenge for neuroscience practitioners is to concomitantly manage cerebral and cardiopulmonary conditions, optimizing blood flow to tissue in one area without administering treatment that will decrease perfusion in the other.

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Nursing Implications

As these cases illustrate, a multitude of complications can occur after an aneurysm ruptures. The presence of these complications often prolongs the length of hospitalization (Brouwers, 1989; Urbaniak et al., 2007). The three women featured in this article’s case studies had nearly twice the normal hospital length of stay for SAH. None experienced a quick resolution of their cardiac dysfunction. Complications such as cardiopulmonary dysfunction often are considered “nonneurologic” and, consequently, a secondary priority; yet changes to heart and lung function often are initiated by an aneurysm rupture and their course and treatment can complicate efforts to preserve neurologic function. Nurses are in a prime position to understand how the initial cerebral insult affects multiple physiologic systems and organize and implement interventions that are designed to improve overall physical function and maximize neurologic and other system outcomes.

The first step in this process is to recognize that cardiopulmonary dysfunction can be a common and often serious complication following aneurysmal SAH. The ability to identify this complication is made more difficult because risk factors are few. The patients in this

series were at different decades of life and none had a history of cardiac disease before their SAH. For this reason, nurses should suspect patients admitted with aneurysmal SAH may exhibit cardiopulmonary dysfunction regardless of their risk status and use lab and ECG results as well as vital signs to trend actual or potential cardiopulmonary compromise. Once a nurse is aware of the potential for this complication, it is vital to know which diagnostic tests are most efficient and accurate in identifying the existence and extent of cardiopulmonary dysfunction. Positive findings on initial tests and changes in a patient's clinical condition can help guide clinicians to order further testing, such as ECHOs, as needed. In addition, nurses should pay attention to heart rhythm, pulmonary status, intake and output, and ICP because increased ICP can potentiate more catecholamine release, furthering cardiac damage (Parr, 1996). Finally, nurses should recognize that it is uncommon for patients to have the symptoms typically associated with acute myocardial ischemia such as chest pain and shortness of breath. Consequently, preliminary tests of cardiac dysfunction, such as cardiac enzymes, ECG, and chest X rays, should be considered for early evaluation. A high index of suspicion on the part of the nurse is crucial in preventing secondary cardiac damage.

Any change in a patient's cardiovascular status should be quickly reported. Nurses should be prepared for additional cardiac monitoring and medications that concomitantly prevent arrhythmias or augment cardiac output and assure adequate cerebral blood flow. Treatment must be titrated so medications that decrease cardiopulmonary dysfunction do not interfere with treatment for vasospasm; administering diuretics to treat acute pulmonary edema, for example, could result in lowering CVP to a level that puts patients at risk for vasospasm (Strippler et al., 2006).

Psychological support also is important for the families of patients who sustain SAH (von Vogelsang, Wengstrom, & Forsberg, 2004). SAH rehabilitation is long and strenuous and may be more so if cardiac complications increase length of stay. When complications arise, recovery for patients and families becomes more difficult. Nurses need to anticipate these challenges and follow up with the appropriate ancillary staff. Education on cardiac dysfunction after SAH is important for families and patients, especially regarding the additional monitoring required to evaluate full cardiac recovery after discharge (Dunleavy, Finck, Overstreet, & Presciutti, 2005).

Summary

The patients included in these case studies represent a subset of patients who experience cardiac dysfunction following SAH and the challenges such patients present to clinicians in the NVICU. Fortunately, the patients presented here experienced good outcomes from neurological and cardiac standpoints. Patients with SAH with profound cardiac dysfunction may not be as fortunate and may not recover. Perhaps through diligence as clinicians, we can improve the potential for positive outcomes for acute SAH patients with cardiovascular compromise (Crago et al., 2004).

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Table 1

Cardiac Enzyme Levels During M. M.'s First Week of Hospitalization

Day	Troponin I (ng/ml)	TCPK	CPKMBRI	CPKMB
Day 0	0.26	114	2.0	2.3
Day 1	<0.10	209	1.3	2.7
Day 4	8.03 ^a	302	5.0	15.2
Day 5	6.94 ^a			
Day 6	2.76 ^a			
Day 15	1.30	51		
Day 16	0.22	62		

^aDay 4–Day 6. The troponin I (normal value < 0.10 ng/ml) levels show a significant increase and possible cardiac damage. Day 0 = Admission. CPKMB = creatine phosphokinase–MB; CPKMBRI = creatine phosphokinase–MB Index; TCPK = total creatine phosphokinase.

Table 2

J. N.'s Troponin and Cardiac Enzyme Levels for 1 Week Postadmission

Day	Troponin I (ng/ml)	TCPK	CPKMB
Day 0	5.26 ^a	184	6.5
Day 1	10.08 ^a	290	8.7
Day 1	9.85 ^a	364	7.3
Day 1	6.44 ^a	307	6.0
Day 2	4.3 ^a	799	1.6
Day 3	2.35	1,236	0.9
Day 4	0.95	726	0.5
Day 5	0.43	542	0.8

^aDay 0–Day 2. The troponin I (normal value < 0.10 ng/ml) levels show a significant increase and possible cardiac damage. Day 0 = Admission. CPKMB = creatine phosphokinase–MB; TCPK = total creatine phosphokinase.

Table 3

J. T.'s Cardiac Enzyme Trends During 5 Days in the Neurovascular Intensive Care Unit

Day	Troponin I (ng/ml)	TCPK	CPKMBRI	CPKMB
Day 0	13.44 ^a	388	5.4	21.0
Day 1	3.01 ^a	764	1.1	8.7
Day 2	1.62 ^a	820	0.4	3.4
Day 3	0.61	410	0.2	0.9
Day 4	0.32	251	0.2	0.5

^aDay 0–Day 2. The troponin I (normal value < 0.10 ng/ml) levels show a significant increase and possible cardiac damage. Day 0 = Admission. CPKMB = creatine phosphokinase–MB; CPKMBRI = creatine phosphokinase–MB Index; TCPK = total creatine phosphokinase.