

Management for the case of twice ruptured anterior communicating artery aneurysm: From nursing perspective

Zhou Xiaoying, RN, Cao Xicai, MMed, and Liu Sheng, MMed

The anterior communicating artery (ACoA) is the most common site of cerebral aneurysms, accounting for as much as 36% of aneurysms. Microsurgical clipping and coil embolization are treatment modalities for ruptured and nonruptured cerebral aneurysms. Compared with surgical clipping, coil embolization has a relatively lower mortality and incidence of cognitive impairment in patients. However, successful management of the patient with twice ruptured ACoA aneurysm is facing critical challenges. This article has described a case of twice ruptured aneurysm with the first rupture occurring when the patient was admitted and the second rupture occurring during coil embolization. Perioperative nursing assessment, monitoring, intervention, patient teaching, and the nurse's role are discussed from a nursing perspective. (J Vasc Nurs 2013;31:107-110)

The anterior communicating artery (ACoA) is the most common site of cerebral aneurysms, accounting for as much as 36% of aneurysms.¹ Microsurgical clipping and coil embolization are treatment modalities for ruptured and nonruptured cerebral aneurysms according to the anatomy, the vessel tortuosity, and other issues. A detailed morphologic analysis should be performed to select the best treatment modality. After the International Subarachnoid Aneurysm Trial, endovascular treatment with coils is now the first-line treatment in the management of ruptured aneurysms. Several studies have reported good outcomes.²⁻⁴ Compared with surgical clipping, coil embolization has a relatively lower mortality and incidence of cognitive impairment in patients. However, a challenge of endovascular treatment is how to successfully manage a case of twice ruptured aneurysm. Nursing care of patients who need to recover from twice ruptured aneurysm also faces many challenges. This article will describe a case of twice ruptured aneurysm with the first rupture occurring when the patient was admitted and the second rupture occurring during coil embolization. Perioperative nursing assessment, monitoring, intervention, patient teaching, and the nurse's role are discussed from a nursing perspective.

CASE REPORT

A 51-year-old woman was brought to the General Hospital with a sudden onset of severe headache, nausea, and vomiting.

From the Radiological Department, Tianjin Medical University, General Hospital, Heping District, Tianjin, China.

Corresponding author: Zhou Xiaoying, RN, 154 Anshan Rd, Heping, Tianjin, China (E-mail: zhouxiaoying13141314@yahoo.com.cn).

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She was transferred to the neurosurgical intensive care unit for hemodynamic neurologic monitoring and assessment. Her initial vital signs included a heart rate of 100 beats/min and a blood pressure (BP) of 154/70 mm Hg. Her neurologic Glasgow coma score (GCS) was 15; pupils were equal in diameter at 3 mm and reacted to light. There was slight nuchal rigidity, which is a sign of meningeal irritation. Verbal response was oriented to person, place, and time, and motor response followed commands with 5/5 motor strength. Emergency computed tomography (CT) scan showed a subarachnoid hemorrhage (SAH). According to the Fisher grading system, she had a grade 2 SAH. CT angiography further revealed an ACoA aneurysm (3 × 2 mm). The patient's history was significant for hypertension of 20 years, which was controlled by an antihypertensive agent; asthma of 31 years; and infectious pseudo-rheumatism of less than 1 year. She also had an allergy to penicillin and sulfanilamide.

Because the patient had increased intracranial pressure (ICP) due to clinical manifestations of severe headache, vomiting, and slight nuchal rigidity, a treatment schema was decided on immediately to reduce ICP by intravenous dehydrants, fluid infusion, and hemostatics, and to prevent rebleeding, vasospasm, seizure, and stress ulcer. A corresponding nursing intervention also was implemented.

Monitor for signs and symptoms of increased intracranial pressure

Assess best eye-opening response, motor response, and verbal response. In addition, assess for changes in vital signs, such as heart rate changes, respiratory irregularities, increase in BP, and widening pulse pressure (systolic BP is kept at 120-150 mm Hg, informing the neurosurgeon if it is >170 mm Hg). Assess pupillary responses and inspect the pupils to evaluate size, configuration, and reaction to light. Compare with both similarities and differences for eyes. Note any other signs and symptoms: vomiting, headache, subtle changes (eg, lethargy, restlessness, forced breathing, purposeless movements, changes in mental activity). These signs may be the earliest indicators of ICP changes.

Reduce intracranial pressure

The head of the bed should be elevated to 30 degrees. Slight head elevation can aid venous drainage to reduce cerebrovascular congestion, thereby decreasing ICP. Carotid massage, neck flexion, and extreme rotation should be avoided because these can increase ICP. If needed, the neurosurgeon can be consulted about stool softeners. Maintain a quiet, calm, environment. Schedule several lengthy periods of uninterrupted rest daily. Cluster necessary procedures and activities to minimize interruptions. These measures promote rest and decrease stimulation. Avoid sequential coughing and repositioning, because these can cause a cumulative increase in ICP. Collaborate with the neurosurgeon for pharmacologic therapy, which may include sedation, anticonvulsants, osmotic diuretics, and so forth. Carefully monitor hydration status and evaluate fluid intake and output. Carefully administer via infusion pump to prevent overhydration, which will increase ICP. Provide bedside instruction on accurate evidence-based information, focusing on positive aspects to decrease anxiety in the patient and family.

As a result of these interventions, the patient's vital signs were stable. The GCS was 15. Both pupils were equal in diameter at 2.5 mm and reacted to light. There was no longer nausea and vomiting, but severe headache and slight nuchal rigidity were as before. The other neurologic assessment results were not abnormal. Two days later, an endovascular embolization was scheduled to prevent the patient from rebleeding. Informed consent was obtained from the patient.

ENDOVASCULAR PROCEDURE

One hour before the procedure, the patient was administered 10 mg of nimodipine intravenously at a rate of 1.2 mg/mL via infusion pump. Endovascular embolization was under general anesthesia. Diagnostic angiography was performed via the right femoral artery through a 6F introducer. A right carotid angiogram (Figure 1) showed a small (3 × 2 mm) aneurysm in the ACoA. A bolus of intravenous heparin was followed by continuous infusion via the catheter flushing system at a concentration of 2 IU/mL. A 6F guiding catheter was placed in the distal right internal carotid artery to obtain a stable position. A microcatheter with 2 tip markers and a microguide wire were used for superselective catheterization of the aneurysmal sac. A coil was then deployed into the aneurysm. An angiogram (Figure 2) was performed immediately and showed contrast to extravasation; subsequently, the patient's BP was elevated. Intraoperative perforation was considered on insertion of the first frame coil. When aneurysm rupture occurred, measures were taken to reduce ICP by immediately reducing BP, continuing to deploy the second coil to limit the extent of the hemorrhage and reversing heparin with protamine at the same time. The sheath was kept in place 2 hours. To decrease the ICP, an external ventricular drainage was inserted to remove cerebrospinal fluid (CSF). After the procedure, an emergency CT of the head showed that the hemorrhage in the SAH was somewhat increased compared with before the procedure, without forming a hematoma and the ventricle clearly displayed. The patient was returned to the neurosurgical intensive care unit for observation. As a result of the timely



Figure 1. Right carotid angiogram shows a small (3 × 2 mm) aneurysm in the ACoA.

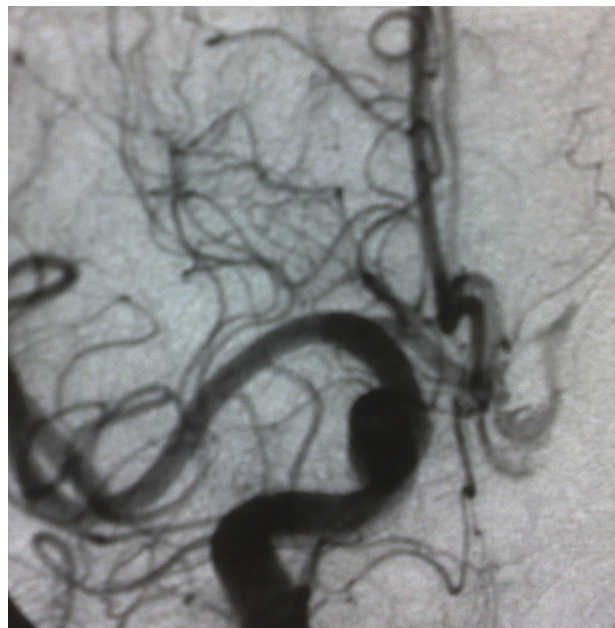


Figure 2. Angiogram shows contrast to extravasation during coil embolization. The aneurysm was perforated on insertion of the first frame coil.

treatment, there was only a small amount of rebleeding in the SAH and intraoperative bleeding was controlled.

POSTPROCEDURE COURSE

The patient must remain intubated and sedated for a prolonged period after procedure. Administration of nimodipine, a calcium channel blocker, and use of triple H therapy are both widely accepted interventions after coiling. These measures seem to be the most effective way to decrease spasm in the

cerebral vascular bed after SAH, especially when used prophylactically. The patient was administered nimodipine intravenously at a rate of 1.2 mg/h via infusion pump for 7 days, and then an oral dosage of 30 mg was given twice per day. The 3 components of triple H therapy are hypertension, hypervolemia, and hemodilution. These strategies are all designed to increase cerebral perfusion pressure, improve blood flow to the brain, and decrease the risk of ischemia. A recommendation for target systolic BP may be as high as 200 mm Hg.⁵ Hypervolemia is produced by infusing sufficient intravenous fluids to optimize a patient's Starling curve. Careful hemodynamic assessment must be performed. The goal for hemodilution is a hematocrit of 0.33 to 0.38, which is achieved by dilution with intravenous fluids. Low-molecular-weight heparin was routinely subcutaneously injected for 3 days, and dual antiplatelet drugs (75 mg clopidogrel and 300 mg aspirin daily) were given afterward. An antibiotic was given for 5 days. After removal of the sheath, a 20-minute manual compression was performed, a gauze roller and elastic bandage for hemostasis covered the access site, and a 2-kg salt bag was pressed above the bandage for 6 hours. The right lower extremity was kept immobile for 24 hours. The elastic bandage was removed after 24 hours.

Nursing intervention should be followed as soon as the procedure is completed. The patient was in sedation status with a BP of 160/90 mm Hg, endotracheal intubation, and assisted breathing with ventilator. Double dorsal arteries of the foot were beating well, and the skin temperature was normal. The patient's pupils were equal in diameter at 2 mm and slightly reacted to light. The head of the bed was elevated to 15 degrees. The drip chamber of CSF was 10 to 15 cm higher than the lateral ventricles. The drainage tube was kept patent. Twenty milliliters of bloody CSF were drained after the procedure. A limit of ventricular drainage (eg, 5-10 mL/h) was used to avoid excessively rapid CSF drainage. Close attention was paid to the drainage amount. An obstruction of circulating CSF will occur if the amount is more than 450 mL/day. The drainage amount of CSF is closely related to ICP and can guide pharmacologic therapies.

The following morning, the patient was conscious, and vital signs were stable after extubation with a GCS of 15. On day 3, 300 mL of light bloody CSF was drained via external ventricular drainage with slight nuchal rigidity. The pupils were equal in diameter at 2.5 mm and reacted to light. Ocular movement was normal. Motor response followed commands with 5/5 motor strength. The lumbar puncture (LP) was performed regularly to measure ICP, releasing CSF to decrease ICP. Shunting of CSF through a lumbar drain after an SAH markedly reduces the risk of clinically evident vasospasm and its sequelae. Its beneficial effects are probably mediated through the removal of spasmogens that exist in the CSF.⁶ This technique shows favorable effects on numerous neurologic outcomes and prognosis.⁷ The first LP of the patient was performed and showed an elevated pressure of CSF of 450 cmH₂O, releasing 50 mL of CSF. On day 4, another LP showed a pressure of 320 cmH₂O, releasing 20 mL of CSF. On day 5, the patient's status was improved; both headache and nuchal rigidity were alleviated. On day 7, the most interesting phenomenon was related to the hyperthermia of 39.1°C. There was no evidence of inflammation, possibly because the intracranial hematoma was being absorbed. The patient's temperature decreased to 37.7°C via administration of dexamethasone. During the next few days, the patient's status

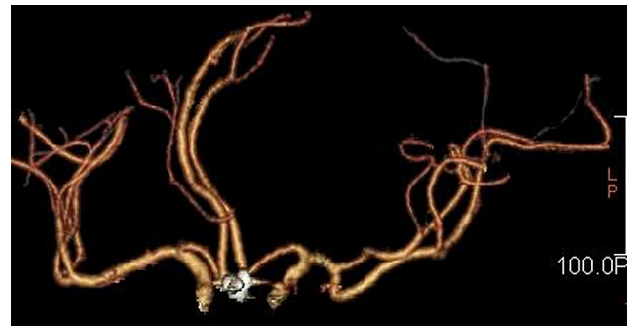


Figure 3. CT angiography shows complete occlusion of the ACoA aneurysm sac.

continued to improve. The patient was scheduled to undergo CT angiography on day 8 after initial therapy. CT angiography (Figure 3) confirmed complete occlusion of the ACoA aneurysm sac, obviously decreasing the hematoma. Clinical evaluation was performed before the patient's discharge from the hospital. The patient was conscious with an oriented verbal response, a good mental state, stable vital signs, a muscle strength grading of 5, and no increased muscle tension. The double-side Babinski sign was negative, and the patient no longer had a headache and resistance to flexion of the neck. On day 11, CT showed a postoperative change in the ACoA aneurysm, and the intracranial hematoma was almost gone. The patient was discharged on day 21 without postoperative deficits. A discharge guideline stated that the patient should rest, perform appropriate activities, and eat a nutritional diet; in addition, mood fluctuation should be avoided, with follow-up if there were any changes.

DISCUSSION

ACoA is an important component of intracranial arteries, which is the main channel for balancing blood flow of both sides of the anterior cerebral artery. It is also the most common location for intracranial aneurysms.⁸ The characteristic initial symptom of patients is SAH. Patients who outlive the original hemorrhage face a multitude of potential complications in the acute phase. The earlier that treatment can be instituted, the lower the risk of rebleeding in patients.⁹ Using an endovascular coil embolization within 48 hours of the initial hemorrhage is imperative to minimize the complications of both rebleeding and vasospasm.⁵ However, the rate of intraoperative rupture is 4.4%,¹⁰ another serious complication of endovascular treatment.² Intraoperative rupture can be remedied, as in the described patient who was cured successfully without postoperative deficits.

Optimizing care for the patient with coil embolization of an aneurysm requires a well-orchestrated team that agrees to work together to achieve the goals established at each phase of illness. Nurses play a pivotal role in terms of perioperative assessment, monitoring, intervention, patient teaching, and collaboration with neurosurgeons for pharmacologic therapy. Nurses must detect any changes in the level of consciousness, a headache, a period of disorientation, inappropriate behavior, language impairment, hemiparesis, and seizures. If unrecognized, deficits can become permanent disabilities. In addition, nurses must understand and respond to the needs of the patient in a prompt manner; patients with twice ruptured ACoA aneurysm who have experienced ICP especially need to receive rapid initiation of treatment to avoid a devastating outcome.

CONCLUSIONS

Coil embolization for ACoA aneurysm has become increasingly common. However, successful management of the patient with twice ruptured ACoA aneurysm is facing critical challenges. Nurses play a key role in the course of patient treatment. From the nursing perspective, nurses will help provide patients prompt and appropriate care.

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