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Aneurysmal subarachnoid hemorrhage

Follow the guidelines

RJ, 51, PRESENTS TO THE ED complaining of the worst headache of her life, nausea, and photophobia. Computed tomography (CT) of the brain shows blood in the subarachnoid space. The blood resulted from a ruptured aneurysm.

Aneurysmal subarachnoid hemorrhage (aSAH) is frequently deadly, and outcomes are poor for many survivors. The American Heart Association and American Stroke Association have updated their guidelines on the management of patients with aSAH. This article reviews the current guidelines, which cover incidence, prevalence, treatment, and prevention of complications associated with aSAH.

John W. Karapelou, CMI/Phototake

Pressure on a weakened wall

Cerebral aneurysms develop when an artery wall becomes thin and weak. When a thinned arterial wall ruptures, blood from the ruptured aneurysm accumulates in the subarachnoid space.[1]

Many factors are known to contribute to aneurysm formation, including smoking, hypertension, and connective tissue disorders.[1] One process recently recognized in aneurysm formation and growth is inflammation. Use of statin drugs and calcium channel blockers may inhibit this process, but research is needed to confirm that these drugs are beneficial for aSAH prevention.[2]

Various patient characteristics affect the probability of aneurysm rupture. Older adults, women, and people of Black or Hispanic ethnicity have higher rates of aSAH.[2,3] (For details, see *Risk factors for aSAH*.)

Patient and aneurysm characteristics can interact to increase the risk of rupture. For example, the risk of aSAH increases when the aneurysm is causing symptoms, such as abnormal extraocular movements associated with deficits in cranial nerves III, IV, and VI.[1]

Size matters too: In general, aneurysms that are likely to bleed are larger than 7 mm. In hypertensive patients who smoke, however, aneurysms that hemorrhage tend to be smaller than those in nonsmoking patients who are hypertensive or

those in normotensive patients who smoke.[2]

An estimated 14.5 per 100,000 people are diagnosed with aSAH, but the actual figure is undoubtedly higher because 12% to 15% of patients die before they can get medical care. The statistics on patients who experience aSAH are grim: About 32% of those who reach the hospital die. Of all patients diagnosed with aSAH in a hospital setting who survive the initial hemorrhage, 50% to 66% suffer complications.[1,3]

Research has identified several factors contributing to aSAH morbidity and mortality. (See *Predictors of poor patient outcomes*.) In addition, the likelihood of survival after aSAH may be affected by sex and race. Some studies suggest that men are more likely to survive than women. Survival rates are also higher for Whites than for Blacks, American Indians, Native Alaskans, Asians, and Pacific Islanders.[2]

To prevent aSAH, hypertension should be treated, and patients should refrain from smoking and excessive alcohol use. Diets with high vegetable content should be encouraged because one study showed aSAH decreased as vegetable intake increased.[2,4] In patients with a known, unruptured aneurysm, the healthcare provider will consider the shape and hemodynamics of the aneurysm when planning treatment. Patients who have a family or personal history of aSAH can be monitored for new aneurysms or blood flow within a treated aneurysm. Screening studies for these patients should be noninvasive to reduce the risk of complications.[2,5]

The setting in which a patient with aSAH receives treatment has also been shown to impact patient outcome. Large teaching institutions and hospitals with endovascular capabilities are associated with lower morbidity and mortality. This seems to be especially true the more frequently a facility employs neurointerventional procedures, such as endovascular coil embolization for cerebral aneurysms. Patients tend to have better outcomes in hospitals that treat more than 35 patients with aSAH per year.[2]

Patients whose presentation suggests aSAH should be diagnosed as quickly as possible to improve the chances of survival and a good recovery. For optimal care, they should be admitted to a facility with neurointerventional capabilities and a neurologic ICU, even if they must be transferred.[2]

Risk factors for aSAH[2,23]

Modifiable

- Smoking
- Drug and alcohol abuse
- Hypertension
- Extremely low BMI.

Nonmodifiable

- One or more first-degree family members with cerebral aneurysm
- Specific genetic syndromes, such as autosomal dominant polycystic kidney disease and Ehlers-Danlos syndrome
- Family history of aneurysm
- Personal history of unruptured cerebral aneurysm
- Family or personal history of aSAH

Predictors of poor patient outcomes[2]

- significant acuity at presentation (the predominant predictor)
- aneurysm rebleeding
- comorbidities
- increased age
- global cerebral edema at presentation
- presence of intraventricular and intracerebral hemorrhage
- symptomatic vasospasm
- delayed cerebral infarction (particularly more than one)
- systemic complications, such as fever, hyperglycemia, anemia, pneumonia, sepsis
- aneurysm-specific characteristics: larger size, anatomical locations that are difficult to treat (such as posterior circulation), irregular shape.

Recognizing signs and symptoms

The headache of aSAH typically begins abruptly. Like 80% of patients experiencing aSAH, RJ described her headache as the worst of her life. However, some patients (10% to 43%) have a less intense headache, known as a *sentinel headache*, before aneurysm rupture. This type of headache is associated with an increased risk that the aneurysm will rebleed in the absence of treatment.[2]

Meningeal signs and symptoms, which develop when the hemorrhaged blood irritates the meninges, include photophobia (light sensitivity), blurred vision, nausea, vomiting, nuchal rigidity (stiff neck), and pain in the neck and back.[1] Other signs and symptoms include altered level of consciousness (LOC), cranial nerve deficits, and focal neurologic deficits, such as right hemiparesis.

Patients may also experience vital sign changes, such as hypertension. Seizures, a sign of aSAH in 20% of patients, usually occur in the first 24 hours.[1,2] Immediately placing patients on antiepileptic drugs (AEDs) and initiating seizure precautions is standard procedure.

Symptomatology is an important issue in aSAH diagnosis because outcomes are negatively affected if the diagnosis is delayed or missed. Approximately 12% of aSAH patients aren't diagnosed correctly.[2] Failure to perform a noncontrast head CT, the gold standard for diagnosing aSAH, is a common reason for a missed diagnosis.

Diagnostic testing

Noncontrast CT of the brain has nearly 100% sensitivity for diagnosing aSAH in the first 72 hours after rupture. Lumbar puncture may be performed if presenting signs and symptoms are consistent with aSAH but the CT fails to show hemorrhage. Computed tomography angiography (CTA) and magnetic resonance imaging (MRI) can also be used for some populations; for

example, patients at risk for radiation overexposure.[2]

After aSAH is diagnosed, the aneurysm needs to be located. If the patient may be a candidate for endovascular treatment, the surgeon must visualize the aneurysm's precise shape. Cerebral digital subtraction angiography (DSA) is the most commonly used tool for locating the site of rupture.

In CTA, another option, a contrast medium is used with CT to create images of the cerebral arteries. However, using CTA alone to locate a ruptured aneurysm remains controversial. Research indicates that CTA may lack the sensitivity to identify aneurysms smaller than 3 mm. Based on current evidence, CTA may not provide as much information as DSA.[2,5,6]

After RJ's aSAH is diagnosed via noncontrast CT, she has two- and three-dimensional DSA, which is more sensitive for identifying aneurysms than two-dimensional DSA alone.[2] DSA shows that her aneurysm is located on her right posterior communicating artery (PCOM). (See *Where to find cerebral aneurysms*.)

Although RJ has just been diagnosed, she's already received

Where to find cerebral aneurysms[1]

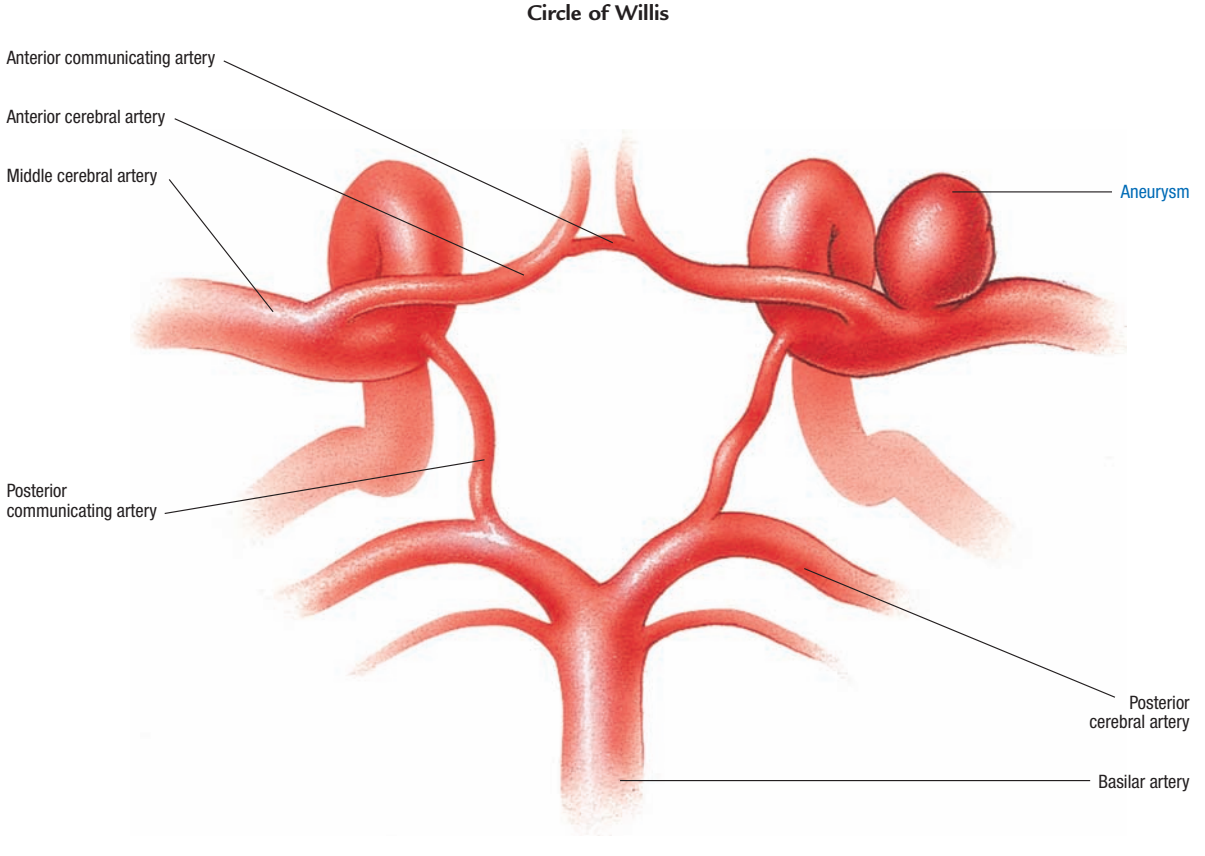
Most cerebral aneurysms form at the bifurcations and branches of the large arteries at the base of the brain, called the circle of Willis, which is located within the subarachnoid space. Arteries in the circle of Willis may be divided into anterior circulation (carotid portion) and posterior circulation (vertebrobasilar portion). The arteries most likely to develop aneurysms are as follows:

Anterior circulation

Anterior communicating artery (ACOM)
Middle cerebral artery (MCA)
Anterior cerebral artery (ACA)
Posterior communicating artery (PCOM)

Posterior circulation

Basilar artery (BA)
Vertebral artery (VA); not shown



radiation from a CT scan and DSA and she's likely to undergo more cerebral radiation throughout her treatment course. Complications from receiving so much radiation, such as dermal erythema, alopecia, and cancer, are a significant threat.[2,7] Her providers and the radiology staff closely monitor RJ's radiation exposure and minimize the amount of radiation she receives by using non-radiation tests whenever possible and spacing tests requiring radiation apart.[7]

Grading aSAH

After diagnosis, aSAH should be graded quickly to aid in predicting patient outcome and making treatment decisions. One frequently used system is the Hunt-Hess classification system, which grades the patient's aSAH based on signs and symptoms.[1,2] RJ is a Hunt-Hess Classification Grade II, which is associated with cranial nerve palsy, moderate-to-severe headache, and nuchal rigidity. (See *Grading aSAH with the Hunt-Hess classification system* for a complete description.)

While RJ awaits treatment, the nurse assesses her vital signs, performs neurologic assessments as prescribed, initiates seizure precautions according to facility policy, and closely monitors her. Standard nursing interventions include:

- ensuring bed rest with head of bed elevated 30 degrees
- initiating continuous cardiac monitoring
- documenting intake/output meticulously
- providing prophylaxis for venous thromboembolism
- ensuring a low level of external stimulation.[1]

Immediately report any changes in the patient's condition. Acute complications of aSAH, which are discussed in detail below, can develop before definitive treatment of the aneurysm is initiated.[3]

To prepare a patient for CT, CTA, or DSA, the nurse explains the procedure and tells the patient that he or she will need to lie still to optimize image quality. Iodinated contrast is used in CTA and DSA, so the nurse assesses for allergies, renal dysfunction, and metformin use before the procedure. Patients of childbearing potential should also be screened for pregnancy. The nurse explains that venous access is needed for contrast in CTA, and arterial access is used in DSA.

After the procedure, the nurse observes for signs and symptoms of contrast reactions, such as angioedema or oliguria. For patients who undergo DSA, the arterial access site (usually the femoral artery) is closely monitored for active bleeding and expanding hematoma. The neurovascular status of the access extremity is also frequently assessed. The patient will be positioned supine, with the head of the bed flat and the affected leg immobilized, as prescribed, if manual or mechanical compression was used to achieve hemostasis.[1,3,8]

Grading aSAH with the Hunt-Hess classification system[1]

Grade	Signs and symptoms
I	Asymptomatic or minor headache, slight nuchal rigidity
II	Moderate-to-severe headache, nuchal rigidity, no neurologic deficit except cranial nerve deficits
III	Minor focal deficit, lethargy or confusion
IV	Moderate-to-severe hemiparesis, stupor
V	Coma, decerebrate rigidity, moribund appearance

Risk factors for seizures after aSAH[2,9]

Early seizure

- ruptured MCA aneurysm
- aneurysm rebleeding
- cerebral infarction
- high-grade aSAH
- large subarachnoid thrombus
- intracerebral hemorrhage
- patient history of hypertension

Late seizure

- ruptured MCA aneurysm
- cerebral infarction
- intracerebral hematoma
- previous seizure
- refractory hypertension.

It's complicated, part I: Before treatment

Throughout diagnostic testing and treatment, the nurse must closely monitor the patient for complications and intervene appropriately. ***Hydrocephalus*** develops in 15% to 87% of patients soon after aSAH because blood from the hemorrhage obstructs the subarachnoid villi and prevents cerebrospinal fluid (CSF) reabsorption.[1,2]

After her CT, RJ becomes lethargic and confused. Besides locating the aSAH, the CT revealed communicating hydrocephalus. A ventriculostomy is performed for external ventricular drainage (EVD). EVD allows clinicians to monitor intracranial pressure (ICP) and control elevated ICP by periodically draining CSF.

As an alternative to ventriculostomy, a lumbar drain can be placed. Although current evidence suggests patients with lumbar drains have less vasospasm than patients with EVD, caution is advised. The guidelines note the risk of tissue shift and herniation after lumbar drain insertion in patients with severe aSAH. In some cases, lumbar drain placement results in brain herniation and death.[2,3]

The nurse monitors RJ's ICP and drains CSF as prescribed to manage ICP. The nurse assesses RJ for signs and symptoms of infection (such as fever and cloudy CSF) and aneurysmal rebleeding and elevated ICP (such as sudden, severe headache, altered level of consciousness, new neurologic deficits, and fresh blood in the CSF).[1]

Early seizures, which develop in 6% to 26% of patients with aSAH, occur in the first 24 hours after aSAH.[9] (See *Risk factors for seizures after aSAH*.) This is why initiation of AED therapy immediately after diagnosis is standard practice. Although not well researched, it's an accepted therapy because early seizures can compound neurologic injury, and rebleeding is a concern if the aneurysm hasn't been repaired. Because serious adverse reactions are associated with AEDs, closely monitor patient response and review lab values for therapeutic serum drug levels and signs of drug-related complications, such as agranulocytosis or thrombocytopenia. Assess for potential drug interactions.

The most serious acute complication of aSAH is ***rebleeding,*** which is a risk until the aneurysm can be definitively treated. Patient morbidity and mortality is drastically increased when an aneurysm rebleeds.[2]

Almost half of aneurysms that rebleed do so within 6 hours of the initial bleed. The earlier an aneurysm rebleeds, the poorer the patient prognosis. A higher risk of rebleeding is associated with:

- large aneurysms
- poor neurologic condition
- decreased LOC at time of rupture
- history of sentinel headache
- systolic BP over 160 mm Hg
- a long delay before the aneurysm is obliterated.[2]

Several measures can be employed to prevent rebleeding until definitive treatment of the aneurysm is complete. RJ remains on bed rest with minimal external stimulation. Preventing hypertension is commonly believed to reduce the risk of rebleeding. Research into exact BP limits and optimal medical therapy to maintain those limits is lacking, but the guidelines note that keeping systolic BP below 160 mm Hg is "reasonable," because higher pressures may be a risk factor for rebleeding.[2] BP should be managed with a drug that can be easily titrated, such as nicardipine, and maintained at a level that's sufficient to sustain cerebral perfusion pressure (CPP) and prevent acute ischemic stroke.[1,2]

An estimate of the adequacy of cerebral circulation, CPP is calculated by subtracting ICP from mean arterial pressure. In healthy adults, a normal CPP is 70 to 100 mm Hg.[1] Before treatment for aSAH, CPP should be kept within normal limits.

RJ's ICP remains unstable despite her EVD. Because she couldn't tolerate lying flat prior to her DSA, definitive treatment was delayed, placing her at increased risk for rebleeding.

For patients whose definitive treatment is delayed, an antifibrinolytic such as aminocaproic acid or tranexamic acid may be prescribed to decrease the risk of rebleeding. Although not currently FDA-approved for this use, these drugs are considered a reasonable precaution for up to 72 hours in cases of delayed treatment under the guidelines.[2]

Both drugs carry warnings regarding the increased incidence of certain neurologic complications when used for patients with aSAH.[2,10,11] One study identified higher rates of hydrocephalus possibly attributable to this therapy.[2,10-13] However, because neurologic complications are associated with aSAH and related diagnostic testing, the role of these drugs in development of these complications is unclear and research is ongoing.

Dilute aminocaproic acid as directed and avoid rapid infusion of either drug. Monitor the patient closely: Bradycardia has been observed in patients receiving aminocaproic acid, and both drugs can cause gastrointestinal upset and hypotension. Also watch for signs and symptoms of systemic thrombosis and monitor lab values as prescribed.[2,10,11]

Surgery and more

The only way to definitively treat a cerebral aneurysm is to occlude it from the healthy circulation. Two methods that achieve definitive treatment are surgical clipping and endovascular occlusion.[1,2]

- *Surgical clipping* is performed via open craniotomy. The surgeon places a clip at the base of the aneurysm, which stops blood from entering the aneurysm and prevents rebleeding. Surgical clipping is done under general anesthesia.[1,3]
- *Endovascular occlusion*, which is less invasive than surgical clipping, involves placing coils into the aneurysm via a catheter. This procedure can be performed with general anesthesia or moderate sedation/analgesia.[1-3]

The International Subarachnoid Aneurysm Trial (ISAT) is the only randomized, multicenter trial that has evaluated surgical clipping and endovascular coiling in patients with aSAH. It yielded valuable information that can assist the neurosurgical and endovascular teams in determining which treatment is in the patient's best interest. Other trials that supplement the information gleaned from ISAT have been used to establish current treatment recommendations.[2,14]

Treatment decisions should involve collaboration between the neurosurgery team, the endovascular team, and the patient. All factors should be considered, including patient acuity, comorbidities, aneurysm morphology, and aneurysm location.

When an aneurysm is amenable to both types of treatment, endovascular occlusion is usually considered. Results of ISAT indicated overall morbidity and mortality one year after aSAH were lower for patients undergoing endovascular treatment. Most studies agree that outcomes are better when aneurysms of the posterior cerebral circulation are treated endovascularly. Outcomes may also

be better in patients who are in vasospasm at the time of treatment and those with a "poor" Hunt-Hess grade. (The guidelines don't specify a grade.)[2,14] Endovascular occlusion may also be preferred if the aneurysm is at the apex of the basilar artery or if the patient is over age 70.[2]

However, endovascular occlusion has some shortcomings. To prevent rebleeding, ruptured aneurysms should be completely occluded if possible. Patients in ISAT who underwent endovascular coiling had an increased incidence of delayed rebleeding, and just 58% were totally occluded.

Coils also can compact over time, allowing blood to reenter the aneurysm. Stents used in conjunction with coiling and biologically active coils have been applied in attempts to achieve more durable occlusions. Some research suggests that stent assisted-procedures may increase the risk of complications, such as hemorrhage and thromboembolism, but more study is needed. Research into the long-term occlusion rates of biologically active coils is also needed.[2,14]

Patients with middle cerebral artery (MCA) aneurysms have better outcomes with surgical clipping. Clipping may also be better for patients with intraparenchymal bleeding from the aSAH because the blood can be evacuated when the aneurysm is clipped. Early evacuation of intraparenchymal blood decreases morbidity and mortality.[2]

Research has been limited on anesthetic care during coiling and other aSAH-related endovascular treatments. The guidelines state that general anesthesia is useful for keeping the patient immobile and is applicable for certain patients undergoing endovascular procedures.[2,15]

BP and blood glucose levels should be well-controlled during definitive treatment. Hypotension and hyperglycemia during definitive treatment may both compound neurologic injury. Induced hypothermia to protect the brain against ischemic injury isn't well studied, but it may be appropriate for certain populations.[2,16]

Imaging should be done at the conclusion of any therapeutic intervention to confirm complete occlusion of the aneurysm. Noninvasive surveillance imaging studies are indicated for treated cerebral aneurysms. The time frame for this is at the discretion of the treatment team. Further treatment may be needed for aneurysms that continue to fill.[2]

Nursing care after treatment

RJ is treated with endovascular coiling. Before treatment, the nurse explains to RJ and her family (with the patient's permission) what to expect. The nurse reviews her lab test results and notifies the treating physician of any abnormalities. The nurse also notes her allergies and renal function; even patients undergoing surgical clipping may undergo angiography during surgery to confirm clip placement. Facility policy for standard preoperative evaluations should be followed.[1,3]

Postprocedure, the nurse monitors the patient's vital signs and neurologic status as prescribed, assessing for complications such as ischemia. The nurse also monitors her femoral access site and immobilizes her affected leg. Because a contrast medium was used, the nurse observes for signs of contrast-related complications such as contrast-induced nephrotoxicity. Craniotomy patients should also be observed for bleeding or infection at the incision.[1,3] Any signs or symptoms of complications must be reported immediately.

It's complicated, part II: After treatment

A standard set of delayed complications face aSAH patients. Delayed complications contribute to the high morbidity and mortality associated with aSAH.

Late seizures occur in 3% to 7% of patients. Long-term therapy with AEDs is a recommendation only when the patient is at risk for late seizures; for example, because of previous seizures or refractory hypertension.[2,3]

Vasospasm is cerebral arterial constriction that can lead to ischemia and infarction. Although it can occur any time after rupture, vasospasm peaks at 7 to 10 days post-aSAH and doesn't persist beyond 21 days.[2]

Signs and symptoms of vasospasm are related to cerebral ischemia and infarction. New focal neurologic deficits may develop and existing neurologic deficits may worsen. Altered LOC, particularly LOC that alternates in intensity, is another hallmark of vasospasm.[1-3]

Transcranial Doppler (TCD) ultrasound can be used to assess blood velocity through the cerebral arteries. TCD studies are considered an acceptable tool to periodically screen patients with aSAH for asymptomatic vasospasm.[2]

Delayed cerebral ischemia (DCI), a common complication of aSAH, is usually caused by vasospasm. Because DCI and ***cerebral infarction*** are the end results of vasospasm, multiple preventive therapies, screening and diagnostic studies, and treatments are indicated. Vasospasm, DCI, and cerebral infarction are the source of most morbidity and mortality in the weeks after aSAH.[2]

RJ's neurosurgeon started her on oral nimodipine as soon as her aSAH was diagnosed. Nimodipine is a calcium channel blocker that decreases neurologic morbidity related to vasospasm. All aSAH patients should be started on nimodipine as soon as possible and continue on it for 21 days. Nimodipine can cause hypotension, particularly in conjunction with other antihypertensives. Nimodipine metabolism can be affected by some AEDs, such as valproic acid, requiring dosage adjustments.[2,17]

Nimodipine is an oral drug. Take precautions to prevent inadvertent I.V. administration if the drug is withdrawn from the oral capsule for enteral administration. (See

"Nimodipine for Subarachnoid Hemorrhage: Patient Dies after I.V. Administration," *Medication Errors*, page 72.)

RJ should be kept euvolemic to help prevent vasospasm. Her hemoglobin and hematocrit should be maintained as well. Although the ideal hemoglobin for patients with aSAH is unknown, those with higher hemoglobin have better outcomes. For patients with anemia, transfusions may be prescribed to improve cerebral oxygenation. In patients for whom DCI poses a significant threat, transfusion is a justifiable corrective measure.[2,18-20]

Chronic hydrocephalus develops in 9% to 48% of aSAH patients. No prophylactic therapy has been found to alter the rate of chronic hydrocephalus. Weaning a patient from EVD over 24 hours doesn't appear to reduce the need for permanent surgical correction of chronic hydrocephalus.[2]

Patients with chronic hydrocephalus are treated with a permanent, internalized shunt. Postoperatively, monitor for standard surgical complications and observe the patient for signs of shunt failure, such as decreased LOC.[1-3]

The incidence of thrombosis, disseminated intravascular coagulation, disability, and death all increase in patients who develop ***heparin-induced thrombocytopenia (HIT)***. A hematologist should be consulted to aid in minimizing heparin use, because it's difficult to avoid in DSA and endovascular treatment. HIT and deep vein thrombosis (DVT) must be diagnosed early. No standard screening procedures have been recommended, but screening for DVT hasn't been shown to decrease the rate of pulmonary embolism.[2]

Managing vasospasm

On day 9 post-aSAH, RJ develops left upper extremity paresis. TCD studies indicate right MCA vasospasm. Recognizing that RJ has developed DCI as a result of vasospasm, her healthcare provider continues to keep her euvolemic, but prescribes an I.V. vasopressor to induce hypertension and improve CPP. Patients with aSAH who develop DCI typically improve with induced hypertension, but why is poorly understood. The guidelines don't specify a means for inducing hypertension, but hypervolemia should be avoided because research has failed to support its usefulness in DCI and vasospasm.[1,2,21]

RJ's signs and symptoms fail to improve with induced hypertension. Preliminary research studies have indicated that perfusion imaging with CT or MRI may be better at localizing ischemia than TCD studies.[2] RJ's perfusion CT confirms MCA vasospasm with corresponding ischemia.

RJ undergoes DSA, which also demonstrates vasospasm in the distal MCA branches. Because medical management has failed, the endovascular surgeon elects to use a balloon catheter to dilate the MCA. To open the distal arteries, a calcium channel blocker is infused intra-arterially. Although balloon angioplasty and vasodilator infusion haven't been studied in large, randomized trials, they've been shown to be effective and are accepted treatments.[2,22]

RJ's angiogram after her endovascular therapies shows her arteries have opened, and her signs and symptoms improve. She'll still require frequent neurologic assessments, nimodipine, fluid management, and TCD studies.

Endovascular therapies work well to open the spastic arteries, but vasospasm can return once the treatment ends.[2] RJ is closely monitored for medical complications, such as hyperglycemia, anemia, fever, sodium imbalance, decreased hemoglobin, and coagulopathies.

Other priorities

Fluid status and sodium level appear to impact the course of aSAH. Documenting intake and output closely and using central venous pressure monitoring are acceptable applications. Fluid status shouldn't be manipulated except in the presence of hypovolemia. Avoid hypotonic fluids in favor of colloids or crystalloids, as prescribed.[2]

Hyponatremia in aSAH may result from cerebral salt wasting or syndrome of inappropriate antidiuretic hormone secretion. Symptomatic vasospasm and asymptomatic vasospasm identified by TCD have been linked to hyponatremia. In this setting, crystalloids, colloids, 3% sodium chloride, and hypertonic sodium chloride solutions have all been shown to be beneficial. Medical management for hyponatremia can also include fludrocortisone and hydrocortisone.[2]

Hyperglycemia and hyperthermia should also be avoided. Normoglycemia in aSAH patients decreases the rate of poor outcomes. Correcting hyper- and hypoglycemia is a recommended strategy. Likewise, centrally triggered, noninfectious hyperthermia needs to be treated to prevent adverse effects on cognition and functionality. Any practical treatment is acceptable to employ in acute aSAH.[2]

The nurse monitors RJ's vital signs, physical assessment findings, and lab test results as prescribed. The nurse also monitors for adverse effects of any treatments. For example, hydrocortisone used to correct sodium imbalance can cause hyperglycemia, and blood transfusions or fluid administration can lead to fluid overload.[1]

Evaluation of cognition, behavior, and psychosocial issues is suggested once the patient is discharged.[2] Many patients experience lingering memory, mood, and cognitive problems after treatment.

With judicious application of the recommendations, RJ's outcome was optimized by the healthcare team. Discharged home with residual cognitive defects and a headache, she'll follow up with the endovascular surgeon and a neurologist, and undergo outpatient rehabilitation. ■

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INSTRUCTIONS

Aneurysmal subarachnoid hemorrhage: Follow the guidelines

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