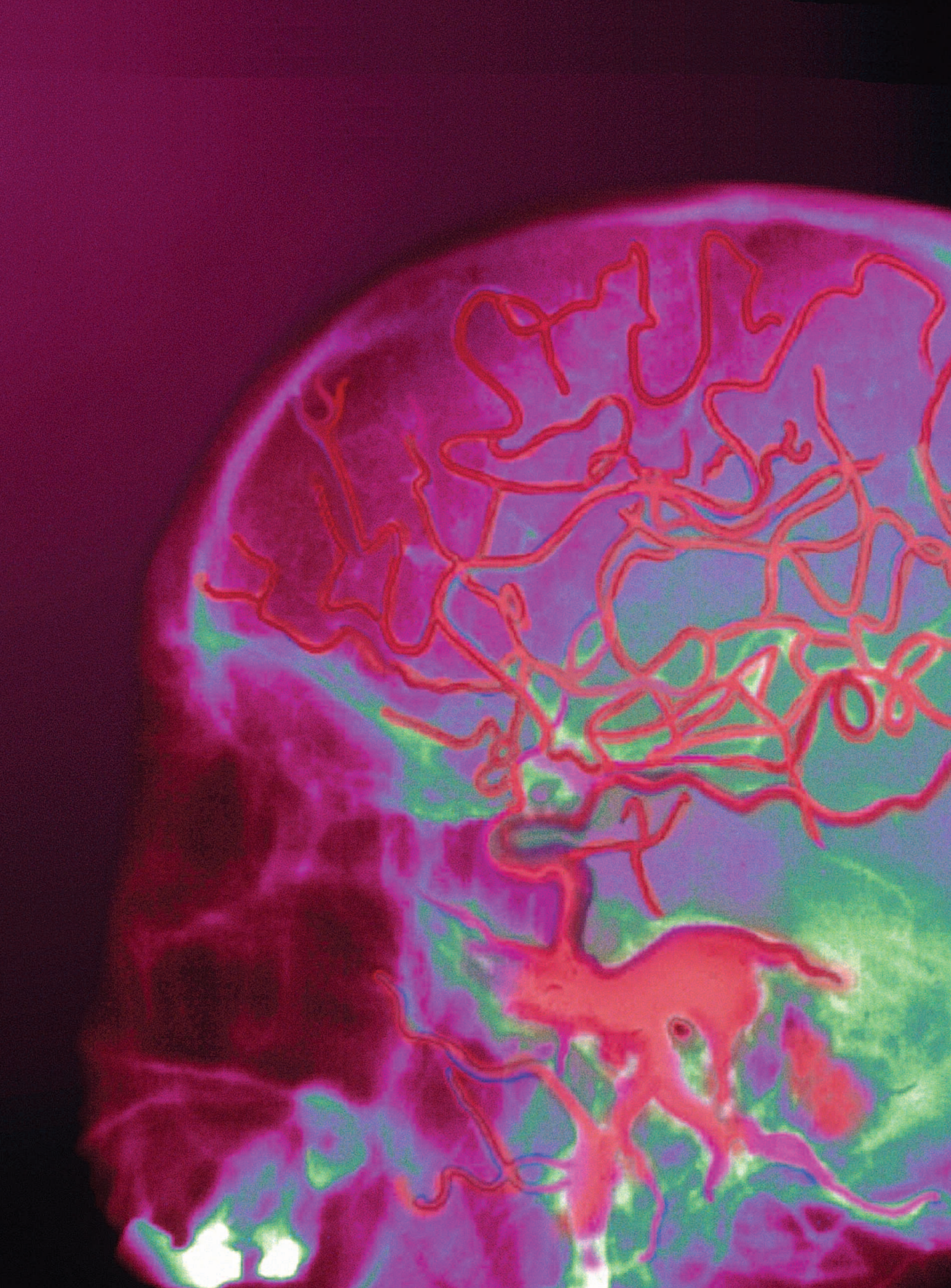
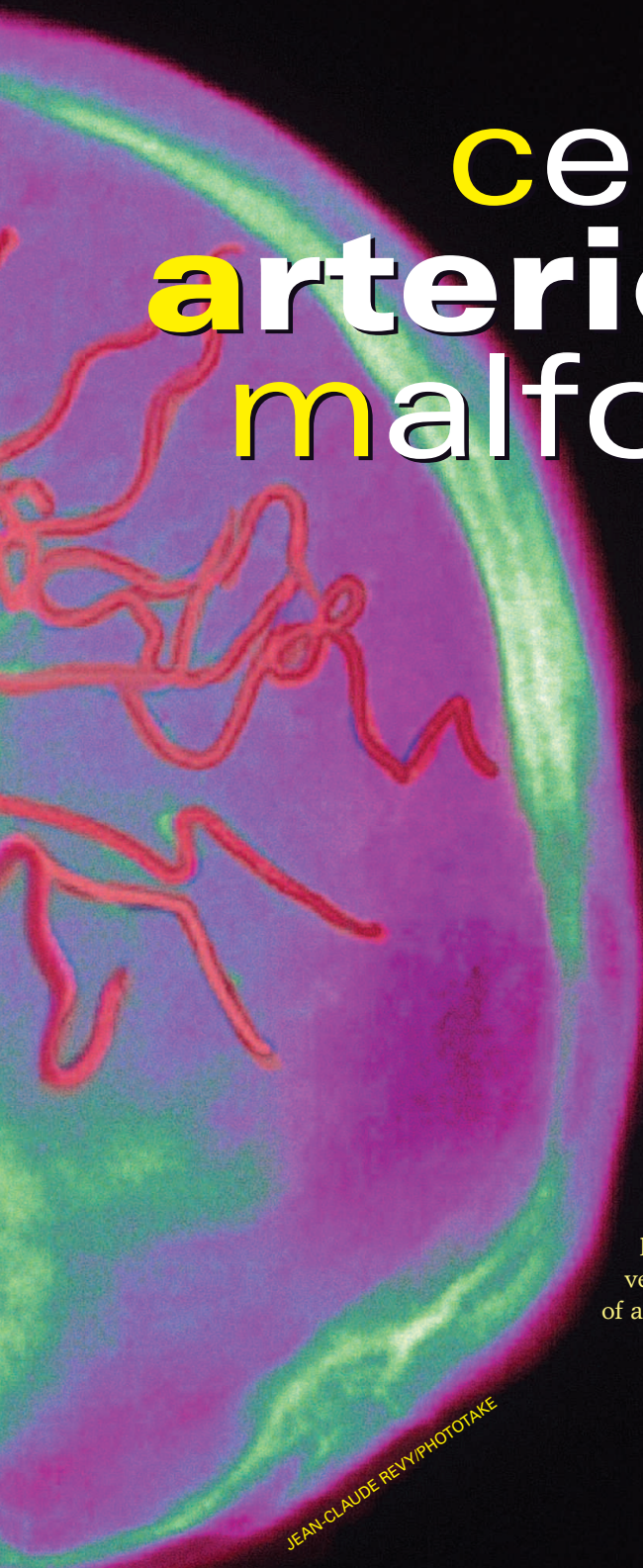


Teamwork integral to treating cerebral arteriovenous malformation



JEAN-CLAUDE REVY/PHOTOTAKE

A multidisciplinary team approach is necessary when the diagnosis is cAVM.

By **Vincent M. Vacca**, Jr., RN, CCRN, MSN, and **Stephen Violett**, RN, BSN

CE
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Cerebral arteriovenous malformation (cAVM) is a complex anatomical defect of the cerebral circulatory system that typically becomes symptomatic in young adults between 20 and 40 years of age. The defect is believed to occur during embryonic or fetal development or soon after birth.[1]

A cAVM develops within the cerebral circulatory system due to the lack of an intervening capillary bed that is normally present between arterioles and venules. The intervening capillary bed functions to modulate pressure and flow of blood between the higher pressure arterial side and the lower pressure venous side. The lack of a capillary bed causes formation of a complex vascular aggregate, or nidus, resulting in a

cAVM. Unregulated high pressure and velocity of arterialized blood flow entering the low pressure distensible cerebral venous system causes dilation of the veins, development of venous fistulae, and shunting of oxygenated blood.[2] Approximately 7% to 20% of individuals with cAVMs also have associated cerebral aneurysms.[1]

The actual prevalence of cAVMs in the United States is not known, as most cAVMs do not result in noticeable symptoms until the affected individual reaches adulthood. Existing estimates cite the approximate prevalence of cAVMs at 0.14% to 0.52% of the general population.[3] (See Case study.)

There are approximately 300,000 persons in the United States with a cAVM of the brain or spinal cord, of which 30,000 to 40,000 become symptomatic annually. The malformation is often found incidentally during treatment for an unrelated disorder or discovered at autopsy.[2,3]

Pathophysiology and symptoms

A history of generalized or migraine-like headaches affects approximately half of all individuals with a cAVM.[4] Migraine-like headaches and seizure may be the presenting symptoms of a cAVM, with or without hemorrhage, in 35% to 45% of cases. Following a seizure or chronic headaches, a cAVM may be found incidentally during the workup.[3]

Although general, nonspecific

Case study

It's a typical busy Saturday morning for 28-year-old healthy, married father of two young children, Dean Ash. After breakfast, Dean and his family head to a soccer match, where he is a coach for his childrens' team. During the drive, Dean begins to experience a migraine-like headache. He has had similar headaches over the past 6 months that he has managed using over-the-counter analgesics. Although he and his wife have discussed making an appointment, so far he has not seen a health care provider for evaluation.

While coaching from the sidelines, Dean's headache suddenly worsens; he becomes unsteady, collapses to the ground and has what appears to be a generalized seizure. Alarmed, his wife calls 911 from her cell phone.

The EMS paramedics arrive within 10 minutes and find Dean breathing spontaneously, but he is unresponsive to verbal stimuli. The paramedics stabilize Dean, including application of a rigid cervical collar, and position him on a long spine board. Both interventions are designed to protect his spinal cord. They quickly prepare Dean for transport to the nearest hospital for further evaluation and treatment.

En route to the hospital the paramedics perform a more thorough assessment of Dean, including a blood glucose level. Hypoglycemia can cause or worsen neurological disorders, and can be quickly corrected if present. His blood glucose is 100 mg/dL, within normal range.

Dean's pulse oximetery is 96% on room air, but as a precaution he is placed on supplemental oxygen at 2 L/minute via nasal cannula. Venous access is established and normal saline solution is administered at 100 mL/hour. During transport, Dean begins to awaken, but is confused.

When Dean arrives at the hospital emergency department (ED), he is fully awake, but does not remember what happened to him, except for the migraine-like headache, which he still complains of. The ED physician obtains Dean's medical history, which is unremarkable for any medical or surgical conditions, except for the recent headaches. Dean denies being a current smoker, he quit 5 years ago, and he admits to consuming alcohol, but only socially. He denies any illicit drug use, he takes no prescription medications, but does take over the counter analgesics for the headaches. The physician inquires about family history of cerebrovascular diseases, which Dean denies.

Because of the seizure and headache, Dean has a noncontrast computed tomography (CT) scan of his brain. He receives intravenous (I.V.) phenytoin (Dilantin), to prevent further seizures. He is also given a small dose of a short-acting I.V. opioid, fentanyl, to treat his headache. The results of his noncontrast CT scan reveal a small hemorrhage in the right hemisphere of Dean's brain. However, there is no evidence of trauma, a brain tumor, hydrocephalus, or cerebral edema at this time.

The ED physician consults with neurosurgery at another facility and based on Dean's age, clinical presentation, and the results of the noncontrast CT scan,

symptoms such as a migraine-like headache are common with cAVMs, the malformations can also cause a wide range of symptoms resulting from their size and location within the brain. Because of the differences in size and location, symptom presentation can vary greatly in individuals with cAVMs. The variation in symptoms can range from nonspecific to highly suggestive of serious neurological disease. This wide variety of symptoms includes: migraine-like headaches, weakness, loss of coordination, inability to concentrate, confusion, visual disturbances, difficulties understanding or using language, and seizures or a range of progressive and significant neurological deficits.[3]

In approximately two-thirds of adults diagnosed with cAVMs, a history of a subtle learning disability is often discovered. Findings as subtle as a learning disability suggest that the effects of cAVM can be subclinical and as such may result in the affected individual only seeking medical attention following a catastrophic event such as rupture with subsequent cerebral hemorrhage.[4]

A cAVM can produce neurological dysfunction from the following five significant mechanisms:

1. hemorrhage into the subarachnoid space, the intraventricular space, or into the brain parenchyma
2. seizure activity
3. chronic cerebral vascular "steal syndrome," in which oxygenated blood flow to otherwise

following a collaborative decision, he is transferred to their neuroscience intensive care unit (ICU). Because of the distance between facilities, the decision to transport Dean aeromedically is made. During Dean's flight, he remains awake, alert, breathing spontaneously, hemodynamically stable, and neurologically intact. He still has a mild headache, but does not experience any further seizure activity.

Following Dean's arrival at the medical center, he has a magnetic resonance imaging (MRI) study and a functional MRI, to identify the cause of his seizure and the source of the small hemorrhage and to determine the location and involvement of any eloquent areas of his brain.

The results of the MRI reveal what the neurosurgeon suspected; Dean has a cerebral arteriovenous malformation (cAVM). Fortunately for Dean, his cAVM is not located in or near any of the eloquent areas of his brain. However, having one hemorrhage or bleeding episode from a cAVM puts Dean at high risk for subsequent rebleeding, which can be fatal. Based on Dean's age, overall good health, and size and location of his cAVM, the decision among him, his family, and the neurosurgery team is to act aggressively to treat and control his cAVM.

After reviewing the MRI and functional MRI, the neurosurgeon decides that it is best to perform a cerebral angiography to assess the blood flow dynamics and grade of Dean's cAVM. Based on the accepted Spetzler-Martin cAVM grading system, Dean has a grade 2 cAVM.

The interventional neuroradiologist performs endovascular embolization prior to surgical resection to help stabilize fragile blood vessels and reduce intraoperative risk from additional hemorrhage that could occur during surgical resection.

The neurosurgeon and interventional neuroradiologist also discuss stereotactic radiosurgery, but this option is ruled out based on his Spetzler-Martin grade and the location of the cAVM.

Following the successful endovascular embolization, Dean returns to the neuroscience ICU to recover. His craniotomy and surgical resection of the cAVM are planned and scheduled to occur 2 days later.

Dean's surgical resection is uneventful and successful. He recovers in the neuroscience ICU for 2 days, and then is well enough to transfer to the intermediate neurosurgery area, where he continues his recovery for three more days. Five days from admission, Dean is well enough to be discharged home to complete his recovery. He will continue to take an oral antiepileptic drug as long as he does not have another seizure and until he is safely healed and no longer at risk for hemorrhage. Then he will be weaned off of the medication.

Dean has returned to his previous lifestyle with his family and enjoys each day knowing that he was lucky to have survived the initial hemorrhage of his cAVM.

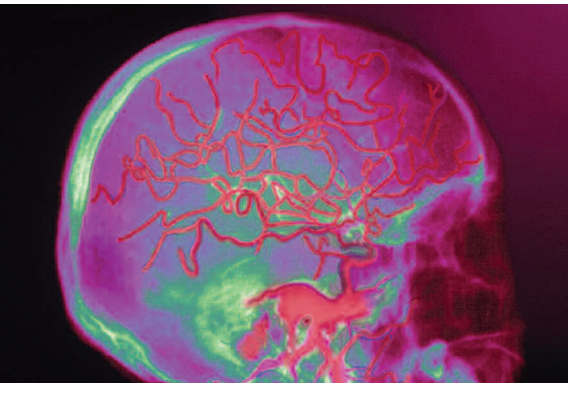
healthy brain tissue is shunted through fistulae, causing hypoperfusion to those areas of healthy brain tissue

4. compression and distortion of brain tissue, causing a "mass effect" due to an enlarging cAVM

5. venous hypertension, leading to excess blood volume within the cranial vault, causing increased intracranial pressure and neurological symptoms.[4]

Rupture and hemorrhage

Neurological impairment following hemorrhage from a cAVM depends on the location and amount of bleeding. cAVMs can range from small to large in size and can occur in any area of the brain. It is possible for individuals to have multiple small hemorrhages from cAVMs before significant neurological symptoms result.[1]



The diagnostic workup of cAVMs is designed and focused to detect, locate, and grade them.

The presence of cAVMs can go undetected for years and often the initial presentation of cAVMs in 40% to 70% of individuals between the ages of 10 and 55 years is from intracranial hemorrhage. The annual bleeding risk from untreated cAVMs is 2% to 4%, but incidence increases to 18% in the year following initial hemorrhage, regardless of lesion size. Rates of morbidity and mortality increase with each episode of rebleeding. Estimates suggest a 50% incidence of new neurological deficit and 10% to 20% incidence of death with each episode of rebleeding from cAVM.[1,4,5]

Prompt recognition and intervention of cAVM is essential to maximize chances for a beneficial outcome. Following rupture and bleeding from a cAVM, emergent surgical hematoma evacuation with effective intracranial pressure control can lead to excellent outcomes if done in time.[6]

Neurological deficits resulting from cAVM hemorrhage tend to be less severe compared with deficits from other causes of cerebral hemorrhage, such as aneurysms causing subarachnoid hemorrhage. There is, however, an approximate 25% risk of significant disability and a 10% risk of death associated with these deficits.[1]

Diagnostic workup

The diagnostic workup of cAVMs is designed and focused to detect, locate, and grade them. The investigative workup begins with a degree of suspicion based on the individual's medical history and presenting signs and symptoms. A noncontrast computed tomography (CT) scan is useful to identify evidence of a cerebral hemorrhage and is often the first imaging technique utilized. If the CT scan is positive for hemorrhage in an otherwise low risk young individual, this unexpected positive finding then focuses the clinicians' attention to the possibility of a cAVM. However, CT scanning is limited in that it can identify only large cAVMs. Magnetic resonance imaging (MRI) is also an essential imaging tool used to diagnose cAVM, as the malformation may be small and deep within subcortical white matter and visible only with MRI.

A variation of MRI known as a functional MRI is useful to localize or map brain function, which can assist in development of the treatment plan. The importance of functional MRI is to identify eloquent brain regions (refers to areas of the brain that have readily identifiable neurological function), including those responsible for language, memory, vision, and motor or sensory function, in or near the cAVM. Damage to these areas during surgery may be unavoidable and can result in significant and permanent neurological deficits. This vital functional information must be known in order to develop the most beneficial treatment plan to avoid or limit damage to these identified eloquent areas.

Once a cAVM is diagnosed, aided by CT, MRI, and functional MRI, a cerebral angiogram will be performed. The benefit of cerebral angiography is to directly and accurately assess the morphology and hemodynamics of the cAVM. Analysis and evaluation of cAVM characteristics are

required and essential to plan appropriate treatment. Important anatomical features identifiable and measurable with cerebral angiograpy include: size of the cAVM, identification of feeder arteries, presence of arterial or venous aneurysms, and venous drainage patterns. Anatomical information is essential and must be known to plan appropriate intervention.[6]

Treatment considerations

Calculating the risk of hemorrhage resulting from the cAVM is carefully done to develop an individualized treatment plan. In some cases, an older adult or someone without identifiable hemorrhagic risk factors might be offered a conservative, noninterventional medical management plan as the best option. For those individuals identified at low risk for hemorrhage, many nonsurgical, noninterventional options exist to manage cAVMs including: seizure prophylaxis, blood pressure control, blood sugar control, weight loss if applicable, cessation of smoking, diet management, exercise, and stress reduction with appropriate analgesia for headaches.

Combination therapy

However, for younger individuals at risk for hemorrhage from a cAVM there are several possibly curative treatment options to consider. The options include endovascular embolization, surgical resection, or stereotactic radiosurgery (SRS). These interventions may be used alone or are often used in combination. Specific treatment decisions depend on the individual's age, condition, and unique cAVM features.[7]

Spetzler-Martin AVM grading scale

Size of cAVM	Eloquence of adjacent brain	Deep venous drainage
Small (0 to 3 cm)=1	Noneloquent=0	Not present=0
Medium (3.1 to 6 cm)=2	Eloquent=1	Present=1
Large (greater than 6 cm)=3		

Source: Spetzler RF, Martin NA. A proposed grading system for arteriovenous malformations. *J Neurosurg.* 1986;65(4):476-483.

Grading the cAVM

Results from the diagnostic evaluation of cAVMs including CT scan, MRI, functional MRI, and cerebral angiography are used in conjunction with the cAVM scoring system known as the Spetzler-Martin AVM grading scale. (See Spetzler-Martin AVM grading scale.) Using the Spetzler-Martin scale, the cAVM is graded based on the location, size, and adequacy of venous drainage. The scale ranges from 1 to 5, with 1 being the lowest surgical risk and 5 the highest. The Spetzler-Martin scale is used to determine both the degree of surgical difficulty in resecting the cAVM, and to predict the likelihood of a successful outcome following surgical intervention.[2,4,6-8]

Treatment plan

The treatment plan for cAVM is developed from careful and precise evaluation of many considerations including, the age of the individual, existing comorbidities including any current neurological deficits, the results from all the imaging studies, the cerebral angiogram, and the Spetzler-Martin grade.

Is surgery an option?

Neurosurgery involving a craniotomy with resection of the cAVM is generally recommended for a Spetzler-Martin grade of 1, 2, or 3 and occasionally grade 4. Due to the substantial risk, surgery is not recommended for grade 5.[1]

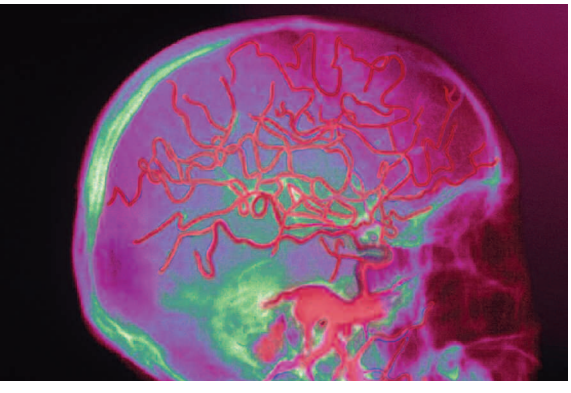
A nonsurgical treatment option for a cAVM is endovascular embolization, using glue particle fibers, microcoils, or microballoons delivered via a special catheter. Endovascular embolization is designed to thrombose and possibly obliterate the pathologic blood vessels within the cAVM. Embolization agents can be liquid or solid, absorbable or nonabsorbable, or cytotoxic. They are designed to induce thrombosis and obliterate the abnormal blood vessels within the nidus of the cAVM. Embolization material is delivered via a catheter introduced into a femoral artery and is performed in the angiography suite frequently with the individual endotracheally intubated on mechanical ventilation and under general anesthesia. Possible complications from endovascular embolization include incomplete occlusion of pathologic vessels, microperforation of vessels leading to hemorrhage, gluing of the catheter to the vessel wall requiring dissection to remove it, catheter failure, or occlusion of necessary perfusing branches.

Maintenance of venous outflow is an essential goal to successfully stabilize and treat a cAVM following endovascular embolization. Promotion and preservation of venous outflow facilitates blood flow from the arterial to venous drainage vessels. Adequate flow protects delicate blood vessels from dilation, and increased pressure that could cause them to rupture and hemorrhage.[6]

Treatment by SRS involves the use of radiation therapy via proton beam, linear accelerator, or gamma knife. The delivered radiation is designed to induce intimal injury or a radiation-induced arteritis, which causes vessel thrombosis and obliteration of the cAVM. SRS, along with endovascular embolization and surgery, is considered to be one of the curative treatment options in select cases. However, the desired thrombotic effect from SRS can be delayed by more than 2 years, during which time the risk for hemorrhage continues to exist. The advantages of SRS are that it can access all areas of brain tissue and it is noninvasive. Possible complications from SRS include damage of healthy brain tissue from radiation exposure resulting in brain tissue edema, and also the possibility of radiation-induced necrosis of healthy brain tissue. Other possible complications from SRS include failure to obliterate the lesion, and postprocedure hemorrhage. Therefore, SRS is most effective in small to medium sized cAVMs (less than 3 cm in diameter), but is also used in high-risk, nonsurgical treatment of cAVMs found deep within brain tissue.[2,5]

For Spetzler-Martin grades 1 and 2, the risk from surgical intervention of a cAVM outweighs the risk for hemorrhage making these grades of cAVM more amenable to endovascular embolization or SRS.[1]

The advantage of surgical resection via craniotomy is an immediate and permanent cure. There are disadvantages associated with surgery, including the possibility of hemorrhage and or brain tissue damage resulting from the sudden alteration in cerebral blood flow patterns causing ischemia to otherwise healthy brain tissue. Surgical resection is possible with many cAVMs, but cannot be performed on all them. For Spetzler-Martin grade 2 cAVMs in an eloquent region of the brain, SRS is used rather than the more risky surgical option.[1]



When treatment options are used in combination, one intervention can enable another to be more successful than if used alone.

Multimodality treatments

When treatment options are used in combination, one intervention can enable another to be more successful than if used alone. For example, endovascular embolization can reduce blood flow to the cAVM, and because of the reduction in high-pressure blood flow to the cAVM, endovascular embolization is often used first and in combination with either surgery or stereotactic SRS. Endovascular embolization as a presurgical adjunct can decrease the size of the cAVM, which can reduce risk of intraoperative hemorrhage and can also reduce the technical difficulty of surgical resection. This therapeutic combination approach is especially useful in the higher grade, Spetzler-Martin 3 or 4 cAVMs.

Even when used in combination therapy, endovascular embolization does have limitations including that it is an invasive procedure and can cause reduced perfusion to brain tissue, resulting in an ischemic stroke. Significant blood flow alterations following endovascular embolization can cause rupture of the cAVM resulting in hemorrhagic stroke. Endovascular embolization is generally not a technique used to achieve complete and permanent obliteration of the cAVM, but is used in combination with SRS or surgery. Recanalization of the pathologic vessels can occur following endovascular embolization, making this procedure more useful as an adjunct to SRS or surgery. Endovascular embolization as a primary therapeutic intervention can be used as either a contributory part of a multimodal treatment plan or as

a palliative intervention.[6]

Combinations of the three treatment options are often used to achieve the desired outcome of reducing or eliminating the risk of devastating permanent neurological sequelae or death from rupture and hemorrhage of a cAVM. There are advantages and disadvantages to each of the three therapeutic approaches to cAVMs. The treatment plan is dependent on the individual presentation and characteristics of the cAVM. In many cases a combination of treatment options may be chosen.[7]

Take a team approach

A multidisciplinary team approach is necessary when the diagnosis is cAVM and will include a neurosurgeon, interventional neuroradiologists, stereotactic radiation specialists, and expert neuroscience critical care nurses.

The desired treatment goal is complete eradication of the cAVM lesion. The cAVM treatment approach is made collaboratively by taking into account the affected individual's physical and psychological symptom profile, current neurological deficits, risk for future neurological deficits, current quality of life, and Spetzler-Martin grade. The treatment plan is based on several factors including degree of immediate risk from the lesion, the degree of risk from treatment intervention, and the possibility of permanent cure. There are many considerations when the diagnosis is cAVM.

For more information on cAVM, visit the National Institute of Neurological Disorders and Stroke (NINDS) Web site at http://www.ninds.nih.gov/disorders/disorder_ index.htm. ❖

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The authors have disclosed that they have no significant relationship with or financial interest in any commercial companies that pertain to this educational activity.

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Teamwork integral to treating cerebral arteriovenous malformation

GENERAL PURPOSE: To provide the registered professional nurse with an overview of cerebral arteriovenous malformations (cAVMs) and their treatment. **LEARNING OBJECTIVES:** After reading this article and taking this test, you should be able to: 1. Describe the pathophysiology and symptoms of cAVM. 2. Explain different treatment options for cAVM.

1. What is the typical age that cAVMs become symptomatic?
a. soon after birth
b. between 20 and 40 years old
c. between 50 and 70 years old
d. after age 70

2. What is a nidus?
a. a complex vascular aggregate within the cerebrum
b. a venous fistula
c. an aneurysm
d. a venous shunt

3. Which statement regarding cAVM is correct?
a. Symptoms usually begin in childhood.
b. The approximate prevalence is 1% of the general population.
c. The malformations are often found incidentally.
d. It's rare to recover from an initial rupture and bleed.

4. Which symptom affects approximately half of all individuals with cAVM?
a. migraine-like headaches
b. weakness
c. seizures
d. inability to concentrate

5. What symptom do two-thirds of adult patients with cAVMs have in common?
a. seizures
b. headaches
c. visual disturbances
d. learning disabilities

6. All of the following can cause neurological dysfunction from a cAVM *except*
a. hemorrhage into the brain parenchyma.
b. seizure activity.
c. blood shunting through a fistula causing brain hyperperfusion.
d. venous hypertension.

7. Each statement about neurological impairment following hemorrhage associated with cAVM is true *except*
a. symptoms are dependent on the size of the bleed.
b. symptoms are dependent on the location of the cAVM.
c. many small bleeds may occur before significant neurological symptoms appear.
d. there is an approximate 45% risk of significant disability.

8. What is the annual bleeding risk after the initial hemorrhage from a cAVM?
a. It is dependent on the lesion size.
b. It is dependent on the lesion location.
c. The risk in untreated cAVM is 2% to 4% during the first year.
d. The bleeding risk decreases annually after the first year.

9. What intervention is absolutely necessary after rupture and bleeding from cAVMs?
a. watchful waiting
b. emergent hematoma evacuation
c. correction of hyperglycemia
d. functional magnetic resonance imaging

10. Which is a noninterventional option to manage low hemorrhage-risk cAVM?
a. diet management
b. insomnia treatment
c. sleep apnea management
d. COPD treatment

11. Which patient is a good candidate for a curative treatment option for cAVM?
a. a pregnant female
b. an older adult
c. a 20-year-old without an identifiable hemorrhagic risk
d. a 40-year-old who is at risk for bleeding from a cAVM

12. The Spetzler-Martin AVM grading scale is used to
a. predict the likelihood of a positive outcome after surgery.
b. diagnose subcortical white matter malformations.
c. map brain function and perfusion.
d. quantify the degree of neurological deficit.

13. Which statement about endovascular embolization is true?
a. A special catheter is inserted into the carotid artery.
b. It permanently resects the pathologic blood vessels in the cAVM.
c. One potential complication is gluing the catheter to the vessel wall.
d. It is performed in the operating suite with moderate sedation/analgesia.

14. Treatment of cAVMs by stereotactic radiosurgery (SRS)
a. will immediately eliminate any risk of hemorrhage.
b. is rarely curative.
c. may take 2 years to achieve full effect.
d. is most suitable for larger (grade 3 or 4) cAVMs.

15. One advantage of craniotomy for cAVM treatment is that it
a. offers a permanent cure.
b. can be used for all grades of cAVMs.
c. has a low risk of hemorrhage.
d. eliminates any need for multimodal therapy.

16. Which statement *best* summarizes the general concepts of this article?
a. There are very few considerations when treating the patient for cAVM.
b. The neurosurgeon is the most valuable team member.
c. The desired treatment goal is to stabilize the patient.
d. A team approach is necessary to achieve complete eradication of cAVMs.

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❑ Charge my ❑ Mastercard ❑ Visa ❑ American Express

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*In accordance with the Iowa Board of Nursing administrative rules governing grievances, a copy of your evaluation of the CE offering may be submitted directly to the Iowa Board of Nursing.