Treatment of Brain Aneurysms
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AMY ROTHMAN SCHONFELD, PhD
MICHAEL A McMULLEN, R.T.(R)

After completing this article, readers should be able to:
■ Recognize the causes of and risk factors for cerebral aneurysms.
■ Understand the etiological and structural differences between saccular and nonsaccular aneurysms.
■ Choose appropriate diagnostic imaging procedures for the detection and analysis of cerebral aneurysms and subarachnoid hemorrhage.
■ Discuss factors that increase the risk of cerebral aneurysm rupture.
■ Describe the signs, symptoms and laboratory findings associated with cerebral aneurysms before and after rupture.
■ Explain the advantages and disadvantages of surgical and endovascular treatment for unruptured and ruptured cerebral aneurysms.
■ Describe the indications, devices and methods available for coiling cerebral aneurysms.
■ Recognize the essential and varied role of the radiologic science professional in the diagnosis and endovascular treatment of cerebral aneurysms.

The National Institute of Neurological Disorders and Stroke (NINDS) defines a cerebral aneurysm as “a dilatation, bulging, or ballooning out of part of the wall of a vein or artery in the brain.” As the aneurysm enlarges, risk of rupture increases. Once an aneurysm ruptures, blood is pumped out of the vessel at high pressure into the subarachnoid space, the ventricles, surrounding brain substance or subdural space. The amount of resulting brain damage generally is proportional to the amount of blood released. Twelve percent of patients die before reaching the hospital, while 40% of patients die within 1 month of rupture. About 33% of the patients are left with major neurological deficits.

Left untreated, aneurysms rebleed in 20% of patients within 2 weeks of the initial hemorrhage, in 30% of patients by 1 month and in 40% by 6 months, adding to morbidity and mortality. Despite improvements in diagnosis, management and follow-up of aneurysms, rupture can have catastrophic consequences for the patient. Accurate diagnosis and prompt intervention, whether through surgical or radiological means, can have enormously beneficial outcomes for the patient.

Background

Epidemiology

Approximately 6 million people in the United States are believed to harbor intracranial aneurysms. The results of adult autopsy series estimate the frequency of aneurysms in the population to be 1% to 6%, while angiographic series estimates vary between 0.65% and 7%. Aneurysms are reported as incidental findings in about 0.5% to 1% of patients undergoing cerebral angiography for other reasons.
Aneurysms can occur at any age, although they are more common in adults. The incidence increases after 50 years of age, with greater frequency among patients in their 60s or 70s. In addition, women are 3 times more likely than men to develop intracranial aneurysms. Most cerebral aneurysms are asymptomatic, and 50% to 80% of aneurysms will not rupture during a person’s lifetime. Rupture rates generally depend on the size and location of the aneurysm. Juvela et al reported an annual rupture incidence rate of 1.3% in a group of 142 patients with 181 unruptured aneurysms during an average follow-up period of 20 years. The Society of NeuroInterventional Surgery, formerly known as the American Society of Interventional and Therapeutic Neuroradiology, estimated that approximately 0.2% to 3% of people with brain aneurysms suffer bleeding each year and more than 30,000 people each year will develop subarachnoid hemorrhage (SAH) caused by a ruptured aneurysm.

**Causes and Risk Factors**

Many factors can contribute to the development of a cerebral aneurysm. Aneurysms tend to appear in blood vessels in which the middle muscular layers (tunica media) are thinner than normal. This weakens the walls of cerebral blood vessels and makes them susceptible to hemodynamic stresses and acquired degenerative changes.

As a general rule, processes that exacerbate hemodynamic stress or undermine the structure of the vascular wall can predispose patients to developing cerebral aneurysms. Hemodynamic effects explain why uncontrolled hypertension, pregnancy-related hypertension, heavy lifting, straining, emotional outburst and drugs such as amphetamines, ephedrine and cocaine are linked to aneurysm formation and bleeding.

Factors that undermine vascular cell walls include diseases, patient characteristics (eg, gender, age, genetic predisposition or other illnesses) and exogenous risk factors (eg, trauma or smoking). The Box lists some of the inherited and acquired connective tissue disorders associated with intracranial aneurysms.

Genetic determinants most likely play a role in the development of cerebral aneurysms. For example, patients with autosomal dominant polycystic kidney disease (PKD) are 4 to 5 times more likely to have an intracranial aneurysm than the general population. These patients have a mutation in the PKD1 gene that disrupts the interaction between arterial smooth-muscle cells and adjacent elastic cells, weakening the vessel wall. About 20% of patients with saccular cerebral aneurysms have a family history of aneurysm or subarachnoid hemorrhage. Although genetic studies are underway, no single gene has been found to be responsible for cerebral aneurysm or rupture.

It is likely that both genetic susceptibility and environmental factors interact to increase risk of aneurysms. Risk factors for aneurysm development and growth include cigarette smoking and being a woman. Smoking causes a decrease in the effectiveness of alpha-1 antitrypsin, which affects the elastase in artery walls. Thus, smoking-related risk increases with the number of cigarettes smoked each day, not the duration of smoking or age at which smoking began. Age and heavy consumption of alcohol are other known risk factors. Cigarette smoking, aneurysm characteristics (eg, size, shape and location) and age also are known risk factors...