Aneurysmal Subarachnoid Hemorrhage: A Potentially Lethal Neurological Disease

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A subarachnoid hemorrhage (SAH) is characterized by bleeding that occurs between the arachnoid and pia mater meningeal layers of the brain. This type of hemorrhage accounts for about 5% of all strokes, and the majority occur as the result of a ruptured aneurysm. The incidence of the disease varies across gender, race, and geographical locations in the world, and it carries significant morbidity and mortality. A patient with an aneurysmal SAH can present to the emergency department with an array of neurological symptoms, and thus it is imperative for ED nurses to be able to quickly recognize the signs and symptoms and initiate stabilization and treatment. This article will review the epidemiology and risk factors of aneurysmal SAH, followed by an overview of pertinent neurological anatomy and the pathophysiology of this disease. A discussion of the interventions an ED nurse may expect to initiate prior to fixation of the aneurysm will be addressed as well.

Epidemiology and Risk Factors

The incidence of aneurysmal SAH varies across race, gender, and geographical regions of the world. In the United States, for example, the occurrence ranges from 9.7 to 14.5 of every 100,000 adults.[1] The average age of occurrence is 55 years, and African Americans are affected more than white persons.[1] A majority of studies on this disease indicate that women are affected more than men. Wide variation exists in the occurrence of aneurysmal SAH across continents, from "2.0 cases per 100,000 population in China to 22.5 cases per 100,000 in Finland."[1] Aneurysmal SAH results in significant mortality; approximately 12% to 15% of persons with an aneurysmal SAH die immediately.[2] For patients who are admitted to the hospital, the mortality ranges from 25% to 28%, and it is worse in those who present with poor neurological function.[2] Ruptured aneurysms may rebleed and are associated with a mortality rate between 50% and 85%.[3] Many patients who recover from an aneurysmal SAH are left with significant neurological deficits.[4]

There are a number of risk factors for the development of an aneurysmal SAH, some of which can be controlled by changes in behavior and lifestyle. These modifiable risks include the use of alcohol and smoking, along with use of cocaine. Gender is a nonmodifiable risk factor; females carry a greater risk for the disease. Furthermore, the incidence of aneurysmal SAH is higher in persons with a familial history of aneurysm, particularly in a first-degree relative such as a parent.[1] In patients with a known aneurysm, the size and anatomic location of the aneurysm may affect the risk of rupture, but the evidence remains unclear.[1]

Cerebral Arterial Circulation

The brain receives its arterial blood supply from a complex network of arteries. Different regions of the brain are supplied with blood by specific arteries. The internal carotid arteries supply blood to the anterior regions of the brain, while the posterior regions are supplied by the vertebral arteries.[4] Branches from these 2 major arteries anastomose to form the circle of Willis, an important anatomic feature of the arterial circulation located at the base of the brainstem. The communication of these major arteries allows for the continued blood flow to the brain should a disruption in one of them occur.[4]

The internal carotid arteries distribute most of their blood supply to the anterior and middle cerebral arteries. From the anterior cerebral arteries, blood reaches the frontal and parietal lobes, as well as parts of major structures such as the thalamus and corpus callosum.[4] Blood from the middle cerebral arteries flows to the lateral basal ganglia, as well as regions associated with language, auditory function, and delicate control of the hand and face.[4]

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The vertebral arteries enter the skull through the foramen magnum and then join to form the basilar artery. Branches from these arteries provide blood to major regions of the brain such as the cerebellum, pons, and medulla, as well as part of the diencephalon and the midbrain.[4] The basilar artery divides into the posterior cerebral arteries, through which blood reaches the occipital lobe, thalamus, and inferior aspects of the temporal lobes.[4]

Pathophysiology of Aneurysm Development

An aneurysm is characterized as a bulge or protuberance that occurs at a weakened spot in a vessel wall.[5] In the cerebral arterial circulation, aneurysms are most commonly found in the circle of Willis at the bifurcations of 2 or more arteries.[6] In spproximately 20% of patients, more than one aneurysm will develop at different locations. The risk of rupturing increases with the size of the aneurysm; those that are larger than 7 mm and located at the origin of the posterior communicating artery and on the basilar artery carry the greatest threat.[6]

As the aneurysm develops, it usually forms into a neck with a dome. The internal lining of the arterial wall vanishes at the base of the neck, the middle layer thins, and smooth muscle is replaced by connective tissue. Ruptures primarily occur within the dome of the aneurysm, where the wall thins. The rapid release of the blood into the brain causes a rise in intracranial pressure (ICP).[6]

Monroe-Kellie Doctrine

The Monroe-Kellie Doctrine provides a foundation for managing the complications of aneurysmal SAH. The doctrine states that the intracranial compartment consists of brain matter, blood, and cerebrospinal fluid (CSF).[7] These 3 components are maintained in a balanced equilibrium under normal physiological conditions. If there is an increase in the volume of one component such as CSF, then the volume of the other 2 components must decrease to maintain ICP within the normal range. If compensatory mechanisms fail, then a slight increase in one of the components can cause a large increase in ICP.

The normal ICP in adults is 5 to 15 mm Hg.[7] Persistent elevations in ICP can lead to a decrease in cerebral perfusion, in turn causing ischemia, herniation, or even death of the patient. The cerebral perfusion pressure (CPP) equals the mean arterial pressure (MAP) minus the ICP.[7] This relationship means that an increase in the ICP or a decrease in the MAP decreases the CPP. The brain maintains CPP between 50 to 150 mm Hg by way of autoregulation. This autoregulation can be impaired by an SAH, because blood products can occlude the reabsorption of CSF in the ventricular system, which can lead to a rise in the ICP. As a result, CPP can fall below 50 mm Hg and cerebral blood flow will passively follow changes in the CPP.[7]

Clinical Manifestations of Aneurysm Rupture

A cerebral aneurysm can rupture during periods of physical activity or stress; however, the majority of ruptures occur when a person is at rest.[1] The hallmark manifestation of a ruptured aneurysm in a conscious patient is a report of "the worst headache of my life."[1] It will be described as a thunderclap, occurring suddenly with maximum intensity.[1] In addition to this abrupt headache, a patient may have other signs such as photophobia, nausea, and emesis, as well as an array of neurological deficits and loss of consciousness.[1] Seizure activity may be present as well, occurring in approximately 20% of patients. Patients with a history of migraine headaches may report a thunderclap-type of pain that can mimic a subarachnoid hemorrhage and thus should undergo a definitive workup to rule out the presence of a bleed.[7] Although some patients may present with the classic headache, it is important to know that the description of the headache may be inconsistent, which can result in either an inaccurate or delayed diagnosis.[1]

Diagnosis and Treatment of Aneurysmal SAH

The American Heart Association and the American Stroke Association published updated evidence-based guidelines for the diagnosis and treatment of aneurysmal SAH in 2012. If the hospital at which a patient initially presents sees a small volume of patients with this diagnosis in a year (ie, fewer than 10), then the patient should be transferred to a high-volume facility (ie, one that sees more than 35 such patients in a year) with "experienced cerebrovascular surgeons, endovascular specialists, and multi-disciplinary neuro-intensive care services."[1] These guidelines further recommend that patients with signs and symptoms of a ruptured cerebral aneurysm undergo a noncontrast computed tomographic (CT) scan of the head, which is considered to be the foundation of diagnosis.[1] The sensitivity of a CT scan in detecting an SAH, if performed within 3 days of rupture, is as high as 100%, but the sensitivity decreases significantly thereafter. If a CT scan does not show a bleed when there is a strong clinical suspicion for a ruptured aneurysm, then a lumbar puncture is indicated.[6] The CT scan must be performed before a