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**Aneurysmal Subarachnoid Hemorrhage**  
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**Introduction**

"Aneurysmal subarachnoid hemorrhage (SAH) is the most devastating type of stroke" (Yu, et al., 2018). It affects up to 14/100,000 adults per year worldwide with up to 14/100,000 of those adults being from the United States (Danse & Mohet, 2017).

- 80% of spontaneous, non-traumatic SAH results from aneurysm rupture
- 20% of SAH patients do not survive
- 60-80% of those who do survive are at risk for lifelong neurological deficits
- Patients with a SAH diagnosis are at high risk for many complications during their treatment (Danse & Mohet, 2017).

For many nurses, the neurological system serves as a weakness in their knowledgebase. It is a specialized field needing continued education. As a registered nurse working in a comprehensive stroke center, it is easy to identify the importance of education related to treating neurological patients and the close monitoring involved in their treatment. Continued education is a key part in reducing morbidity and mortality in these patients. By giving both nurses and providers a better background in treatment options, they can feel more comfortable and confident in the care they are providing to their neurological patients.

**Pathophysiology**

When an aneurysm forms at a vessel bifurcation, it weakens the walls of the vessel. This in combination with high intravascular pressure can cause a rupture of the aneurysm, which displaces blood into the subarachnoid space in the brain (McCance & Huether, 2018).

**Intracranial Aneurysms**
- Weak bulging areas of an arterial vessel wall
- Aneurysm development involves hypertension causing wall shear stress at sites of inflammation and remodeling
- Aneurysm development leads to a thin endothelial layer, absence of fragmented internal elastic lamina, and muscularis layer of the media ending at the aneurysm
- Usually located within the bifurcations of the circle of Willis
- Due to a combination of genetic, congenital, and acquired defects
- Size: 2mm – 3cm
- Classified based on shape and size (McCance & Huether, 2018)

**Aneurysmal Subarachnoid Hemorrhage**
- Caused by rupture of intracranial aneurysm
- Ruptured aneurysm leaks blood into subarachnoid space
- As rupture progresses and blood is forced into subarachnoid space, it irritates the neural tissues and produces an inflammatory response
- Blood clots arachnoid granulations and impairs CSF reabsorption and circulation
- Increase in blood volume and CSF volume in the brain can cause increases in intracranial pressure (ICP)
- Increased intracranial pressure can lead to hypoxia in parts of the brain causing infarction and, if not treated, brain death and death (McCance & Huether, 2018)

<table>
<thead>
<tr>
<th>Hunt Hess Scale</th>
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<td>Grade</td>
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(Lee, Chi, Edwards, Chang, & Side, 2014)

**Clinical Presentation**

- Thunderclap headache typically known as the "worst headache of my life"
- Nausea, vomiting, nuchal rigidity, photophobia
- Other neurological deficits seen in stroke patients such as paresis, cranial nerve deficits, coma, etc.
- Seizures
- Severity of bleed graded with presenting symptoms using Hunt Hess Scale – the larger the grade, the increased risk of mortality (Danse & Mohet, 2017; Lawson & Gates, 2006)

**Risk Factors**

- Family History
- Hypertension
- Anticoagulation use
- Substance abuse
- Oral contraceptive use
- Cerebral AVMs
- Ehlers-Danlos Syndrome type IV
- Marfan Syndrome
- Polycystic kidney disease
- Fibromuscular dysplasia
- Women 21 to 55 years old
- Higher incidence in African Americans and Hispanics (McCance & Huether, 2018; Danse & Mohet, 2017)

**Complications**

- Hydrocephalus: Obstruction of CSF flow or reabsorption causing its build up within the ventricles of the brain (Danse & Mohet, 2017)
- Delayed Cerebral Ischemia (DCI): Impairment of cerebral blood flow or hypoperfusion due to inadequate cerebral perfusion pressure (CPP), cerebral vasospasm, or microthrombi that typically occur 3 to 15 days post-injury (Yu et al., 2015)
- Seizures (Danse & Mohet, 2017)
- Re-bleeding: occurs in 4% of patients within the first 24 hours post injury (Danse & Mohet, 2017)
- Hypotension & Hypovolemia: most likely related to excess artifial natriuretic factor release and/or excess anti-diuretic hormone excretion (Danse & Mohet, 2017)

**Diagnosis**

- Initial diagnostic: non-contrast head computed tomography (CT) scan
- Head computed tomography angiography (CTA): Gold standard for confirming diagnosis, hemorrhage source, and aneurysm size and location
- Lumbar Puncture: performed 1-2 hours after onset of symptoms. The presence of xanthochromia and an elevated opening pressure is indicative of a SAH
- Magnetic Resonance Imaging (MRI)

(Danse & Mohet, 2017)

**Conclusions**

The occurrence of an aneurysmal SAH could cause detrimental changes in an individual’s life. The promotion of signs and symptoms will raise awareness of the condition and, in turn, will decrease the time between injury and treatment in those experiencing early signs. Within the hospital, knowledge of the most recent evidenced-based treatment modalities ensures adequate patient care and safety. “Early aggressive resuscitation and critical care management have been shown to improve outcomes” (McLeod & Salmons, 2014). By promoting quick and adequate care along with education to providers we can give patients diagnosed with a SAH a greater chance at a positive outcome and quality of life.

**Treatments/Management**

**Procedural**

- Clipping
- Coilings (Yu et al., 2018)

**ICSI prevention/Management**

- Risk measured using modified fisher scale
- Daily transtuminal doppler (TJD) velocities
- Nimodipine 60 mg every 4 hours
- Cerebral Angiography for diagnosis of DCI
- Intra-arterial vasodilator or balloon angioplasty for refractory cases (Bouloud et al., 2017)

**Hydrocephalus**

- External venous device (EVD) insertion to drain CSF and avoid increased ICP
- Ventriculoperitoneal shunt if CSF diversion is needed long term (Danse & Mohet, 2017)

**Nursing Implications**

- Hourly neurological checks to monitor for prompt recognition of change in neurological status, which signals re-bleed or DCI
- Hourly input and output monitoring for wash of hypovolemia and hypovolemia
- Close temperature monitoring for hyperpyrexia
- Systolic blood pressure < 140 before aneurysm uncerrent
- Systolic blood pressure 160-180 for DCI prevention after aneurysm uncerrent
- Monitor ICP and CSF output hourly if EVD is present

(Danse & Mohet, 2017; McLeod & Salmons, 2014)

**References**

- McCance & Huether, 2018; McLeod & Salmons, 2014;
- Moheet & Moheet, 2017
- Lawson & Gates, 2016)