

Otterbein University

Digital Commons @ Otterbein

Nursing Student Class Projects (Formerly MSN)

Student Research & Creative Work

Fall 7-29-2019

Aneurysmal Subarachnoid Hemorrhage

Paxton Schwaderer

paxton.wenger@otterbein.edu

Follow this and additional works at: https://digitalcommons.otterbein.edu/stu_msn



Part of the [Nursing Commons](#)

Recommended Citation

Schwaderer, Paxton, "Aneurysmal Subarachnoid Hemorrhage" (2019). *Nursing Student Class Projects (Formerly MSN)*. 373.

https://digitalcommons.otterbein.edu/stu_msn/373

This Project is brought to you for free and open access by the Student Research & Creative Work at Digital Commons @ Otterbein. It has been accepted for inclusion in Nursing Student Class Projects (Formerly MSN) by an authorized administrator of Digital Commons @ Otterbein. For more information, please contact digitalcommons07@otterbein.edu.

Aneurysmal Subarachnoid Hemorrhage

Paxton Schwaderer BSN, RN, CCRN
Otterbein University, Westerville, Ohio

Introduction

"Aneurysmal subarachnoid hemorrhage (aSAH) is the most devastating type of stroke" (Yu, et al., 2018). It effects up to 16/100,000 adults per year worldwide with up to 14/100,000 of those adults being from the United States (Darsie & Moheet, 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 life-long neurological deficits
- Patients with a SAH diagnosis are at high risk for many complications during their treatment (Darsie & Moheet, 2017)

For many nurses, the neurological system serves as a weakness in their knowledgebase. It is a specialized field requiring specialized care. 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 morbidities 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

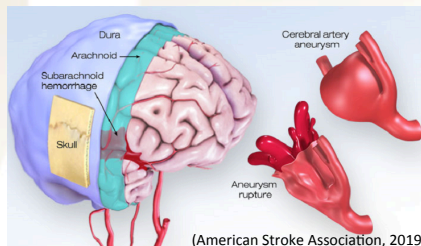
When an aneurysm forms at a vessel bifurcation, it weakens the walls of the vessel. This in combination with high intravascular pressure can cause a rupture of the aneurysm, which displaces blood into the subarachnoid space in the brain (McCance & Huether, 2018).

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 clogs 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 herniation (McCance & Huether, 2018)



(American Stroke Association, 2019)

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, ect.
- Seizures
- Severity of bleed graded with presenting symptoms using Hunt Hess Scale – the larger the grade, the increased risk of mortality (Darsie & Moheet, 2017; Lawson & Gates, 2016)

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 2:1
- > 55 years old
- Higher incidence in African Americans and Hispanics (McCance & Huether, 2018; Darsie & Moheet, 2017)

Hunt Hess Scale

Grade	Description
1	Asymptomatic, or mild headache, slight nuchal rigidity
2	Moderate to severe headache, nuchal rigidity, cranial nerve palsy may appear
3	Drowsiness and/or confusion, appearance of other focal neurologic deficits
4	Stupor, moderate to severe hemiparesis
5	Coma, decerebrate posturing

(Lee, Choi, Edwards, Change, & Slade, 2014)

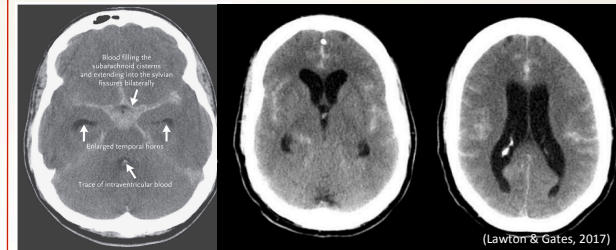
Complications

- Hydrocephalus: Obstruction of CSF flow or reabsorption causing its build up within the ventricles of the brain (Darsie & Moheet, 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 occurs 3 to 15 days post-injury (Youself et. al., 2015)
- Seizure (Darsie & Moheet, 2017)
- Re-Bleeding: occurs in 4% of patients within the first 24 hours post injury (Darsie & Moheet, 2017)
- Hyponatremia & Hypovolemia: most likely related to excess arial natriuretic factor release and/or excess antidiuretic hormone excretion (Darsie & Moheet, 2017)

Diagnosis

- Initial diagnosis: non-contrast head computerized axial tomography (CT scan)
- Head computerized tomography angiography (CTA): Gold standard for confirming diagnosis, hemorrhage source, and aneurysm size and location
- Lumbar Puncture: performed >12 hours after onset of symptoms. The presence of xanthochromia and an elevated opening pressure is indicative of a SAH
- Magnetic Resonance Imaging (MRI)

(Darsie & Moheet, 2017)



Treatments/Management

Procedural

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

DCI prevention/Management

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

Hydrocephalus

- External ventricular device (EVD) insertion to drain CSF and avoid increased ICP
- Ventriculoperitoneal shunt if CSF diversion is needed long term (Darsie & Moheet, 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 watch of hyponatremia and hypovolemia
- Close temperature monitoring for hyperpyrexia
- Systolic blood pressure < 140 before aneurysm securement
- Systolic blood pressure 160-180 for DCI prevention after aneurysm securement
- Monitor ICP and CSF output hourly if EVD is present

(Darsie & Moheet, 2017; McLeod & Salmons, 2014)

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.

References



OTTERBEIN
UNIVERSITY